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CHEMICAL AND MOLECULAR PROCESSES IN CONTRACTILE ACTIVITY

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ANY MODERN DISCUSSION on the chemical and molecular nature of cardiac activity is largely based on the presumed analogy between the processes in the heart and those in striated muscle. This is due to the circumstance that, while skeletal muscle has been the classical material for those biochemical researches which lead to present-day knowledge concerning myosin, actin, phosphocreatine and adenosine triphosphate (ATP), similar experimental investigations on cardiac muscle have been carried out only sporadically. While an upsurge of interest in this direction has become noticeable, we still have to lean heavily on information derived from skeletal muscle.

The chemical physiology of the heart seems to stand by itself only in one respect, namely, with regard to those processes which cause its automaticity. Morphologically, this regulation is ascribed to the nodal and conductive tissues. There has recently been an increased interest in the biochemical investigation of these tissues, as evidenced by valuable reports on its ionic composition (Davies, *et al*) and its lipid content (Robb and Mallov). In view of the resemblance between its task and that of nervous tissue, and of the importance of the acetylcholine system in the generation and conduction of impulses in the nervous system, we have recently investigated the distribution of cholinesterases in the heart. It appears that the myocardium contains an unspecific cholinesterase, while the Purkinje tissue is endowed with the specific acetylcholinsterase commonly found in nerve. This enzyme is characterized by a high rate of splitting of acetylcholine and, to a lesser extent, of β-methylacetylcholine; by a characteristic dependence of its activity upon the substrate concentration; and by its inability to hydrolyze butyrylcholine and benzoylcholine. The enzyme was found in the bundle of His and the AV-node in about the same amount as in typical peripheral nerve, and occurs also, in about half the concentration due to the admixture of connective tissue, in the moderator bands.

Returning now to the chemistry of the myocardium, the first question to be discussed is its dependence upon continuous aeration. Incorrectly, this is sometimes regarded as a sharp difference between cardiac and voluntary muscle. The purpose of metabolism—whether respiratory or glycolytic—is to generate high-energy phosphate compounds, primarily ATP. Much more ATP is generated by aerobic than by anaerobic dissimilation of foods. There are types of muscle which are well provided with all the enzymes involved in tissue respiration and aerobic phosphorylation, such as cytochrome oxidase, cytochromes, flavin enzymes, et cetera; sufficient to resynthesize ATP at the same rate as it is used in the highest activity of the organ. The heart is an example of such muscles, but there are many instances among skeletal muscle as well. Since in these cases, respiratory metabolism is so well developed, and is supported by an ample circulatory oxygen supply, no need arises for additional metabolic pathways like glycolysis. On

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the other hand, there are muscles which function only intermittently, in periods of high activity but short duration. These are usually not well endowed with respiratory enzymes so that during intense activity the consumption of ATP far exceeds its respiratory resynthesis. Such muscles usually possess an active glycolytic enzyme system, which delays the exhaustion of the energy-rich phosphate compounds without being able to prevent it in the long run. These muscles are therefore not able to work at a high rate for unlimited periods, while in the former group a stationary balance between energy output and metabolism can be maintained. That the heart belongs in this group for functional reasons is obvious, but clearly its metabolism does not set it apart from all other muscles.

We have already referred to the fact that ATP is used in contractile activity. This important insight rests first of all upon the results obtained in the laboratory of O. Meyerhof, up to the late 1930's. As other substances such as lactic acid and phosphocreatine, became eliminated as the direct causative agents of contraction, ATP remained as the substance which, as far as one could tell, must be directly linked with the contractile process.

To cause contraction, or to provide energy for it, ATP must interact with the contractile structure which we may presume to be of protein nature. While there is a large number of different proteins in the sarcoplasm, the protein composition of the fibril is fortunately much simpler. For a time, the contractile filaments were even believed to consist of one protein only—myosin. A fundamental study by Von Muralt and Edsall (1930) revealed that this protein displays the property of flow-birefringence, which suggests that it consists of very long molecules or particles, well suited to build up a fibrous structure. Weber prepared myosin threads by extrusion into a precipitating bath of dilute KC1; these threads are good optical models of the anisotropic bands of the muscle fibril. In 1939, it was then found by Engelhardt and Ljubimova that this structure protein has enzymatic activity in that it acts as an adenosinetriphosphatase, myosin-ATPase. This was a discovery of great importance—quite independent of our eventual judgment concerning its direct physiological meaning—since it pointed for the first time to a concrete connection between energy-yielding reaction and contractile structure. The same authors as well as Needham *et al* (1942), also discovered that ATP exerts some physical effect upon myosin.

These important indications were fully explored in the classical investigations of A. Szent-Györgyi and his school, beginning in 1940. It was found, first of all, that myosin in the older sense is not a well-defined substance, but that products of highly different behavior can be obtained. dependent on such seemingly trivial details as the duration of the extraction or the degree of mincing of the tissue. Short extraction of coarsely ground muscle with 0.5 M KCl yields a product which approaches what is now known as myosin itself. This was crystallized, with some denaturation, by Szent-Györgyi, and was prepared in entirely pure form by Weber *et al*, and by Mommaerts and Parris. Myosin consists in all probability of rod-shaped molecules of 160 mμ. long, with a molecular weight which is estimated as 850,000 in Weber's laboratory and 650,000 in ours. Prolonged extraction of minced muscle, on the other hand, leads to the appearance of a complex protein, actomyosin, in which myosin is combined with actin, discovered in Szent-Györgyi's laboratory by Straub. Actin is a protein which can exist in the form of globular molecules (G-actin) or as a fibrous modification (F-actin) originating by polymerization of the globular form. In actomyosin, myosin molecules are randomly attached to the strands of F-actin, while in the muscle fibril, the two proteins apparently are arranged parallel in a highly organized structure. These same proteins also occur in the heart, but their study in this case has considerably lagged behind. Actin and myosin constitute the bulk of the contractile structure, but other proteins such as tropomyosin (Bailey) or Y-protein (Dubuisson), of unknown function, may occur in the fibrils in smaller amounts. That actomyosin is the material substrate of contractility will be documented later; in addition, its participation in contraction is also suggested by the observation of Dubuisson that it is temporarily rendered insoluble in shortened muscle.

The second important discovery by Szent-Györgyi was that both myosin and actomyosin have ATPase activity, but that only actomyosin is strongly affected by ATP in its physical behavior. The influence of ATP can be detected in actomyosin solutions by physical measurements, but shows very striking manifestations in precipitated actomyosin systems. Going from the lowest to the highest forms of organization, these actomyosin systems present the following features.

In suspension, flocculated actomyosin becomes denser, because the individual flocks contract and expell water, a phenomenon called supercontraction by Szent-Györgyi. Under some conditions, the individual flocks adhere, and the entire system shows strong synaeresis.

Threads of actomyosin contract upon the addition of ATP to one-third of the original length. The thickness decreases equally, since there is little preferred orientation in the threads, but by rolling and stretching they can be made to contract preferentially in a longitudinal direction.

Threads that contract only lengthwise, becoming thicker when shortening, and that can lift a load, have been prepared by controlled drying of an extruded thread (Weber) or by compression of actomyosin layers spread on a water surface (Hayashi).

Finally, Szent-Györgyi has introduced a preparation of fiber bundles from the *musculus psoas* of the rabbit, extracted in the cold by aqueous glycerol. This is a preparation which contains actomyosin in its original orientation without prior dissolution, but from which most other active constituents are removed in the extraction process. Such fibers likewise contract lengthwise and develop tension, and this tension is equal to the maximal tension of the muscle from which they are derived. Similar preparation obtained from cardiac muscle have been studied by Taeschler and Bing.

Last but not least, Marsh and Bendall have discovered that when such contracted fibers are treated with ATP in the presence of a water-soluble muscle protein, they can be made to relax again. In investigations by Bozler and by Goodall and Szent-Györgyi, relaxation has been obtained by means of phosphocreatine, but this too seems to depend upon the participation of the Marsh-Bendall factor, and, since phosphocreatine may phosphorylate a nucleotide retained in the fibers, it does not necessarily cause relaxation by itself.

In this series, we have arranged the actomyosin systems according to increasing perfection of their molecular organization. In the flocks and extruded threads, the actomyosin particles are unoriented and are held together by few random weak links. The better the parallel orientation obtained, and the stronger the cross linkage achieved, the more will the systems resemble *in vivo* contraction. Throughout the series, salts play a decisive role: a basic concentration of KCl is always added, while Mg promotes and Ca counteracts the contraction. One remaining discrepancy should not remain concealed: the contraction and relaxation phenomena *in vitro* are very slow, requiring minutes instead of fractions of a second, and this difference does not seem due entirely to diffusion of the ATP. We may hope that also this detail will eventually be understood; otherwise, the results will speak for themselves.

While these achievements have gone far in answering the question as to what causes and constitutes contraction, we also have to investigate how fast, by what amounts, when, and how these effects are brought about.

On another occasion, I have given some quantitative statements regarding the metabolic changes correlated with muscular work. It was found that the activation of one gram of muscle involves the breakdown of 0.25 to 0.3 μM. of ATP, and up to 0.5 μM. under conditions of maximal work. Further, it was derived that continuous activity, for example, a tetanic contraction of striated muscle, involves a steady breakdown of ATP, the rate of which depends on the type of muscle, but which may be of the order of 1 mM. per gram per minute for average mammalian muscle in tetanus, and up to about one-tenth of this for the myocardium in extreme activity. Any biochemical mechanism proposed must be in accordance with these estimates.

It is partly in the light of these estimates that I have criticized a too eager acceptance of myosin-ATPase as the *Deus-ex-machina*. The activity of this enzyme depends enormously on the pH and ionic composition of the medium—a fact which makes it very difficult to estimate its possible activity *in vivo*. But doing the best we can, the conclusion is obtained that myosin-ATPase can explain 1 to 2 per cent of the actually occurring rate of breakdown in muscular activity. This conclusion, which is largely based on the strong inhibition of myosin-ATPase by magnesium, has not been generally accepted by other workers in this field. Some o fthem point out that there are cases in which Mg does not inhibit the ATPase; even activations have been reported. But such objections have never been made in quantitative form, and it is not realized that such activation effects have hitherto been found only when the basic activity is very low, for example, at pH 7 in the absence of Ca. Our views have, for instance, been strongly attacked by Hasselbach who described that his actomyosin preparations are activated by Mg, but overlooked that his activated preparations were no more potent than our maximally inhibited ones. I maintain, therefore, that myosin-ATPase cannot be relied upon to explain the events *in vivo*.

An entirely new viewpoint has come from the study of actin. This protein has some remarkable properties, early recognized by its discoverers. While in aqueous solution, it shows no abnormal behavior, but after the addition of salt, its solution becomes extremely viscous and strongly birefringent. As mentioned before, actin occurs in the form of single globular molecules (G-actin) in water, but upon the addition of salt it polymerizes to F-actin. Recently, Straub and Laki and their collaborators showed that actin preparations contain ATP, and that without this substance actin connot polymerize. Following this development, I prepared pure actin by ultracentrifugal isolation of F-actin from a crude extract, followed by depolymerization in the presence of ATP. With pure actin, the mechanism of the polymerization reaction could be investigated quantitatively, and it was found that the following stoichiometric equation holds:

$$\text{actin} + \text{ATP} \rightarrow \text{actin}_{(\text{polym.})} + \text{ADP} + \text{P}$$

This result not only opens up the study of the mechanism of this striking molecular transformation, but has also a direct physiological consequence. For the stoichiometrical proportions are such that if all the 30 mg. actin (mol. wt. about 57,000) in one gram muscle would polymerize once, it would dephosphorylate exactly the 0.5μM. of ATP which, as calculated previously, activate one full twitch of this amount of muscle. This identity suggests strongly that the polymerization of actin is one of the elementary reactions in activity, and is possibly identical with the "fundamental process" of Gasser and Hill which precedes the macroscopically visible contraction and coincides with the initial heat production. The actin polymerization, furthermore, can be very fast, although it has not yet been possible to estimate its actual speed under conditions like those in muscle.

After these observations regarding speed and amount, we can turn to the question, "When?" referring to the problem of the sequence of events in contraction, investigated, by physical means, with such high perfection by A. V. Hill. The occurrence of polymerization of actin during the activation phase is only a hypothesis. Even the all important question of whether ATP is indeed broken down during contraction has never been decided. Encouraged by a recommendation from Hill that the relatively slow skeletal muscles of turtles should provide a favorable material in which to investigate this phenomenon, we have given considerable time to developing a method permitting the instantaneous cooling of such a muscle at any desired phase of contraction. This is achieved by high-speed immersion of a contracting and a control muscle into liquid propane cooled to 90° absolute temperature by which very rapid cooling is obtained. With this method, the sequence of chemical and structural changes is now being investigated.

There remains, then, the final question, "How?" referring to the actual molecular mechanism of the contracting process. I feel strongly that, as long as the preceding questions have not been answered, this last one is altogether too ambitious. Theories about the mechanism of contraction are, at best, fascinating intellectual exercises as long as the basic facts are not yet known.

Bibliography

1. Dubuisson, M.: Muscle activity and muscle proteins. Biol. Rev., 25:46, 1950.
2. Hill, A. V., et al: A discussion on muscular contraction and relaxation: Their physical and chemical basis. Proc. Roy. Soc., London, B 137:40, 1950.
3. Mommaerts, W. F. H. M.: Muscular Contraction, A Topic in Molecular Physiology. New York: Interscience Publishers, 1950.
4. Mommaerts, W. F. H. M.: The nature of contractility. Editorial, Circulation Research 1 (in press) 1953.
5. Mommaerts, W. F. H. M.: The biochemistry of muscle. Ann. Rev. Biochem. (In press).
6. Szent-Györgyi, A.: Chemistry of Muscular Contraction. New York: Academic Press, 1951.
7. Szent-Györgyi, A.: Chemical Physiology of Contraction in Body and Heart Muscle. New York: Academic Press, 1953.
8. Weber, H. H., and Portzehl, H.: Kontraktion, ATP-Cyclus und Fibrilläre Proteine des Muskels. Ergebn. d. Physiol., 47:369, 1952.

SOME CHEMICAL ASPECTS OF THE CONTRACTILE MATTER OF MUSCLE

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CLASSICAL HISTOLOGY provides a clear picture of the striated muscle fiber as it appears externally. A long sarcolemmal sheath contains material which displays regular striations. The histologist describes the muscle fiber as composed of repeating units of relatively isotropic bands (I) and optical anisotropic bands (A). A membrane or band Z bisects the I band and another band M bisects the A band. The finer details which can be seen using the electron microscope will not be discussed here.

The complex architecture of the cross striated muscle and its alterations in various physiological states has led to many theories about the localization of the different substances in the bands. The interest in this question is due to the fact that the visible alterations are considered to furnish clues regarding the contractile mechanism. Muralt and Edsall[8] tentatively suggested after having demonstrated the double refraction of flow in actomyosin solutions that the actomyosin was located in the anisotropic band. Schmitt *et al.*,[5] considered from investigations on formalin fixed material that the fibrils were composed of actomyosin threads. The relative isotropy of the I band was ascribed to a lack of preferred orientation in this region or to some substance compensating the double refraction in this band. Our experiments have led to a somewhat different conclusion.

We have tried to prepare non-fixed myofibrils by using the following method.

The myofibrils were prepared from rabbit skeletal muscle which were minced in fairly large pieces in a cooled mincer. A cooled isotonic buffer solution was added and the minced muscle was ground in a cooled mortar. The mass was suspended in more cooled buffer solution and centrifuged for a short time. The precipitate was discarded and the particles in the supernatant solution were spun down in the centrifuge and resuspended in cooled isotonic solution.

Investigations with a common light microscope showed that the solution contained ribbon like particles with the characteristic cross striation in a fairly pure state which also was shown by an inspection in the electron microscope. The figure shows a bundle of such fibrils at low magnification. No staining has been used. The white bands are the anisotropic ones. From the picture it can be seen that the electron scattering mass is far greater in these bands than in the isotropic bands. This is an indication of an uneven distribution of material. The different bands had about the same length 4μ showing that the contraction must have been small. When a small amount of ATP was added, they contracted violently. I think that they can be considered as part of the structural elements of the muscle.

The following analyses have been made on these fibrils. When they were suspended in 0.5 M KCl for a short time nearly all of the isotropic bands went into solution but the anisotropic bands remained. An inspection in the electron microscope showed that generally little of the material in the isotropic bands was left. From the appearance of the material in the anisotropic bands it could be recognized as being F-actin. It was also found possible to bring into solution the remaining anisotropic bands either by using 0.5 M KI or a pH of about 9, methods well known for bringing actin into solution.

The nitrogen ratio of the two solutions thus obtained was determined. The ratio was $N_{KI}/N_{KCl} = 1.97$. This is in rather good agreement with the result of Engström[1] using x-ray absorption measurements. He found a mass ratio of 1.8 for A/I using the sheath of the striated muscle from Chironomus larvae.

A determination of the phosphorus ratio has also been made. This ratio was 3.70. I think that all the phosphorus is not so firmly bound so this value is probably too low. The figure shows, however, that the greatest part of the phosphorus is bound in the A band. I believe nevertheless that some of the phosphorus really is bound in the I band too, probably as nucleoprotein, the latter conclusions is arrived at in terms of ultraviolet absorption measurements.

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Our results indicate that the actin together with the greater part of the phosphorus is in the A band in the resting state. During the first phases of the contraction the substances move (probably the actin[13]) and unites. An indication that such is indeed the case follows from the fact that on raising the temperature of the fibril suspension the formerly KCl soluble substance no longer goes into the solution on short KCl treatment. This behavior could be accounted for if actomyosin were formed under such conditions.

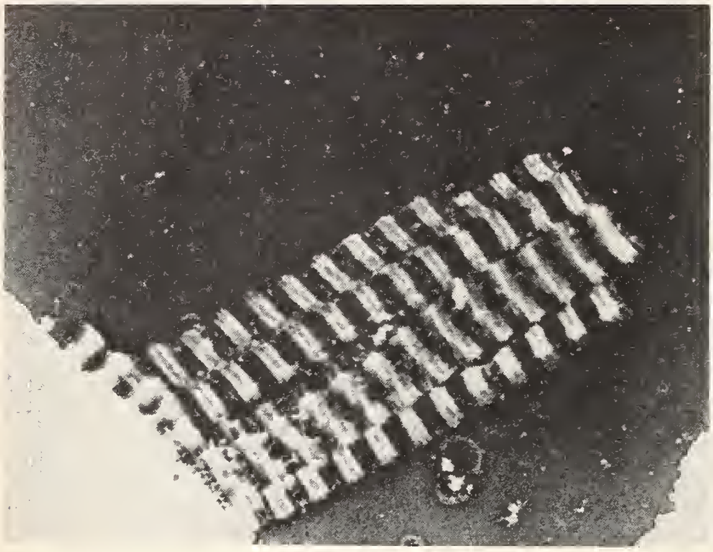

Fig. 1. Electron micrograph of a bundle of muscle fibrils. No staining (x 2 600).

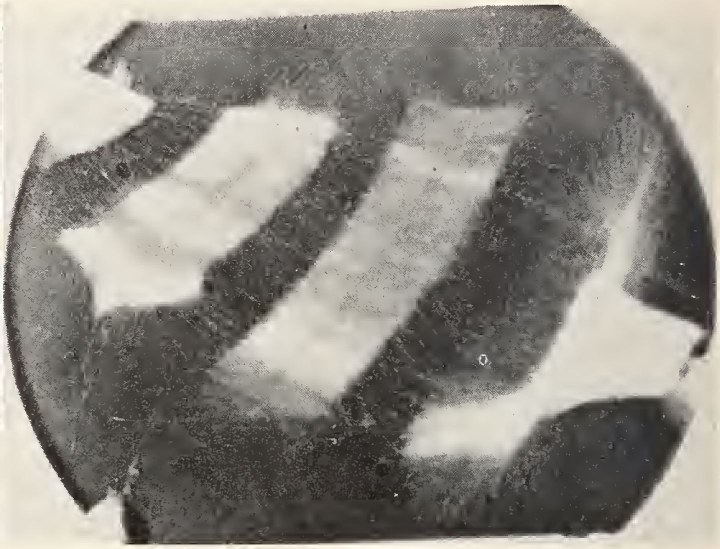

Fig. 2. Electron micrograph of an extracted muscle fibril. The isotropic band extracted with 0.3 M HCl. Gold-manganine shadowed (x 7 200).

In the case of the skeletal muscle the greatest part seems to be myosin in the I band even if, as is probable, some other substances also occur. In such a case, chiefly, actomyosin is built up during the contraction process which in turn probably then reacts further.

Up to the present time heart fibrils have not been investigated extensively. They are more difficult to obtain in a pure state. We must therefore be a little more cautious in making statements about the nature of the substance in the isotropic bands in such tissue even if the fibrils are cross striated. From other investigations we have observed that myosin and actomyosin can only be obtained in relatively poor yields.[2] If the heart muscle is more finely divided, another contractile complex is easily extracted. This complex, we have called actotropomyosin.[11] Our investigations on that complex are, however, mainly done on actotropomyosin extracted from a gravid uterus where it predominates. Our analyses indicate that actotropomyosin is composed of actin, a protein which we have called the phosphate-absorbing protein and tropomyosin mostly as nucleotropomyosin. It may be that in the case of the heart, the isotropic band contains much of the nucleotropomyosin and the phosphate-absorbing protein in a complex. Further studies must, however, be done to elucidate the questions concerning this complex in the heart.

It seems to me that the problem of contractility is connected with structure and alterations in structure. If we want to approach the problem on a molecular level we have (during the first phases) to think of alterations in the structure of the actin. During subsequent phases in the muscle action, alterations in the molecular structure of myosin or the tropomyosin-complex, may also be of importance.

Concerning the actin it is known that it can go from the molecular state of G-actin to the associated form, F-actin. It contains ATP in the G-state and liberates one phosphate (in solution) whence it goes over into the F-state containing ADP. This reaction occurs rapidly when some salt is added to the solution. Under certain circumstances the reaction can be reversed by adding ATP. Straub[14] was the first to show this, followed by Laki[6] and Mommaerts.[7]

Schematically the reaction, following Laki, can be written: $\text{G-actin ATP} \rightleftharpoons \text{G-actin} \sim \text{P ADP} \rightarrow \text{F-actin ADP} + \text{P}$

It seems, however, very unlikely to me that only a split of the phosphate residue would take place without the energy rich phosphate bond being used in a chemically satisfying way. It is therefore unsatisfying to think that, for example, that the formation of any serine-phosphate bond in the actin plays a rôle. Such a bond as it occurs in casein, is too stable.

It is more probable from the general behavior of ATP that a certain substance in the actin reacts with the energy rich phosphate and in that manner raises the actin up to a more reactive level. Also in this reaction divalent ions ought to take part as is usually the case when ATP participates in a reaction. This is also indicated by the fact that the reaction is inhibited by chelating agents.

We have tried for a long time to find in the actin the substance responsible for the reaction. I think now that Dr. Gelotte,[3] during his last time in my laboratory brought evidences for the existence of a substance (X) of low molecular weight nature in the actin. He succeeded in isolating it from purified F-actin by repeatedly shaking it with water and chloroform. In the first water extract, adenine nucleotides and sometimes even hypoxanthine derivatives are obtained in large part. The adenine derivatives are loosely connected with the actin and it is also possible to get rid of them before the experiment. Later on the prosthetic group of the actin appears. It is thus much more firmly bound. The adenine nucleotides are bonded to the actin in much the same way as a substrate molecule is bonded to the special surface of an enzyme. It may be mentioned at this point that the actin contains between 1 to 2 per cent of the new substance calculated on the basis of dry weight.

We have not as yet been able to prepare the new substance (X) in quantities sufficient to enable a determination of its constitution. It has up until present time given much trouble during our investigations because it appears in several forms. Some of them are obviously associated forms. They have also different ultraviolet spectra and can be transformed reversible one into the other. One form, which we think is a kind of monomeric form, has the ultraviolet spectrum given here. Another form can be prepared unassociated with any ions and that form has no maximum in the ultraviolet up to 210 mμ.

Obviously the presence of divalent ions in the substance (X) plays an important role in its reactivity. When possessed of its ions it spontaneously reduces silver nitrate. On the other hand, however, when devoid of its ions or if heated up to boiling point in solution prior to silver nitrate treatment, it cannot reduce it. From the infra-red spectrum it can be concluded that it contains labile carbonyl and amine groups and that it is not an aromatic compound.

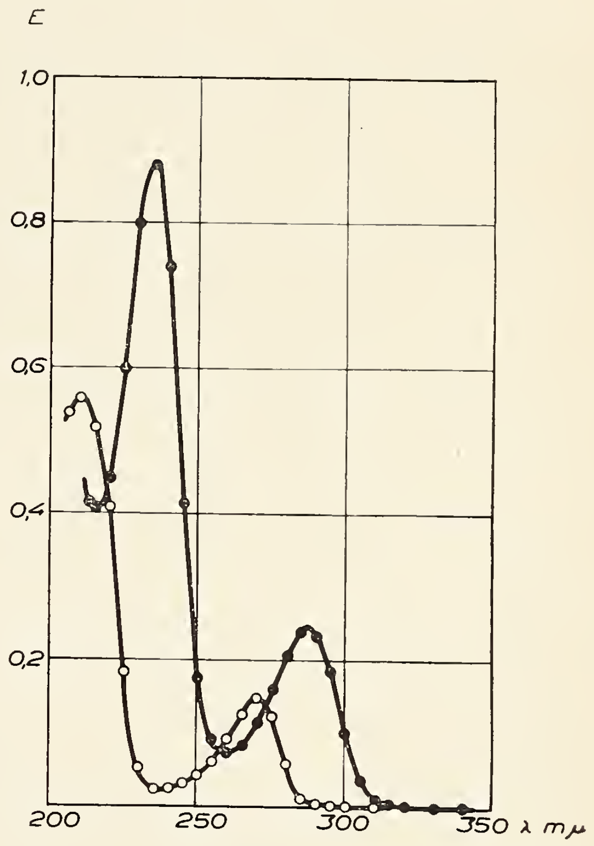

Fig. 3. Ultraviolet absorption curve for the substance (X).
Open circles: in 0.01 M HCl.
Solid circles: in 0.01 M KOH.

These investigations should give a clue to the dependence of the actin on its divalent ions and on energy rich phosphate when they are finished.

The reaction of the actin seems, at the present time, to be peculiar in many ways. One actin molecule reacts with ATP as an enzyme but at the same time it can react with other actin molecules going over to an associated state that is not enzymatically reactive as a phosphatase. This probably depends on the manner in which the reactive substance (X) is bound to the actin. However, with some alterations in the bonds it can be supposed that the actin can act only as a phosphatase.

In this connection I want to discuss another protein which we have found, and which may be derived from the actin in just such a manner. It is the phosphate-absorbing protein that has been prepared from the heart[4] and from the uterus,[12] and is connected with the actotropomyosin complex. I am inclined to consider it (in the free form) as a partly denatured protein but even so it shows some interesting properties. It contains about 10 per cent nucleotide material originating from derivatives containing the substance (X). We consider it, therefore, as derived from the actin. The protein takes up phosphate but only from ATP. It does not associate as actin does.

This phosphate uptake from ATP is probably a residue of its former phosphatase activity which is lost during its preparation. Actually when newly prepared it has a very rapidly lost phosphatase activity. When this protein is in the actotropomyosin it obviously acts as a phosphatase together with the nucleotropomyosin while this system reacts quite as actomyosin. On addition of ATP to an actotropomyosin solution and spinning down the actin the phosphate absorbing protein is found with the nucleotropomyosin in the supernatant.

When preparing the nucleotide material from the phosphate-absorbing protein there was one component of special interest which comes out from the ionic exchanger as one sharp peak containing some phosphorylated compound. A paper chromatograph was made on this using amyl alcohol-phosphate buffer. There could be distinguished six different spots with R_f-values of 41, 49, 57, 64, 69, 78. The spectra of them indicate that they can be related in the same sequence to guanine, xanthine derivatives, hypoxanthine, two derivatives which resembles guanosine and inosine and at last one uracil derivative It can be mentioned that as a certain component on the anionic exchanger a phosphorylated uric acid derivative also appeared. All these derivatives of oxy pyrimidines and purines seems to have the same origin of phosphorylated derivatives with the substance (X). That such a suggestion can be made is revealed also from certain features in the infra-red spectrum from one form of the substance (X).

It seems that the transformation to hypoxanthine and the inosine resembling derivative can occur easily. I think that sometimes when hypoxanthine has been observed in solutions of the contractile matter it can have its origin from such transformations. Therefore, it is not for the moment possible to say when a real deaminase effect has been observed.

Our investigations[9] of the action of cardiac glucosides on the contractile proteins have been quite superficial thus far, for we are in the orientation phase, trying to find the right starting point for the chemical aspect of the problem. The cardiac glucosides have certainly (beside other reactions) a direct influence on the contractile element, that is, reversing the bonding of ions in the contractile system and in that manner influencing the ionic balance in the environment of the muscle fibril.

The function seems to be a regulatory one in which the reactive substance (X) is maintained in an appropriate state by adjusting its ionic bonds in a steady state condition. This view is one which must be made the subject of some future detailed study.

In fact there is already some indication that the cardiac glucosides can act in this manner. We have observed that when a fresh anionic exchanger was used we did not obtain the form of the substance (X) which we have called the monomeric one, this being true even if we started with that form. Using a resin which had been used before, this alteration did not happen. This can be related to an exchange of ions brought about by the resin. If we started with a sample of substance (X) previously treated with cardiac glucosides the monomeric form was obtained even if a fresh resin was used. It seems, therefore that the cardiac glucosides can preserve the reactive substance in an appropriate state and that it acts so when the substance (X) is coupled to protein.

In our investigations of heart actin from cattle prepared according to Straub the preparations were made some hours after the death of the animal. This actin was contaminated with actotropomyosin which was partly degraded. This so-called actin did not associate in the usual way characteristic of skeletal actin when salt was added. An investigation in the electron microscope showed that the actin had formed clusters.[10] On adidtion of cardiac glucosides it associated rapidly to F-actin. On the same kind of actin we observed on investigating the dialysis water that also hypoxanthine derivatives were given off. On using an actin solution which was treated with cardiac glucosides merely adenine derivatives could be detected in the dialysis water.

It remains to be shown how these effects are related. When the actin was treated with alcohol we got an actin solution which did not give off any hypoxanthine derivatives; nevertheless the actin did not associate unless cardiac glucosides were added.

It thus seems that the cardiac glucosides are involved in several of the reactions and that they can do this even in the very small quantities used may depend on the fact that their action is directed against one and the same substance at least in these *in vitro* experiments. We believe that this is the new substance (X) which in different states conjugated to protein can show both ATP: ase property and be transformed in any purine derivatives.

Evidently, our present observations open a path which, when sufficiently traveled, will enable us to learn something about the mode of action of the cardiac glucosides on the contractile element.

References

1. Engström, A.: Progress in Biophysics, Vol. 1. London: Butterworth, 1950.
2. Gelotte, B.: Biochim. et Biophys. Acta, 7:378, 1951.
3. Gelotte, B.: Biochim. et Biophys. Acta, 77:595, 1953.
4. Gelotte, B.: Nature, 168:461, 1951.
5. Hall, C. E.; Jacus, M. A., and Schmitt, F. O.: Biol. Bull., 90:32, 1946.
6. Laki, K., and Clark, A. M.: Arch. Biochem., 30: 187, 1950.
7. Mommaerts, W. F. H. M.: J. Biol. Chem., 198:469, 1952.
8. Muralt, A. L., and Edsall, J. T.: J. Biol. Chem., 89:315, 1930.
9. Snellman, O., and Gelotte, B.: Nature, 165:604, 1950.
10. Snellman, O., and Gelotte, B.: Exp. Cell. Res., 1:234, 1950.
11. Snellman, O., and Tenow, M.: Biochim. et Biophys. Acta (in press) 1954.
12. Snellman, O.: Unpublished.
13. Speidel, C. C.: Am. J. Anat., 65:971, 1939.
14. Straub, F. B., and Feuer, G.: Biochim. et Biophys. Acta, 4:455, 1950.
15. Szent-Györgyi, A.: Acta Physiol. Scand., 9: suppl. 25.

Discussion

Victor Lorber, Minneapolis, Minnesota.—How chemical energy is converted to work in living systems constitutes one of the fundamental problems of biology and medicine. The papers presented by Dr. Mommaerts and Dr. Snellman have indicated the direction which the work on this problem is taking in muscle, where it has been studied most intensively and fruitfully. Much detailed information is at hand concerning the source of the chemical energy and the form in which it is made available for work. Similarly, a plausible concept of the working machinery of muscle is emerging from contemporary studies of the contractile proteins. However, the intimate nature of the link between the two, that is, the exact manner in which the energy of the high-energy phosphate bond is applied to the work process by the contractile elements, is still largely an area for conjecture. At the moment, the most fruitful line of attack would appear to be that exemplified by this morning's essayists, who are working to complete our knowledge concerning the nature of the contractile proteins, the small molecules associated with them, and their behavior under carefully controlled conditions.

The pronounced effect of digitalis on the mechanical efficiency of the myocardium suggests that this drug may be a tool of strategic importance in studying the contractile process. Before the intimate mechanism of its action can be investigated successfully, two outstanding questions must be settled. First, the biochemical point (or points) at which it exerts its effect must be localized. Several lines of evidence suggest that digitalis influences the conversion of chemical energy to work. Conclusive proof, however, is not yet at hand. Next, it is necessary to know what chemical entities are actually responsible for the observed effect. In other words, is the biological action due to the glycoside *per se* (or a metabolite thereof), or is it the consequence of a structural resemblance between the glycoside and a chemical agent native to heart muscle? If the latter is the correct alternative, it should be possible using currently available techniques to demonstrate the presence of a digitalis-like factor in heart muscle. We are at present examining this question.

Assuming that the foregoing problems have been resolved in one way or another, it may be permissible to speculate briefly concerning the direction in which an answer to the final question may be sought. Of the various factors known to influence the activity of biological systems, that of specific ionic concentration would appear to be the most plausible one through which digitalis might operate, perhaps through a membrane effect. Finally, it may not be amiss to point out that the final solution to the digitalis problem may be materially hastened by studying the physiological function of the glycoside in the plant in which it occurs, since we now know that over a wide range of living forms many fundamentally similar problems have been solved in essentially similar ways.

REGULATORY MECHANISMS RELATING TO BLOOD VOLUME

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SCHEMATICALLY, one can compare the blood circulation with a closed pumping system, with elastic channels and a pressure pump which subsequently is filled by pressure on the pump's filling side. In such a pumping system there is an optimal relationship between the maximal velocity of the flow, the resistance and the capacity of the channel system, the liquid volume and the capacity of the pump itself. If the channel system is dilated, without the liquid volume being increased to a corresponding degree, the pressure on the filling side of the pump diminishes and thereby the filling rate also decreases. A reduction of the liquid volume has naturally the same effect. An increase of the fluid volume produces improved conditions for filling the pump, but this effect can only be made use of, if the pump's capacity is increased. An increase in the pump's capacity does not augment the effect of the system, unless the fluid volume and thereby the filling pressure of the pump, is also increased. It might seem futile to compare the circulation, which seems so complicated with its many regulatory mechanisms, with a simple pump system. However, this comparison has been found to elucidate some basic regulations of importance for an understanding of the circulation.

The peripheral part of the vascular system is adapted to the metabolically active tissue mass. Each tissue entity is provided with one or more capillaries; and as the tissue grows, the peripheral vascular system increases in direct relation thereto. Different skeletal muscles, which are used to a different degree show, however, much different capillary density; and in investigations on rats, it has been found that with physical training, there is an increase in the capillary density of the skeletal muscles, as well as of the heart musculature.[12] It looks, therefore, at least in reference to the skeletal and heart musculature, as if the peripheral blood vessel network is adapted to the metabolic rate. The blood conducting vessels appear to be adapted to the peripheral vascular bed, and their plasticity which can be observed under pathological conditions and in congenital defects, makes it probable that this part of the vascular system is adapted both to the tissue mass supplied and the functional demands.

Blood volume shows a direct relationship to body weight during development (Fig. 1). After maturity, the blood volume remains unaltered in humans, practically up to old age, in spite of the fact that the body weight increases in consequence of an increase in fat deposit. As a result thereof, the correlation of blood volume to body weight is low in adults. The same relationship can be observed in rats.[5] This relationship indicates that blood volume is well correlated with lean body mass, or rather, to the metabolically active tissue. The difference between the sexes as regards blood volume, would practically disappear, if the blood volume was correlated to lean body mass.

The blood volume does not appear, however, to be correlated with the metabolically active tissue mass in any simple fashion. If different kinds of animals are compared, it is obvious that the blood volume per unit body weight, varies to a great degree: for example, if rabbits are compared with cats or dogs, or sheep with men. Davis and Brewer[2] have also found that the blood volume increases in dogs during physical training. The blood volume in relation to body weight, body length, or calculated basal metabolism, has also been found to be essentially greater in athletes of both sexes, such as cross-country skiers, middle distance runners, cyclists, than in average subjects. In all, determinations have been done in my laboratory on about eighty athletes and the average differences found, are so large that they appear to exclude every doubt.

With physical inactivity it appears that the blood volume diminishes. Deitrick et al[3] found a lesser difference during immobilization, the determinations being done with Evans' blue dye. In a patient suffering from a fractured leg I have found a 15 per cent decrease. In pulmonary tuberculosis which predisposes to decreased physical activity, the blood volume is also less than

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normal, according to several investigations. During convalescence from different sicknesses, an increase in blood volume has also been found.[11,14] The blood volume accordingly appears to be adapted, on the one hand, to the metabolically active tissue mass, but also, on the other hand, to the metabolic rate.

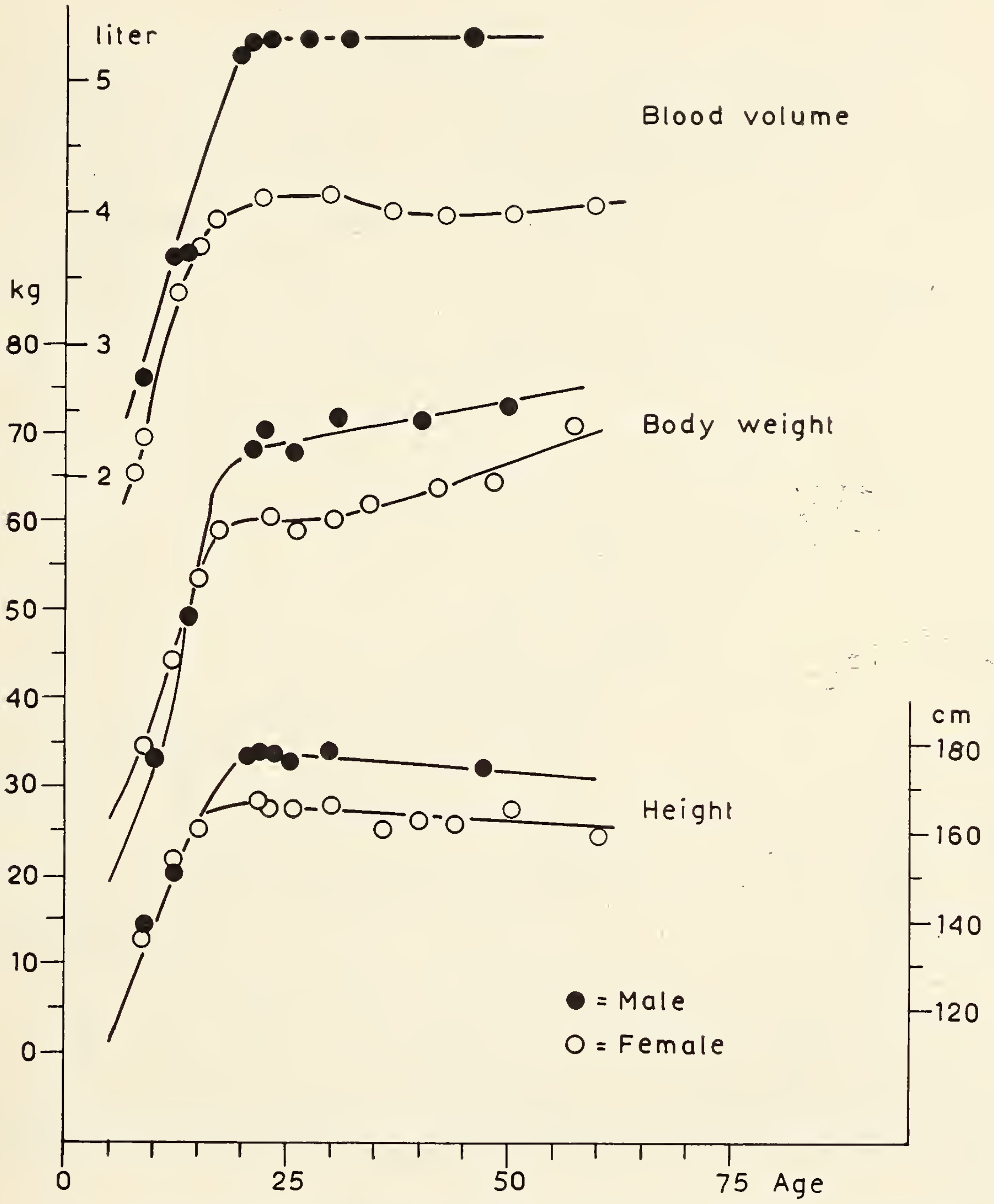

Fig. 1. The relationship between blood volume, body weight, and height and age, respectively. Each dot is a mean of determinations on at least ten different subjects.

The heart volume is greater when lying down than when standing or sitting, according to several x-ray investigations. The heart volume has been measured by x-ray photography in two perpendicular planes at the end of diastole, in the lying position, before, during and after, the effect of drugs like adrenalin, noradrenalin, atropin, nitroglycerin, digitalis in toxic doses: and it has been found that the heart volume remains unaltered or is decreased, but never becomes greater than before the administration of the drug, in spite of the fact that the stroke volume can vary essentially under these conditions. Even during arduous work on the cycle ergometer, the heart volume has proved to be somewhat less than, or the same as, in the lying position.[9] Consequently under the optimal conditions for heart filling, as exist in recumbency, the heart appears to be filled maximally with blood during diastole.

The heart volume has been shown to be directly correlated with body weight. During the years of growth the correlation is very high,[10] but thereafter lower.[8] After puberty, one finds a corresponding difference between the sexes as regards the blood volume, if one correlates the heart volume with body weight or body surface area. As is well known in physical training the heart volume increases. These variations in heart volume are conditioned, above all, by changes in the heart blood volume. The heart volume measured during life on material from men, women and athletes, respectively, has been compared with the anatomical measurement of the heart muscle volume on comparable material reported by anatomists (Fig. 2). It seems obvious from these relations, that the differences in heart volume are due chiefly to increase in the blood volume of the heart and to a minor extent to an increase in the heart muscle mass. It appears that the average thickness of the heart wall is fairly constant. The variations of heart volume with sex or physical training, appear therefore to correspond chiefly to an increase in the diastolic filling volume of the heart.

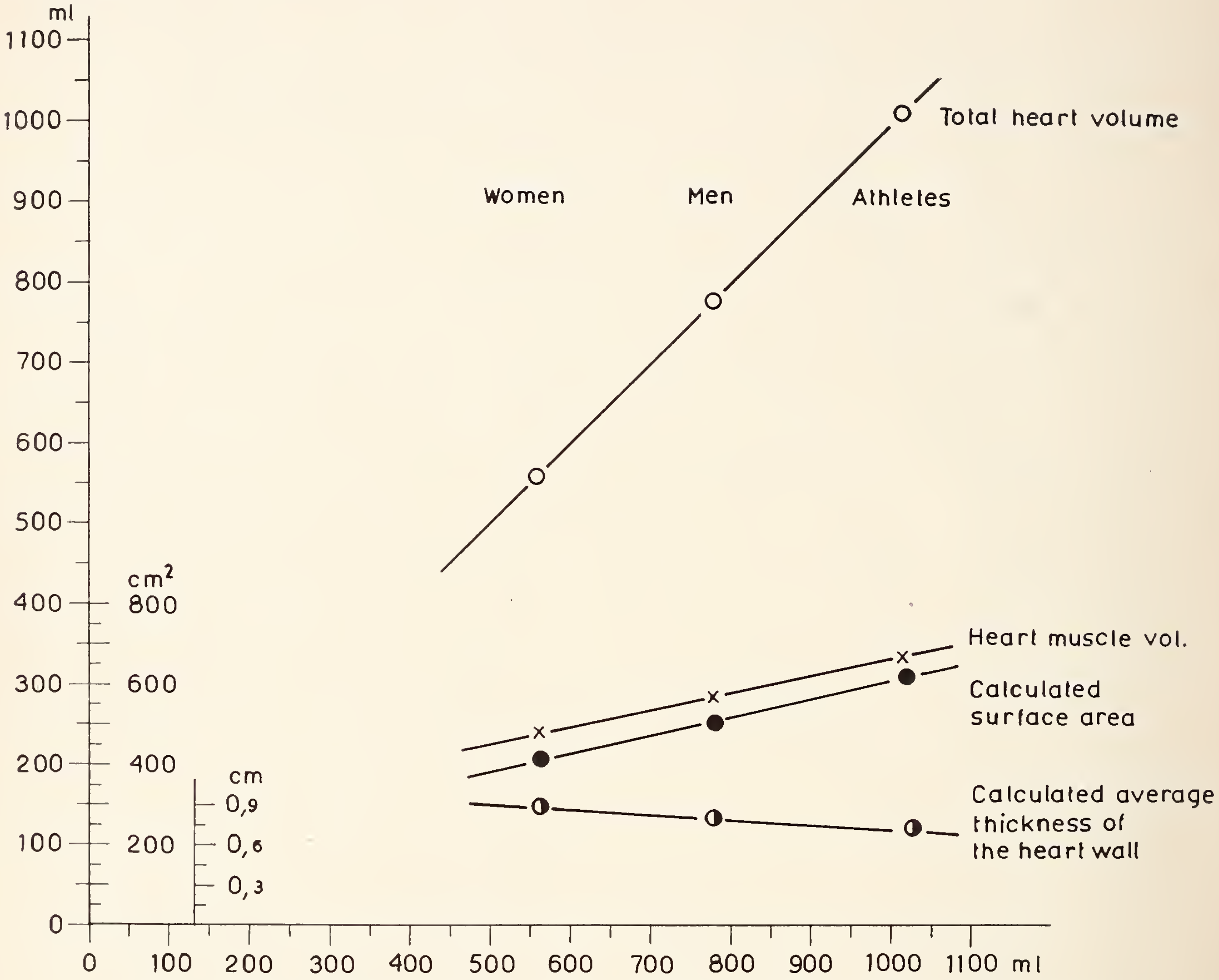

Fig. 2. The relationship between, on the one hand, total heart volume determined by x-ray during recumbency, on material composed of women, men and well-trained athletes, respectively; and on the other hand, the heart muscle volume determined postmortem on comparable material.

Now how are the capacity of the vascular system, the blood volume and the filling volume of the heart in recumbency, related to each other? By correlating the blood and heart volumes with each other, it is possible to gain an understanding of the relationship between these and the vascular system. It has been shown that there is to be found a linear correlation between blood volume and heart volume on a material from men, women and children.[8] Even in a material derived from men exclusively, but including athletes of different types, the same relation is present. Figure 3 shows the mean value for material derived from children, women, sportswomen, pregnant women; cases with pulmonary tuberculosis; average material from male subjects; and athletes of two categories, according to the degree of physical training. The values for these groups lie along one and the same regression line which shows that there is a very close adaptation of the heart's filling volume in recumbency to blood volume and capacity of the vascular system.

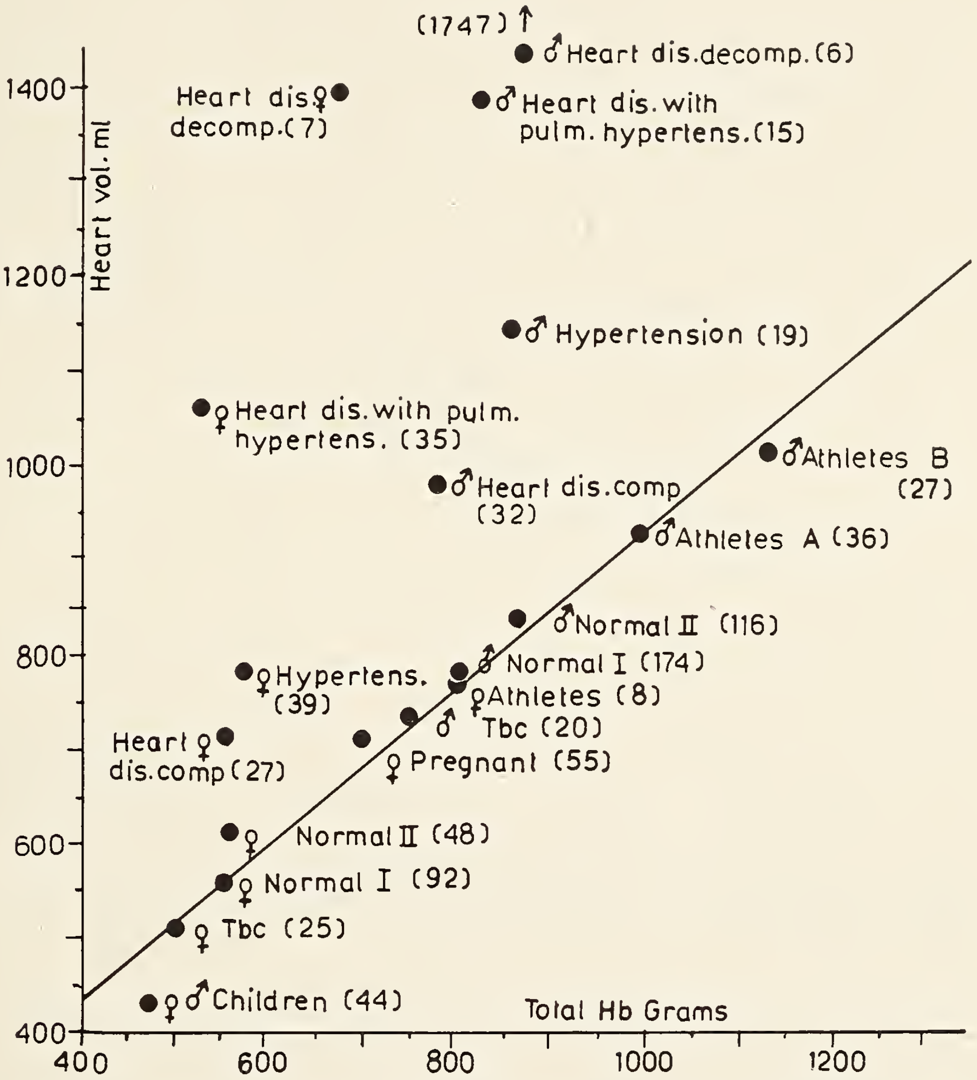

Fig. 3. The correlation between the heart volume determined by x-ray, with the subject lying down, and the total blood hemoglobin. The number of observations in each group is shown by the figure within parentheses.

How are the heart's filling volume and the blood volume related to the heart's maximal pumping effect? Adult men and women, who are made to perform work during three to five minutes on the cycle ergometer, or the treadmill until full fatigue, attain a maximum pulse rate of 190 to 200 beats per minute. Men and women attain about the same maximal pulse rate. Children reach somewhat higher values than adults, while, on the other hand, older individuals show somewhat lower values. The differences are, as a matter of fact, comparatively small. This means that the difference between individuals, in relation to the heart's maximal minute volume during work, is, for the main part, determined by the difference in the heart's maximum stroke volume. Whereas, the cardiac output increases in linear relation to the degree of the work, the pulse frequency during work stands in inverse relation to the stroke volume, and work with a pulse freqency of, for instance 170 per minute, can therefore be made use of as a relative measure of the maximum stroke volume. This work-measure is directly correlated to heart volume in the lying position and blood volume, respectively. Fig. 4 shows the correlation between work with a pulse of 170 and heart volume in material from ordinary men and women and athletes. The greater heart volume in men as compared with women, in the physically trained, as compared with the untrained, corresponds thus to the greater work performance per pulse beat. The same has been found for the relationship between the work pulse rate and the total blood hemoglobin in a material from men, including athletes, women and children.[9] There exists even a direct correlation between the blood volume and the maximal oxygen intake during exercise of short duration.[1]

Thus the maximal blood flow seems to be directly dependent upon the blood volume and the maximal filling volume of the heart. Under normal conditions these factors are adapted to each other and to the capacity of the vascular system.

It has also been shown that between the pulse frequency at rest and at submaximal work, respectively, on the one hand, and the heart and blood volume on the other, there is an inverse linear relation.[8,9] This implies that the greater the maximum stroke volume, the greater the stroke volume at any given cardiac output, independent of whether the body is at rest or performing physical work. This implies also that the stroke volume is regulated in relation to the blood volume and the capacity of the vascular system.[13]

What regulatory mechanisms can be supposed to take part in this adaptation of the vascular system, blood volume, and the filling volume and stroke volume of the heart? One can distinguish between a morphological adaptation, of the vessel wall, peripheral vessels' density, hematopoietic tissue, and heart muscle, and a functional adaptation, by way of the nervous and hormonal regulation. The latter regulation may adapt the capacity of the vascular system but may also influence the regulation of the blood volume.

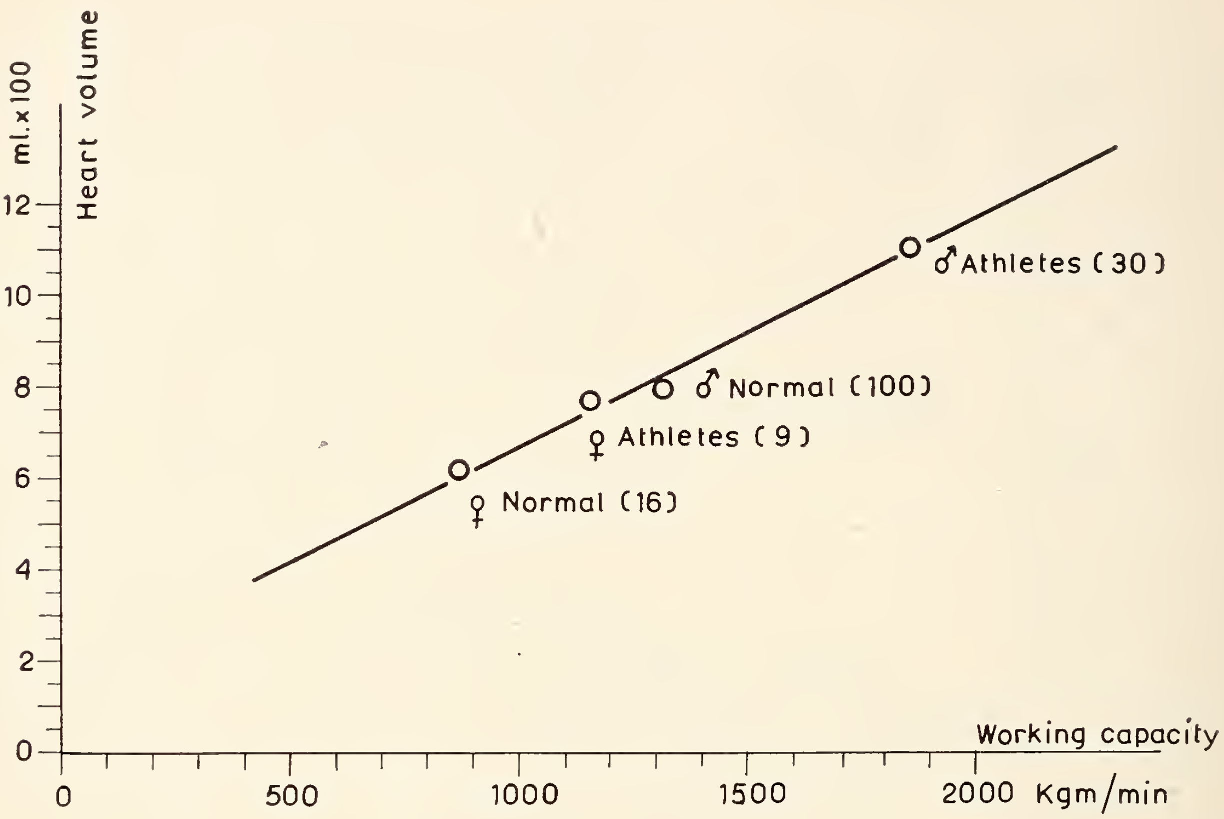

Fig. 4. The correlation between heart volume determined by x-ray and the work performance at pulse frequency 170 on average materials of women and men and well-trained athletes, respectively.

The most important primary control of the blood volume seems to occur by way of the water interchange between the extracellular space and the blood, which among other things, is proved by the fact that the blood volume after a hemorrhage is restored up to 90 per cent of the original amount in a short time. The mechanism in this case is probably, in broad outline, the following: as a result of the decrease in blood volume, the relationship between the capacity of the vascular system, blood volume, diastolic filling volume of the heart and stroke volume is disturbed; the blood volume on the filling side of the right and left heart is diminished and under unchanged conditions, even the stroke and minute volumes of the heart. These changes would initially evoke a decrease in the aortic pressure. This causes a change in vegetative control, in the sympathetic direction, which effects an increase in pulse rate and of vasomotor tone on the arterial side. The aortic pressure increases. As a consequence, the regulatory reflexes on the arterial side may not be responsible for the maintenance of the new equilibrium, and for the subsequent restoration of the original circulatory equilibrium. The stimulation for these regulatory changes may be found in the peripheral and venous parts of the circulation.

Because of the decrease in central venous pressure, coincident with the increased arterial vasomotor tone, the hydrostatic pressure falls in the peripheral vessels, which in turn causes a change in the relationship between filtration and reabsorption of water in the capillaries and so reabsorption increases. Through this the relationship tends to revert to the original state of equilibrium.

The increase in vasomotor tone on the arterial side, involves in itself only an insignificant diminution of the capacity of the vascular system, and causes a reduction of total blood flow in the body. During the first phase after a hemorrhage the adaptation of the circulation is therefore markedly depressed and there is danger of insufficiency. The dilution of the blood secondary to increased vasomotor tone, has essentially a greater importance in the restoration of the circulatory equilibrium than has the direct decrease in the capacity. If this mechanism is inhibited from functioning in consequence of an alteration of vasomotor control, centrally or peripherally, or of an alteration of the water balance, a moderate blood loss may lead to a circulatory insufficiency.

A regulation in the opposite direction has been demonstrated by investigations of Fretheim[4] on the effect of preoperative blood transfusions. If blood is given in a moderate anemia, a shift of water from the blood to the extracellular space takes place and probably secondary to this, diuresis increases. The plasma volume diminishes parallel with the increase in blood cell volume. The total blood volume remains constant. With more pronounced anemias the blood volume can even diminish through transfusion, as Fretheim observed, which is explained by the fact that the capacity of the vascular system is influenced by the oxygen concentration of the blood.

With a primary increase in the capacity of the vascular bed such as occurs during pregnancy, the heart volume as well as the blood volume increase, the latter in some cases alone, through an increase of the plasma volume.

In heart disease, the normal relationships between blood volume, heart filling volume and stroke volume are changed, often to a very great degree. The blood volume has been determined in 270 heart cases, with a well-established diagnosis, and where the circulatory relationships in most cases were analysed by means of cardiac catheterization. It was found, that in cases without stasis in the pulmonary or systemic circulations, but with a slight or moderate heart enlargement, the blood volume was lower than normal, which might be related to physical inactivity which the heart disease forces upon these cases. Heart patients who have stasis in the lesser circulation; and who on the average, had somewhat low minute volume values, showed a small increase of blood volume, especially in men. Cases with increased venous pressure in the systemic circulation, showed in the female subjects, a still more pronounced increase of blood volume. However, the greatest blood volume was found in cases of congenital heart disease with polycythemia, which cases as a rule have neither stasis nor diminished cardiac output. The vascular system can be expected to have become adapted in these cases to the altered circulatory relationships, particularly with regard to the reduction of the arterial oxygen saturation.

In heart disease, the heart volume varies to a much greater extent than the blood volume and the relation between blood and heart volumes is changed which is evident from Fig. 3. One can also show that the relationship between heart filling volume and blood volume, on the one hand, and pulse frequency during work on the other, also departs from the normal to a marked degree.

This condition of things makes possible a more exact basis for the use of the radiological determination of heart volume,[6,7] and for testing the physical working capacity which has been used in cardiological practice during the last three years. Such an analysis has been found to be of definite value, as much in establishing a diagnosis, as in evaluating different cardiac symptoms from the functional point of view.

References

1. Åstrand, P. O.: Experimental Studies of Physical Working Capacity in Relation to Sex and Age, Copenhagen, Munksgaard, 1952.
2. Davis, Y. E., and Brewer, N.: Am. J. Physiol., 113:506-591, 1935.
3. Deitrick, J. E.; Whedon, G. D., and Shorr, E.: Am. J. Med., 4:3-36, 1948.
4. Fretheim, B.: In press 1953.
5. Gemzell, C., and Sjöstrand, T.: Acta physiol. Scandinav., 1954.

(Continued on Page 29)

VENOMOTOR TONE AND BLOOD VOLUME CONTROL

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IN THE PRECEDING paper by Dr. Torgny Sjöstrand, the author has presented a small portion of his comprehensive work concerned with the quantitative assessment of the relationships between blood volume on one hand and sex, geometry and activity of the body on the other hand. From his detailed measurements related to the distribution of blood volume,[11] he pointed to the fact that the size of the heart, particularly the diastolic volume, is an aliquot portion of the blood volume. The interesting observation that the heart rate at a given work output can be predicted from the available blood volume suggests that hemostatic parameters form the base on which possible response patterns of hemodynamics are laid out. In the following I want to present some of our own work, together with Dr. J. P. Henry, Dr. H. O. Siker, Dr. W. E. Wendt and Dr. J. L. Reeves, on blood volume control which throws light from a different angle on issues raised by Dr Sjöstrand.

The close relationship of blood volume to geometry and activity of the body calls for the existence of a blood volume regulatory mechanism which continuously adjusts the blood volume to the size of the vascular bed and the activity of the body. Because the size of the vascular bed depends on the state of contraction of the vascular walls, one must anticipate also a close interplay between control of blood volume and control of venomotor tone. Since von Goltz showed that the blood volume is much too small for the potential capacity of the vascular bed, many investigations have been directed towards the clarification of the mechanisms which control the tension of the blood vessels. It was, thereby, always assumed that the blood volume was constant. This assumption is not justified. During modest blood volume changes induced by hemorrhage or blood transfusions the low pressure system, in contrast to the arterial system, does not aim for constancy of pressure. When, for instance, 500 cc. of blood was drawn or infused at a rate of 300 cc. per minute, the central venous pressure varied linearly by approximately 7 cm. water per 1000 cc. Except for transient initial disturbances rapid transfusions followed the same course. There was from subject to subject surprisingly little scatter, indicating, that vascular tone of the low pressure system, as expressed by $\frac{\Delta P}{\Delta V}$ is in the normal man under resting conditions a rather constant biological parameter. Regardless of the initial rate of blood volume change, the venous pressure only very gradually returned to normal witin 3 to 4 hours.[2,4] The persistence of the hemostatic unbalance induced by blood volume changes is remarkable for two reasons. It may furnish a long lasting stimulus, influencing the dynamics of the arterial system and thereby be responsible for the peculiar behaviour of heart rate described by Dr. Sjöstrand, which would be difficult to explain on grounds of the carotid sinus receptors alone. Another even more important question is this: Does the long persistent change of pressure and volume in the low pressure system represent the adequate stim-

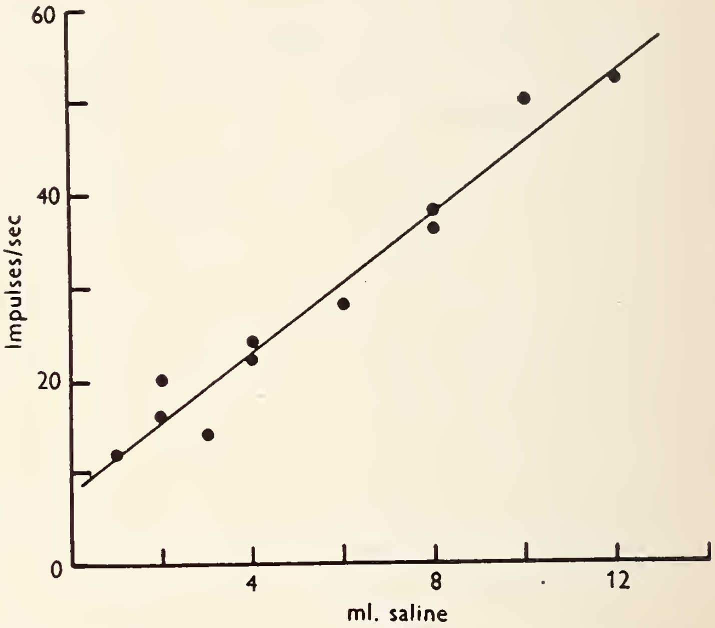

Fig. 1. Paintal: Relation of frequency of discharge in a type B left atrial fibre to various levels of atrial filling in an isolated left atrium.

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ulus for a volume regulatory mechanism which restores the physiological balance, not by changing the tension of the vascular walls, but by restoring the volume to normal?

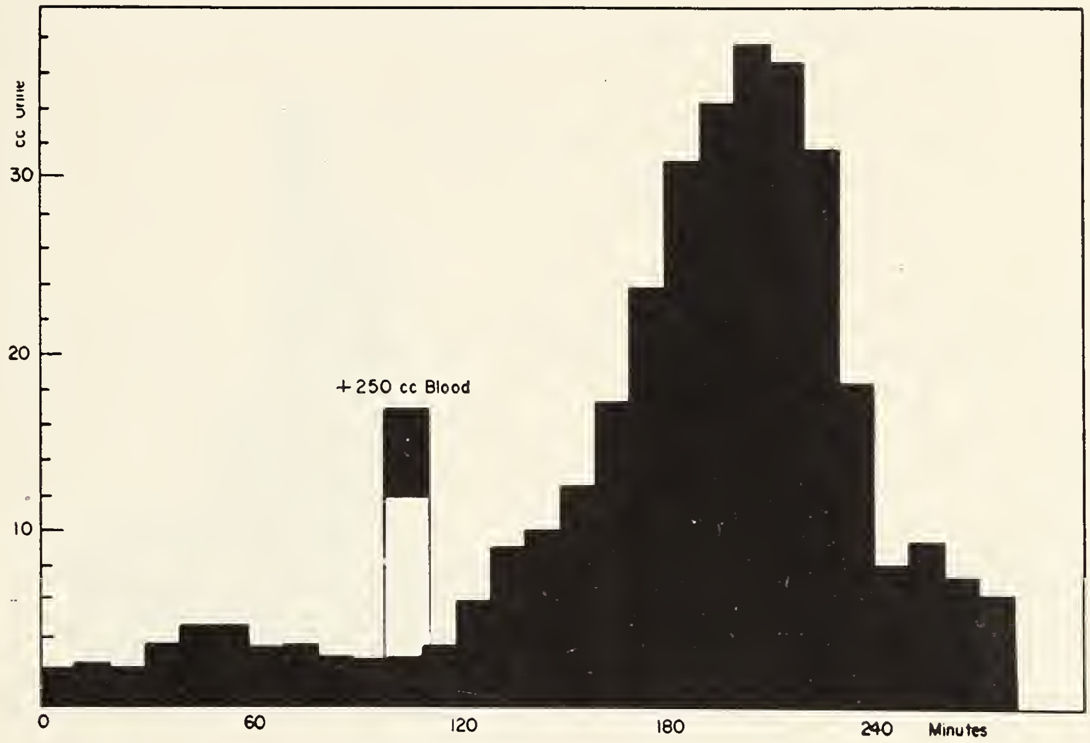

Fig. 2. Effect of a blood transfusion on urine flow in a chloralosed dog. 250 cc. of blood were infused during a period of 15 minutes.

Such a mechanism presupposes the existence of volume sensing elements either in the total circulation, or in parts of the circulation where the distensibilities are representative of the distensibility of the total circulation. While little is known about stretch receptors in the peripheral circulation, a whole variety of such elements has been described in both auricles by Nonidez.[8] Recently Paintal[9] has clearly demonstrated that the vagus carries centripetal impulses which are related to the degree of distention of the auricles rather than the pressures (Fig. 1).

Looking for an indicator organ, for a volume regulatory reflex, one has to keep in mind that final adjustments of blood volume comprise effects on blood protein formation, blood cell volume and water balance. While the first two are rather inaccessible, the excretory part of water balance could with proper precautions be used. One should expect an increase of water excretion with blood transfusion and an oliguria with hemorrhage. Figure 2 is an example of a diuresis produced by a blood transfusion which cannot be attributed to changes of the osmotic conditions or the arterial hemodynamics. It is irrelevant whether the volume increase is induced by a blood transfusion,[1] plasma transfusion,[13] or the injection of washed red cells.[12] If the concept is true, that this effect of blood volume changes on water balance is initiated by Nonidez receptors, and probably executed through the ADH mechanism, the signals being carried to the center in the vagus, it should be possible to obtain the same response of the kidney by a local distention of the circulatory organs which have stretch receptors intercalated in their walls. A slight engorgement of both auricles along with the pulmonary circulation can easily be achieved in lightly anesthetized dogs and normal humans by application of 5 to 15 cm. water constant negative pressure breathing. This procedure leads to an increased urine flow of the type of a water diuresis[5,10] (Fig. 3). Considerable strength has been added to the arguments in favor of the proposed volume regulatory mechanism by the following two observations. In dogs, the effect of negative pressure breathing on urine flow could be eliminated by vagus block.[3] Furthermore, acute inflation of a cherry size balloon, suspended in the left auricle was accompanied by a dramatic diuresis which subsided promptly on deflation of the balloon.[7] This procedure was accompanied by no change or a slight decrease of arterial pressure and an increase of left auricular and pulmonary arterial pressures of approximately 5 to 10 cm. water.

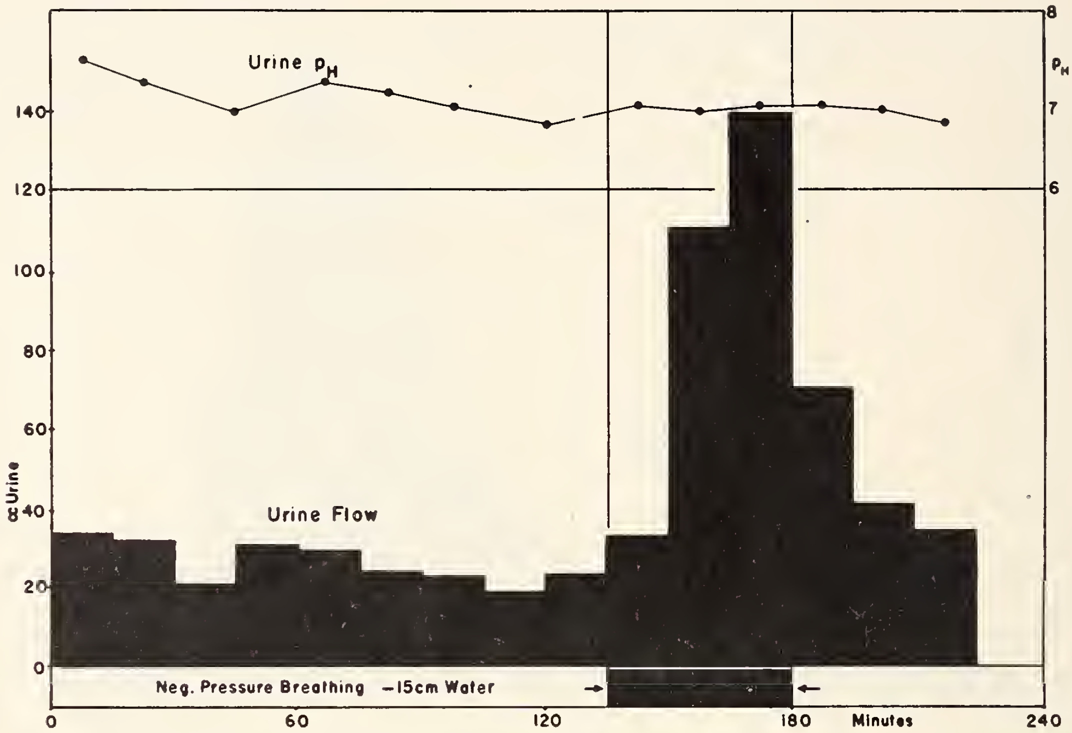

Fig. 3. Sieker et al: Effect on urine flow in humans of 15 cm. constant negative pressure breathing. Negative pressure breathing was applied for 45 minutes (heavy black band on bottom of chart).

The latter results are at variance with those of Hamilton,[6] who found retention of sodium and edema formation in dogs with induced chronic mitral stenosis. In this respect it is interesting to note, that in a normal subject all physiological events which produce a decreased filling of the thoracic pulmonary organs are accompanied by oliguria, while increased filling of the thoracic circulatory organs are usually accompanied by diuresis.[10] The great exception to this rule being congestive heart failure, tentatively leads to the working hypothesis that certain forms of edema formation may be caused by a perversion of the described hemostatic reflex for the control of blood volume.

Both this work and that of Dr. Sjöstrand converge in the emphasis of herostatics as a basic part of the functional structure of the circulation.

References

1. Borst, S. G. G.: The maintenance of an adequate cardiac output by the regulation of the urinary excretion of water and sodium chloride, an essential factor in the genesis of oedema. Acta physiol. Scandinav. supp., p. 207, 1948.
2. Gauer, O. H.; Edelberg, R.; Henry, J. P., and Sieker, H. O.: The pressure-volume relationship in the low pressure system of the circulation. (In preparation.)
3. Gauer, O. H.; Henry, J. A., and Reeves, J. L.: Effect of interference with the thoracic vagosympathetics on urine flow. (In preparation.)
4. Gauer, O. H.; Henry, J. P., and Sieker, H. O.: Blood volume control and venomotor tone. Abstr. Internat. Physiol. Congress, 29:381.
5. Gauer, O. H.; Henry, J. P.; Sieker, H. O., and Wendt, W. E.: The effect of negative pressure breathing on urine flow. (To be published, J. Clin. Investigation, Feb. 1954.)
6. Hamilton, W. F.: The physiology of congestive failure of the circulation. George E. Fahr. Minnesota Med., 36: (Jan.) 1954.
7. Henry, J. P.; Gauer, O. H., and Reeves, J. D.: Effect of local mechanical stimulation of the thoracic circulatory organs on urine flow.
8. Nonidez, J. F.: Studies on the innervation of the heart. II. Afferent nerve endings in the large arteries and veins. Am. J. Anat., 68:151, 1941.
9. Paintal, A. S.: A study of right and left atrial receptors. J. Physiol., 120:596, 1953.
10. Sieker, H. O.; Gauer, O. H., and Henry, J. P.: The effect of negative pressure breathing on renal function. J. Clin. Investigation, 31:662, 1952.
11. Sjöstrand, T.: The regulation of the blood distribution in man. Act physiol. Scandinav., 26:312, 1952.
12. Weech, A. A.: The significance of the albumen fraction of serum. Bull. New York Acad. Med., 15: 63, 1939.
13. Welt, L. G., and Orloff, S.: The effects of an increase in plasma volume on the metabolism of excretion of water and electrolydes by normal subjects. J. Clin. Investigation, 30:751, 1951.

HEART SIZE AND STROKE VOLUME

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IN RECENT YEARS, the traditional descriptions of cardiac function and control have been found inadequate for explaining many experimental and clinical observations. Commonly the heart's response has been predicted from two basic concepts: (a) that the diastolic volume is determined by the effective filling pressure,[31] and (b) that increased initial length of myocardial fibers (greater diastolic volume) causes increased energy release during the next contraction.[16] In other words, the typical ventricular response to a greater pressure, or volume load, is generally described as an increase in diastolic volume. The inevitability of this response is being questioned more and more frequently. Wiggers[28] recently emphasized that Starling's law of the heart applies only "under equal states of responsiveness" of the myocardium since the physiological state of the myocardium is changed by coronary blood supply, humoral agents and nervous actions.

The basic principles of cardiac function were originally based on cardiometer and pressure records obtained from the hearts of anesthetized, thoracotomized dogs. Controlled experiments of this type indicate the capabilities of the heart, i. e., what the heart can do under these particular experimental conditions, but they do not necessarily delineate how it responds under normal conditions. To apply these experimental results directly to normal cardiac function is to imply that the heart behaves in the same way in both normal and abnormal conditions. Obviously this is not so for many reasons, including: (a) Anesthesia unquestionably depresses neural controlling mechanisms. (b) Induced experimental loads are not the same as circulatory adjustments to normal activity; for example, increasing the volume load by elevating a reservoir of blood does not necessarily reproduce the normal adaptations to exertion. (c) Thoracotomy, as we have recently demonstrated, is generally associated with a striking reduction in heart size.[21] A typical example of this response is illustrated by the tracings in Fig. 1. The area of the cardiac silhouette diminished strikingly even before the heart itself was manipulated and the reduced size persisted in this

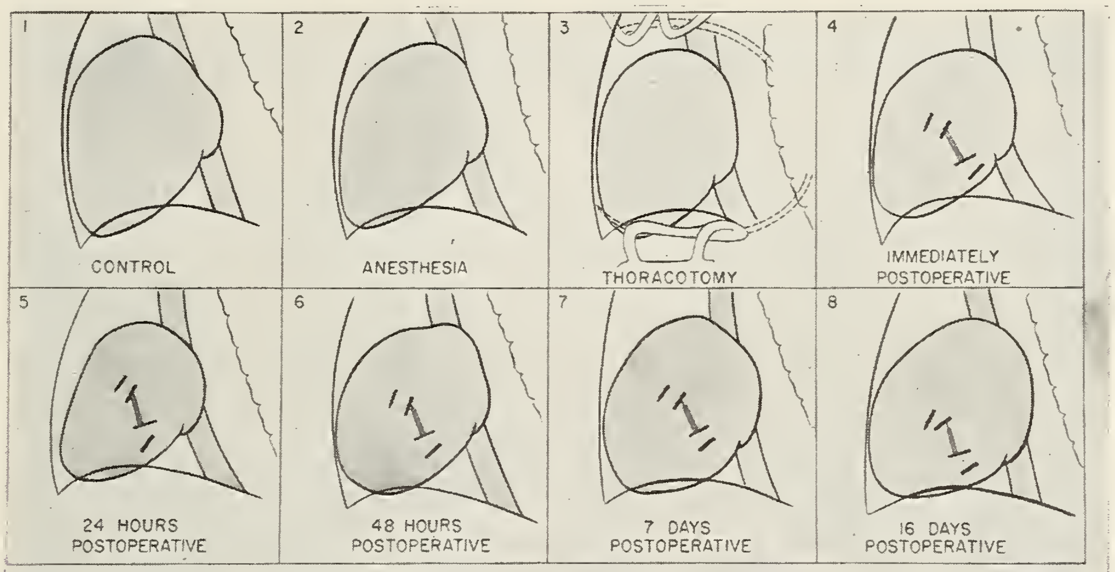

Fig. 1. Tracings of the cardiac silhouette from roentgenograms taken before, during and after thoracotomy, indicate the extent to which the heart becomes smaller when the thorax is opened. Diminished heart size persisted for more than twenty-four hours in this case, and the changes in left ventricular diameter were recorded after forty-eight hours.

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instance more than twenty-four hours after the thorax had been repaired. Changes in the left ventricular area were recorded by cinefluorographic angiocardiography. When the chest was open, the left ventricle emptied almost completely during systole, but considerable quantities of blood remained at the end of systole when the thorax was intact. If the ventricles were abnormally small at the beginning of the original cardiometer measurements, greater diastolic expansion might occur because the ventricles could not get any smaller.

In the previous presentation, Dr. Sjöstrand[27] made the interesting observation that the diastolic volume of the heart of reclining humans is apparently maximal, since he and his colleagues were unable to induce any further increase in size by various drugs and maneuvers which should, according to accepted concepts, cause diastolic expansion. Relatively large volumes of residual blood in the heart of both intact experimental animals[21,23,25] and man[1,9,10,14] have been reported as has a reduction in systolic volume of the heart during exercise.[11,13] Most of these studies utilized roentgenographic techniques which do not indicate the size or volume of the individual chambers of the heart. Thus, the changes in total heart volume represent variations in the volume of any or all of the cardiac chambers. Since the atria, and right and left ventricles, differ widely in their anatomy, geometry, functional architecture and operating conditions,[22] they must be studied individually, not collectively. No method is available at present for measuring directly the absolute volume in a cardiac chamber, but a technique has been devised for continuously measuring the diameter of the left ventricular chamber in the intact unanesthetized dog.[20]

Measurements of Left Ventricular Diameter in Unanesthetized Dogs

The left ventricular diameter was recorded from variable inductance gauges, consisting of a coil 3 mm. x 15 mm., anchored to the free wall of the left ventricle, and a magnetic steel stylus anchored at one end to the interventricular septum (Fig. 2B). The gauge was installed within the ventricle under aseptic conditions by means of trochars.[20] When the ventricular walls contracted and relaxed, the stylus moved in and out of the coil. Delicate wires from the coil extended through the heart wall, between pulmonary lobes, and through the thoracic wall to the outside. The variations in inductance were continuously recorded to determine the position of the stylus within the coil. Since the thorax was open when the intraventricular gauges were installed, the very small left ventricle would barely admit the coil (Fig. 1), and the stylus was immobilized within the coil throughout the cardiac cycle. After twenty-four to twenty-eight hours, the heart had expanded to approximately preoperative size. (The marked reduction in heart size noted during operation on these dogs is in line with our previous contention that thoracotomy induces shrinkage of the heart.[21]) When the animal had recovered, changes in left ventricular diameter were measured continuously during all manner of activity including rather strenuous exertion (Figs. 2 and 3).

The cyclic changes in left ventricular diameter resembled cardiometer records except for a sharp upward deflection during isometric contraction (Fig. 3A). This deflection apparently represented a sudden increase in diameter due to a change in shape of the ventricles without a change in volume. The "end diastolic" diameter was measured at the peak of the A wave (Fig. 3A), which was produced by atrial contraction. The diastolic diameter ranged around 25 mm. The change in diameter during systole amounted to only 3 to 5 mm. which affirms the presence of relatively large volumes of residual blood within the ventricle.

The Startle Response:—The immediate response to an unexpected loud noise was a transient increase in both systolic and diastolic diameter, often followed by a sustained increase in diastolic and stroke diameters (Fig. 3C). The initial increase in diameter and the accompanying tachycardia began during the first cycle after the stimulus (Fig. 3C). We have interpreted these prompt changes in heart size and heart rate as due to neural reflexes acting directly on the heart and circulation.

The Cardiac Response to Exercise:—The first change in cardiac function during exercise was a prompt acceleration of the heart rate, and a progressive reduction in the diastolic and systolic diameters. These changes usually began immediately after the onset of the exercise, but sometimes began in anticipation of the exertion. After a few seconds, the heart rate slowed and the diastolic diameter returned to approximately the pre-exercise level while the systolic diameter usually remained smaller. Systolic ejection during exercise was generally beyond the recording range of the gauge, which blocked during every systolic interval. For this reason, it was impossible to ascertain the extent of the systolic deflection. After the exercise, both the systolic and diastolic diameters generally increased beyond and then returned to the control level. The absolute diameter changed less than was anticipated. However, relatively large changes in the volume ejected can result from small differences in the degree of filling or ejection when the ventricle operates at large dimensions. The fact that the diastolic diameters increased during startle and after exercise indicates that the ventricles were not operating at maximum diastolic volumes during the control period or during exercise. The diastolic diameter increased during a startle reaction and decreased at the onset of exercise even though tachycardia was present on both occasions. Thus, the heart rate is not the principal factor affecting left ventricular diameter.

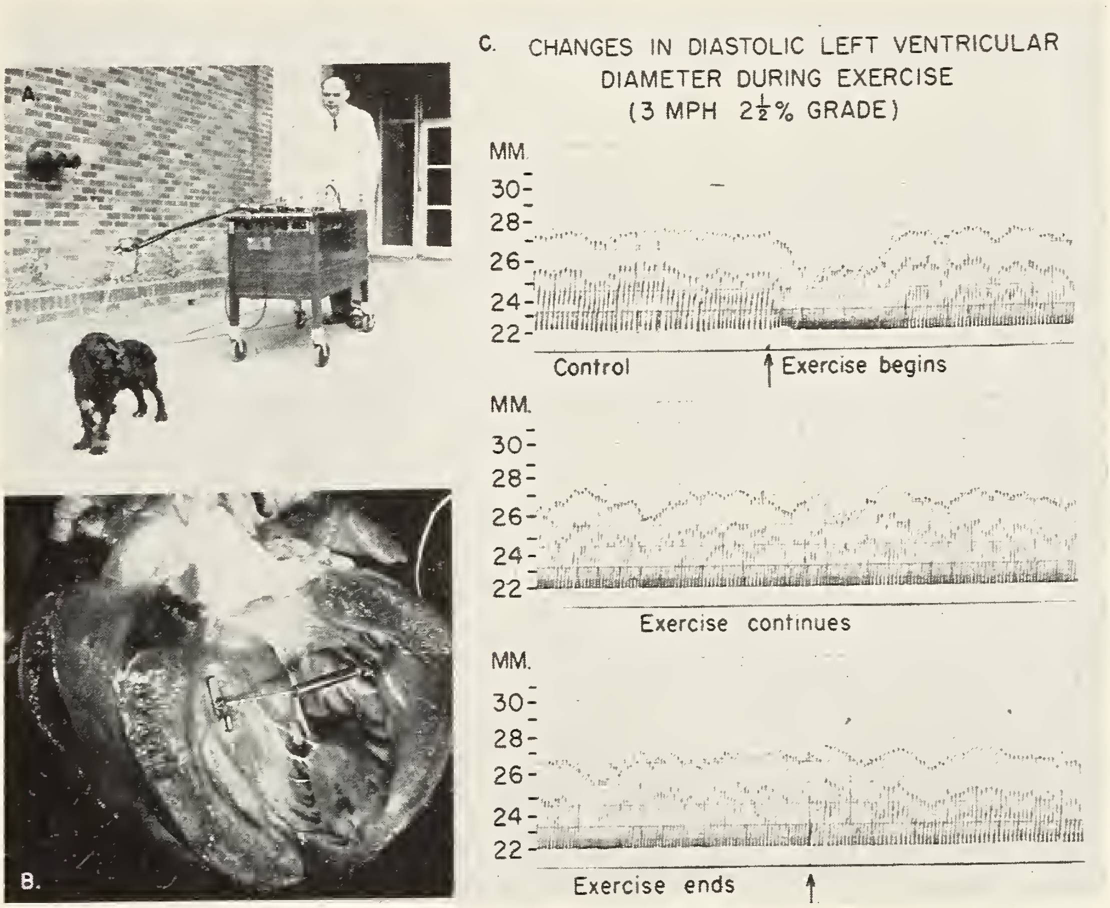

Fig. 2. (A) After recovery from the operation, dogs were exercised while running freely on a treadmill. (B) The variable inductance gauge, consisting of a coil and stylus, which recorded the changes in left ventricular diameter. (C) The initial response, to exercise on a treadmill, was a precipitous reduction in both diastolic and systolic diameters, as the heart rate accelerated. The gauge blocked with each systolic excursion during exercise indicating that the systolic diameter was less than during the control period.

These experiments represent merely the first step along an arduous path toward describing cardiac function and control in intact animals. Thus far, gauges have been successfully installed in fourteen animals. The response to exertion has been recorded forty times in four animals. Although the details of the response vary during exertion or startle, even in the same dog, the general patterns appear to be consistent. Nonetheless, data are still insufficient to derive principles of cardiac function which could be applied with confidence to other dogs or to man.

The Systolic and Diastolic Reserve Volumes

In the current experiments, the cardiac response to voluntary exertion is significantly different from the changes induced by increased venous return to exposed hearts enclosed within a cardiometer as illustrated schematically in Fig.

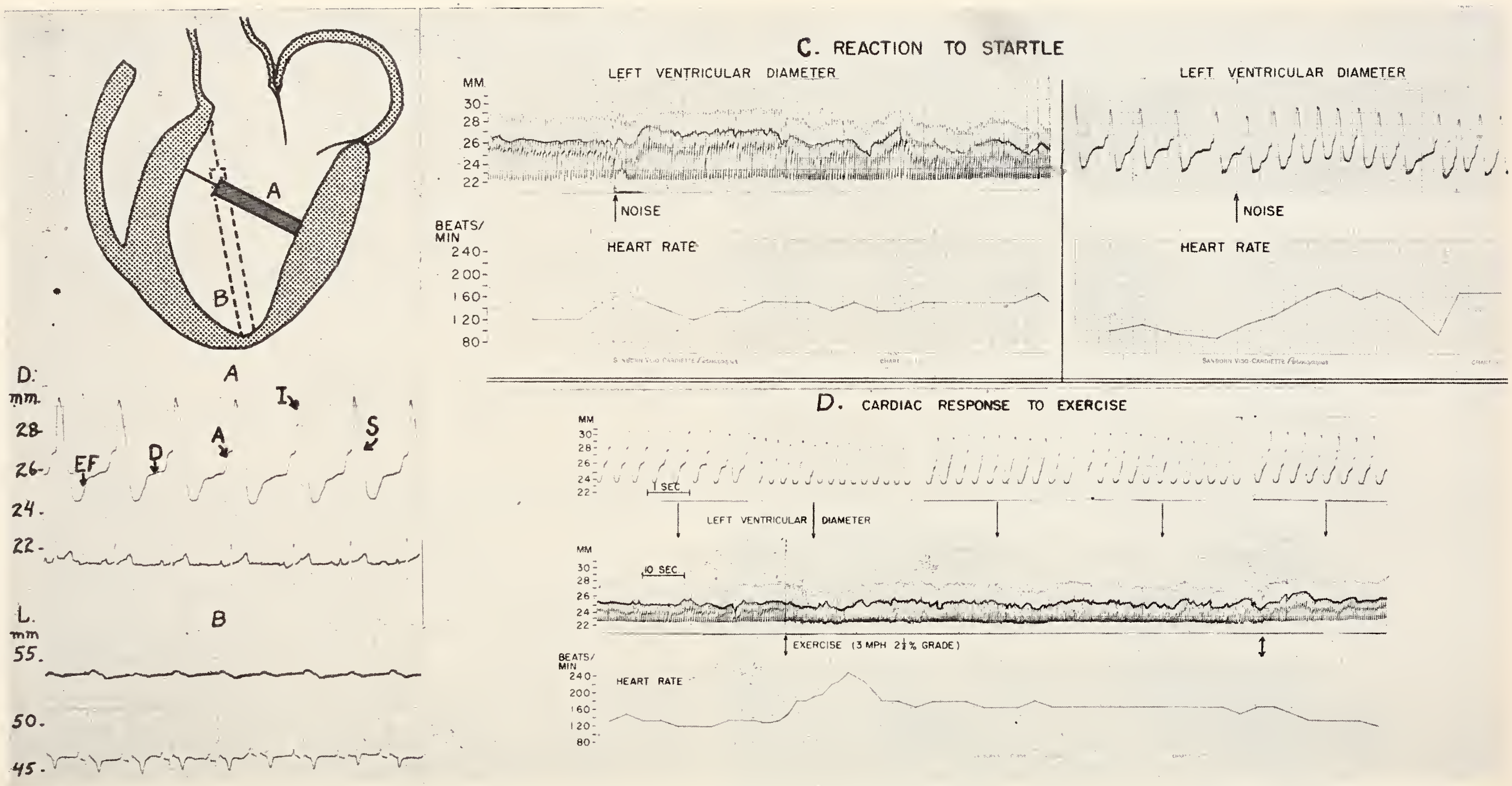

Fig. 3. (A) The changes in left ventricular diameter during each cycle consist of five phases: (EF) early filling, (D) slow filling or diastasis, (A) auricular filling, (I) isometric expansion of the diameter, and (S) systolic ejection. (B) The length of the chamber from the root of the aorta to the apex changes very little during each cycle, but the axis from the apex to the mitral valve ring shortens significantly on cinefluorographic films. (C) The response to startle is illustrated using slow paper speed (2.5 mm./sec.) on the left and the initial effects with fast paper speed (25 mm./sec.) on the right. (D) The changes in left ventricular diameter and heart rate are shown in these sample records. The end diastolic diameters at the peak of the A waves are connected by a continuous line.

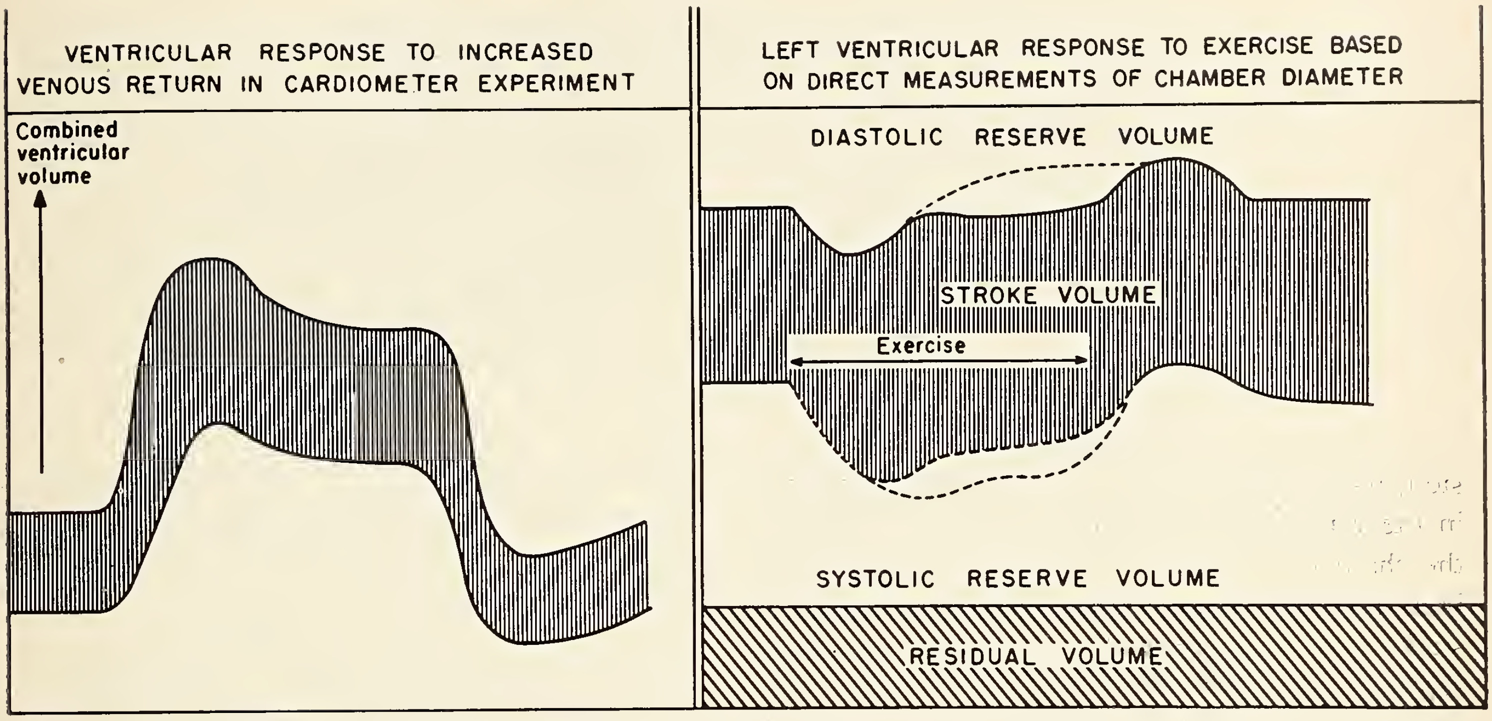
VENTRICULAR RESPONSE TO INCREASED VENOUS RETURN IN CARDIOMETER EXPERIMENT
Combined ventricular volume
LEFT VENTRICULAR RESPONSE TO EXERCISE BASED ON DIRECT MEASUREMENTS OF CHAMBER DIAMETER
DIASTOLIC RESERVE VOLUME
STROKE VOLUME
Exercise
SYSTOLIC RESERVE VOLUME
RESIDUAL VOLUME

Fig. 4. In cardiometer experiments, the diastolic volume, stroke volume, and systolic volumes are all increased during increased venous return (after Patterson, Piper and Starling[16]). According to the author's concept, the stroke volume of the left ventricle is increased during exercise primarily by greater systolic ejection, encroaching upon the systolic reserve volume. By utilizing both the diastolic and systolic reserve volumes (dotted lines), larger volumes of blood can be ejected during each cycle.

4. The increase in stroke volume during exercise is usually attributed to increased venous return, and increased central venous pressure producing greater diastolic distention. The current studies indicate that under resting conditions, the left ventricle is not fully distended during diastole, and is far from being completely emptied during systole. Stroke volume may be increased by more complete systolic ejection or by greater diastolic distention. For convenience, we suggest that the term "systolic reserve volume" be applied to the quantity of blood remaining in the ventricle at the end of a normal systolic ejection at rest. Similarly, the additional quantity of blood which could be accepted by greater diastolic filling might be labeled the "diastolic reserve volume" (Fig. 4). Increased stroke volume might be attained by encroaching on either systolic reserve or the diastolic reserve. Even with a constant stroke volume, the magnitude of the systolic and diastolic reserve volumes may be varied by changes in the "physiological" condition of the ventricular myocardium. We would limit the term "residual" blood to the small quantity of blood remaining in the ventricle after maximal systolic ejection. These terms correspond to those employed in describing lung volumes and have the same logical basis.

Since the left ventricle is neither completely filled during diastole nor completely emptied during systole, the factors which limit the expansion and contraction of the chamber must ultimately be established. In this way the mechanisms for increasing stroke volume might be approached more logically.

Factors Which May Influence Stroke Volume

Stroke volume is the quantity of blood which leaves each ventricular chamber between the end of diastole and the end of systole. Some of the factors which promote or impede diastolic distention and systolic ejection will be considered individually (Table I). Several of these are not generally considered in this regard and deserve attention even though the available evidence is inadequate to indicate their relative importance. Those items which might be directly affected by nervous or hormonal controlling mechanisms will be mentioned.

Systolic Ejection.—The ventricles eject blood into the arterial systems as long as the intraventricular pressures exceed the pressures in the roots of the corresponding arterial trunks. The intraventricular pressure is elevated by contractile tension developed by myocardial fibers within the ventricular wall. The ventricular walls are composed of layers of myocardial bundles, intricately interwoven and oriented in different directions. The contribution of any particular myocardial fiber depends upon its contractile tension, its anatomical position and orientation within the wall, and the geometrical characteristics of the chamber itself.[22] The right ventricular cavity is a crescentic space between the concave-convex surfaces of the free wall and the interventricular septum. The right ventricular wall consists primarily of two myocardial layers which spiral in opposite directions. Simultaneous contraction of these two layers draws the base of the heart (tricuspid valve ring) toward the apex of the heart. Any movement of the right ventricular wall toward the septum produces a bellows-like action which will displace large volumes of blood per unit of myocardial shortening. In the left ventricular wall, a thick cuff of muscles lies between the two spiral layers and encircles the cavity like a clenched fist. Changes in size and shape of the two ventricles are very different during systole. Cinefluorographic angiocardiography has shown that during left ventricular contraction, the relative decrease in diameter is much greater than the relative shortening along its longitudinal axis. Until more data are available, discussion of cardiac contraction must be restricted to general terms with no attempt to analyze the contributions made by individual myocardial bundles and layers. Thus, the stroke volume is established by the amount the various layers of myocardial fibers can shorten before the effective contractile tension is insufficient to maintain an intraventricular pressure higher than arterial pressure. Since arterial pressure is maintained within a relatively narrow range during changes in the level of activity, this factor will not be considered further.

TABLE I. FACTORS INFLUENCING STROKE VOLUME

Systolic Ejection	*Diastolic Filling*
I. Outflow Resistance	I. Effective Filling Pressure
(a) Arterial pressure	(a) Diastolic ventricular pressure
(b) Turbulence	(b) Intrathoracic pressure
II. Ventricular "Contractility"	II. Ventricular Distensibility
(a) Myocardial "contractility"	(a) Myocardial distensibility
(b) Myocardial viscosity	(b) Myocardial viscosity
(c) Law of Laplace ($P = \frac{T}{R}$)	(c) Law of Laplace ($P = \frac{T}{R}$)
(d) Interfascicular tension	(d) Interfascicular tension

Turbulence in the arterial trunks and resistance to the flow of blood through the ventricular chambers[8] represent energy waste. Energy is dissipated in many other ways as well so that only about ten to twenty per cent of the energy released by the myocardium appears as useful work: pressure and flow of blood leaving the chambers. The energy released by the heart and total work accomplished are both expressions of myocardial "contractility."

Myocardial Contractility.—No definition of "contractility" has been universally accepted. For purposes of this discussion, the term will be employed to describe the degree of myocardial shortening in relation to the work accomplished. According to this definition, ventricular myocardium, under constant or increased load, displays greater "contractility" the more completely it empties the chamber during systole. In this light, it is profitable to consider some of the factors which affect contractile tension in relation to myocardial shortening.

Contractile tension of myocardium is affected by the initial length or diastolic volume of ventricles at the onset of contraction.[28] The familiar length-tension diagram describes this relationship by a single curve indicating that contractile tension increases as the fibers are stretched up to a critical level.[5] However, fiber length is only one of many factors influencing myocardial contraction. It is well known that epinephrine increases "contractility" as evidenced by greater intraventricular pressure, faster elevation of pressure, reduced duration of systole,[29] and more complete systolic ejection.[24] Sympathetic nerves, distributed throughout the ventricular walls, discharge epinephrine-like substances which apparently have the same functional effect as circulating epinephrine.[7,17,18,19,26] The rapid reduction in systolic and diastolic diameter at the onset of exercise (Fig. 2), might well result from sympathetic discharge to the myocardial wall. Since heart rate and diameter may change in anticipation of exertion, we must not ignore the possibility that such autonomic controlling mechanisms are initiated in the higher levels of the central nervous system. It is commonly stated that tachycardia is responsible for reduced heart size. The fact that an accelerated heart rate may occur with either an increase or a decrease in left ventricular diameter (Fig. 3) suggests that these changes in size are not the direct result of shorter cycle length. It seems more likely that the factors which produce tachycardia, such as autonomic discharge, usually, but not invariably, promote increased systolic ejection by inducing increased "contractility."

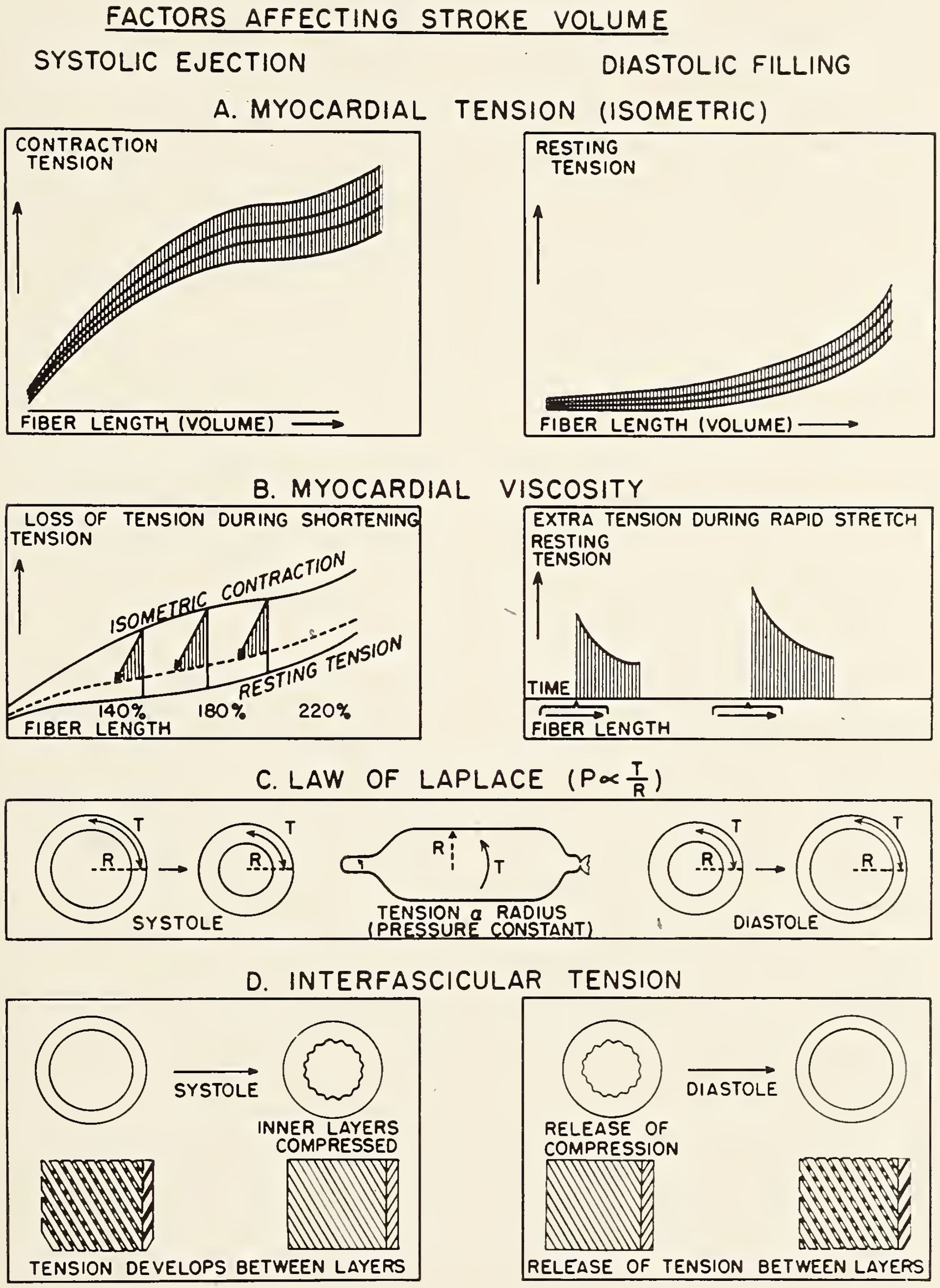
FACTORS AFFECTING STROKE VOLUME
SYSTOLIC EJECTION
DIASTOLIC FILLING
A. MYOCARDIAL TENSION (ISOMETRIC)
CONTRACTION TENSION
FIBER LENGTH (VOLUME)
RESTING TENSION
FIBER LENGTH (VOLUME)
B. MYOCARDIAL VISCOSITY
LOSS OF TENSION DURING SHORTENING
TENSION
ISOMETRIC CONTRACTION
RESTING TENSION
140%
180%
220%
FIBER LENGTH
EXTRA TENSION DURING RAPID STRETCH
RESTING TENSION
TIME
FIBER LENGTH
C. LAW OF LAPLACE (P∝T/R)
T
R
SYSTOLE
TENSION α RADIUS (PRESSURE CONSTANT)
DIASTOLE
D. INTERFASCICULAR TENSION
SYSTOLE
INNER LAYERS COMPRESSED
TENSION DEVELOPS BETWEEN LAYERS
DIASTOLE
RELEASE OF COMPRESSION
RELEASE OF TENSION BETWEEN LAYERS

Fig. 5. Some of the factors which may influence stroke volume are schematically illustrated. Each of these factors affects both diastolic filling and systolic ejection (see text).

Thus, changes in "contractility" of the myocardium represent altered "physiological" conditions of the myocardium. Therefore, the traditional length-tension curve alone does not adequately describe "myocardial contractility." Instead, a family of curves must be established to indicate the range within which the musculature may operate under varying physiologic conditions of the myocardium (Fig. 5A). This will require a great deal more investigation of myocardial activity during contraction at constant length (isometric) and with myocardial shortening during contraction. The schematic length-tension diagrams in Figure 5A represent muscles contracting isometrically. However, muscular contraction without shortening can perform no external or useful work.

Myocardial viscosity is a term used by Lundin[12] to describe a pronounced drop in contractile tension which occurs when myocardial strips shorten during contraction. Viscosity effects in contracting muscles have also been observed by other investigators.[2,6] During isometric contraction, myocardial fibers develop high tension rapidly and sustain it well during their contraction. In contrast, if the muscle shortens rapidly after the peak of isometric contraction has been reached, the tension falls off abruptly even though contraction continues. The drop in tension is increased as the rate and extent of shortening increases so that with extreme shortening, contractile tension approaches resting tension. The term "viscosity," as applied to this phenomenon, indicates that the loss of tension is probably due to "friction" within the myocardial fibers as the contractile elements are rearranged during a sudden change in length. Since the ventricles cannot eject blood without shortening of the myocardial fibers, this drop in tension assumes great importance in cardiac function.

The larger the diastolic volume, the less the myocardial fibers must shorten to eject a particular volume of blood. Both the rate and extent of myocardial shortening are reduced when the ventricles contract from large diastolic volumes. The loss of energy required to overcome internal friction (myocardial viscosity) is similarly diminished by diastolic expansion. Thus, the contractile tension, and the work accomplished per unit of energy released (efficiency), are theoretically increased when diastolic volumes are large and the initial length of myocardial fibers is relatively great. If nervous or hormonal influences could reduce viscosity, this effect would be another mechanism for direct neural control of "contractility."

The Law of Laplace.—The pressure within a hollow cylinder is maintained by tension in the walls. The amount of tension required to support a particular pressure is proportional to the radius of the cylinder.[4] The relation between the pressure (P), the wall tension (T) and the radius of the cylinder (R) is expressed by the formula: $P = \frac{T}{R}$ (the law of Laplace). A partially inflated, sausage-shaped rubber balloon illustrates the principle.[32] During the initial inflation, considerable pressure is required to produce any distention. When one portion of the balloon begins to distend, further inflation is much easier. At the stage illustrated in Fig. 5C, the inflated portion has a large radius; the wall is very tense and resists indentation. The portion of the balloon with a small radius supports precisely the same pressure, but the walls are relatively flaccid and readily compressed. In this case, the pressure is constant throughout the balloon and the wall tension is directly proportional to the radius.

If the left ventricle is considered a cylinder[25] surrounded by circularly oriented fibers (the deep muscle bundles), the tension in the myocardial fibers required to sustain a particular level of intraventricular pressure is greater when the ventricle is distended than when it is more completely emptied. If all myocardial layers developed the same tension, the inner layers of myocardium would be more effective in elevating intraventricular pressure than the outer layers.[22,25] If the myocardial fibers sustained the same tension during shortening, the intraventricular pressure could rise progressively as the ventricle emptied. Actually, the tension lost during myocardial shortening is compensated to an unknown extent by the decreasing radius described by the myocardial fibers as the chamber is emptied. The spiral muscle layers in the right ventricular wall describe a circle of very great radius and would have to develop extremely high tension to produce right ventricular pressures as high as the normal left ventricular pressure.

Interfascicular Tension.—Obviously the degree of myocardial shortening is not the same in different myocardial layers. The internal circumference of the left ventricular wall is the most reduced; the outer layers of circularly arranged fibers shorten to a less extent and the superficial spiral muscles shorten least of all. The various muscle layers are entwined and interconnected. If one layer contracts more than the other during systole, stress is applied to the connections between them.[22] This might compress the inner layers of the left ventricular wall as in Fig. 5D.[3] The various myocardial layers are oriented in different directions, schematically represented by a basket work in Fig. 5D. If the fibers in these two layers are connected together and all shorten simultaneously, tension will develop in the connections between them. These two forms of stress in the connections between muscle bundles have been termed "interfascicular tension."[22] The energy of contraction expended in placing these connections under tension is wasted so far as systolic ejection of blood is concerned. Assuming that minimal tension between layers occurs at or near the normal diastolic level, the greater the myocardial shortening, the greater the interfascicular tension. Obviously then, the lesser degree of myocardial shortening in the various layers, when the ventricles function at large diastolic dimensions, also diminishes the energy wasted in developing interfascicular tension.

Thus, the extent of systolic ejection depends upon the contractile tension which is developed and sustained by the myocardial fibers as the ventricles eject blood. Myocardial tension becomes progressively more effective in elevating the intraventricular pressure as the radius of the chamber is diminished. This beneficial effect is countered by the loss of tension during myocardial shortening due to myocardial viscosity and the energy wasted as interfascicular tension. There are undoubtedly other factors, but it is clear that the ventricles derive certain important mechanical advantages by functioning at large diastolic dimensions with relatively large quantities of systolic reserve blood. The author believes that the systolic reserve volume is normally greater than the diastolic reserve as indicated in Fig. 4. Encroachment upon the systolic reserve when the ventricles accomplish greater amounts of work requires increased myocardial "contractility."

Diastolic Filling

Ventricular filling occurs in three stages when the heart rate is sufficiently slow (Fig. 3A). A major portion of inflow occurs during the early diastolic period, beginning immediately after completion of systole. The rapid filling phase ends with a relatively abrupt transition to a period during which inflow is slow or stopped (diastasis). Finally, a variable increment of blood enters the ventricle during atrial systole just before the succeeding ventricular contraction. Since space is limited, the discussion will be largely restricted to the factors which affect ventricular filling during early diastole. The volume of blood in the ventricles during diastole is determined by two principal factors: (a) The effective filling pressure and (b) the resistance to distension offered by the ventricular walls (Table I). The rate of filling is determined by the atrioventricular pressure gradient and the resistance to distension. The "effective" filling pressure refers to the combined action of intraventricular pressure and the distending influence of the subatmospheric intrathoracic pressure. If the pressure within the ventricles and central veins and the fluctuating intrathoracic pressure are considered to remain within fairly narrow limits, ventricular resistance to stretch (distensibility) is the principal limiting factor to be discussed. Distensibility is defined as the diastolic ventricular volume per unit of effective filling pressure. If the ventricle accepts a greater quantity of blood without an increase in effective filling pressure, the distensibility is increased.

Myocardial Distensibility.—When relaxed myocardium is progressively stretched, the resting tension increases, gradually at first and more rapidly as the elongation continues. The relation between resting length and resting tension was illustrated by Frank[5] as a single curve, which implies that the diastolic volume is determined by the diastolic filling pressure (e.g. distensibility is constant). Actually there is direct evidence that epinephrine increases ventricular distensibility,[15,17,24,29] summarized by Wiggers[29] as follows:

> "Restated, the diastolic volumes of the ventricles can be increased beyond their normal capacity (1) by an increase in initial pressure overcoming the inherent tendency of the ventricles to resist stretching or (2) by a reduction in this inherent power of the ventricles to resist stretching, i.e., by a reduction in their tonus. Under normal conditions the former mechanism operates; under epinephrine the latter mechanism not only enters, but predominates."

A similar conclusion was derived from measurements of left ventricular area and effective filling pressure in intact, anesthetized dogs.[24] If the diastolic volume increases without a corresponding increase in effective filling pressure, the length-tension relationship for resting myocardium must be represented by a family of curves or a range within which the myocardium functions under varying physiological states. These observations indicate that circulating epinephrine or sympathetic discharge releasing epinephrine-like substances within the ventricular walls can increase both myocardial distensibility and contractility to provide greater stroke volume by both greater diastolic filling (without increased filling pressure) and greater systolic ejection. The fact that the systolic and diastolic diameters tended to change in the same direction during exercise and startle in intact unanesthetized animals (Fig. 3) cannot be rationalized effectively with existing data.

Myocardial Viscosity.—Relaxed myocardial fibers respond to a sudden stretch by a marked increase in tension.[12] Thus, the internal friction within myocardial fibers opposes any sudden change in length, either stretching or shortening (Fig. 5B). At first sight, ventricular distension should be markedly impeded during early diastole when, in fact, the filling is extremely rapid. The sudden transition between rapid filling and slow filling might be due to myocardial viscosity, but this would mean that sudden stretch of myocardium does not occur until filling is largely complete. This possibility will be considered in relation to the effects of interfascicular tension *(vide infra)*. Diminished myocardial viscosity would facilitate diastolic filling and could be another mechanism for neural and hormonal control of stroke volume.

The Law of Laplace.—At the end of systole, the radius described by the myocardial fibers is minimal for that particular cycle. As ventricular filling begins, the effective filling pressure required to overcome myocardial resistance to stretch would be greater than at any other time during the cycle. Actually the filling rate is greatest, and the effective filling pressure is often minimal, during early diastole. It seems clear that little or no resistance to distention is offered by the myocardium during early diastolic filling. The apparent paradox between the early rapid filling, and the theoretical resistance to distention offered by both myocardial viscosity and the relation $P=\frac{T}{R}$ may be at least partially resolved by considering the interfascicular tensions developed during systolic contraction.

Interfascicular Tension.— During ventricular systole, the inner layers of the myocardial bundles are probably under compression and the connections between the various layers under tension. The energy of contraction which is dissipated in the form of interfascicular tension acts to impede systolic ejection, but is stored as potential energy until the ventricles relax. At the beginning of diastole, this potential energy is rapidly released, acting to restore the ventricular chambers toward their diastolic dimensions. Thus, interfascicular tension acts to facilitate rapid filling during early diastole and resistance to distention may be minimal during this phase. If this is true, active stretch on the myocardium due to the effective filling pressure may not begin before the transition between the rapid filling and the slow filling phases. On this basis, more complete systolic emptying would be expected to promote more rapid early filling so that tachycardia would be less likely to encroach on the rapid filling period. This prediction has not yet been confirmed.

Summary

The heart is generally believed to respond to an increased pressure or volume load by distending to greater diastolic volume. This concept is based on experiments conducted on anesthetized thoracotomized dogs in which the heart is characteristically much smaller than under normal conditions. Direct recordings of left ventricular diameter of intact unanesthetized animals demonstrate that during exercise, diastolic diameter did not usually increase. Instead, the systolic diameter appeared to be reduced. Both diastolic and systolic diameters are increased after exercise and after an unexpected loud noise. On the basis of these experiments, it is proposed that stroke volume can be increased by utilization of either the diastolic reserve volume or the systolic reserve volume. Rapid changes in ventricular diameter from cycle to cycle suggest direct effects of neural reflexes on myocardial "distensibility" and "contractility." Some of the factors which influence

ventricular "distensibility" and "contractility" were considered with particular emphasis on possible mechanisms for neural control of stroke volume.

References

1. Bing, R. J.; Heimbecker, R., and Falholt, W.: An estimation of the residual volume of blood in the right ventricle of normal and diseased human heart *in vivo*. Am. Heart J., 42:483-502 (Oct.) 1951.
2. Buchtahl, F.; Kaiser, E., and Knappeis, G. G.: Elasticity, viscosity and plasticity in the cross striated muscle fibre. Acta physiol. Scandinav., 8:16-37 (Sept.) 1944.
3. Burch, G. E.; Ray, C. T., and Cronvich, J. A.: The George Fahr Lecture. Certain mechanical peculiarities of the human cardiac pump in normal and diseased states. Circulation, 5:504-513 (April) 1952.
4. Burton, A. C.: On the physical equilibrium of small blood vessels. Am. J. Physiol., 164:319-329 (Feb.) 1951.
5. Frank, O.: Zur dynamik des herzmuskels. Ztschr. f. Biol., 32:370-437, 1895.
6. Gasser, H. S., and Hill, A. V.: The dynamics of muscular contraction. Proc. Roy. Soc. London, s. B. 96:398-437 (Aug.) 1924.
7. Goodall, McCh.: The presence of noradrenaline, adrenaline and an unknown sympathicolytic factor in cattle heart. Acta physiol. Scandinav., 20:137-152 (Mar.) 1950.
8. Günther, B., and Landis, E. M.: Cardiac resistance to flow; pressure-flow relationships in quiescent and beating turtle heart. Am. J. Physiol., 169:412-422 (May) 1952.
9. Kjellberg, S. R.; Lonroth, H.; Rudhe, U., and Sjöstrand, T.: The relationship between the heart volume and the blood volume and its physiological and pathological variability. Acta med. Scandinav., 140:446-453 (Fac. VII) 1951.
10. Kjellberg, S. R.; Rudhe, U., and Sjöstrand, T.: The amount of hemoglobin and the blood volume in relation to the pulse rate and cardiac volume during rest. Acta physiol. Scandinav., 19:136-145 (Dec.) 1949.
11. Liljestrand, G.; Lysholm, E.; and Nylin, G.: The immediate effect of muscular work on the stroke and heart volume in man. Scandinav. Arch. Physiol., 80:265-282, 1938.
12. Lundin, G.: Mechanical properties of cardiac muscle. Acta physiol. Scandinav., 7:(Suppl. 20) 1-86, 1944.
13. Meek, W. J., and Eyster, J. A. E.: Cardiac size and output in man during rest and moderate exercise. Am. J. Physiol., 63:400-401 (Feb.) 1923.
14. Nylin, G.: On the amount of, and changes in, the residual blood of the heart. Am. Heart J., 25:598-608 (May) 1943.
15. Opdyke, D. F.: Effect of changes in initial tension, initial volume and epinephrine on ventricular relaxation process. Am. J. Physiol., 169:403-411 (May) 1952.
16. Patterson, S. W.; Piper, H.; and Starling, E. H.: The regulation of the heart beat. J. Physiol., 48:465-513 (Oct.) 1914.
17. Rabb, W., and Humphreys, R. J.: Secretory function of sympathetic neurones and sympathin formation in effector cells. Am. J. Physiol., 148:460-469 (Feb.) 1947.
18. Rabb, W., and Lepeschkin, E.: Heart "sympathin." Circulation, 1:741-752 (April) 1950.
19. Rabb, W., and Maes, J. P.: Effect of sympathectomy without and with adrenal inactivation on the concentration of epinephrine and related compounds in various tissues. Am. J. Physiol., 148:470-477 (Feb.) 1947.
20. Rushmer, R. F.: Continuous measurements of left ventricular dimensions in intact, unanesthetized dogs. Circulation Res. In press.
21. Rushmer, R. F.: Shrinkage of the heart in anesthetized thoracotomized dogs. Circulation Res. In press.
22. Rushmer, R. F.; Crystal, D. K., and Wagner, C.: The functional anatomy of ventricular contraction. Circulation Res., 1:162-170 (Mar.) 1953.
23. Rushmer, R. F., and Crystal, D. K.: Changes in configuration of the ventricular chambers during the cardiac cycle. Circulation, 4:211-218 (Aug.) 1951.
24. Rushmer, R. F., and Thal, N.: Factors influencing stroke volume: a cinefluorographic study of angiocardiography. Am. J. Physiol., 168:509-521 (Feb.) 1952.
25. Rushmer, R. F., and Thal, N.: The mechanics of ventricular contraction. Circulation, 4:219-228 (Aug.) 1951.
26. Shaw, F. H.: VI. The estimation of adrenaline. Biochem. J., 32:19-25 (Jan.) 1938.
27. Sjöstrand, T.: Regulatory mechanisms relating to blood volume. Symposium on recent advances in cardiovascular physiology and surgery. Minnesota Heart Association. (Sept. 14) 1953.
28. Wiggers, C. J.: Determinants of cardiac performance. Circulation, 4:485-495 (Oct.) 1951.
29. Wiggers, C. J.: Studies on the cardiodynamic actions of drugs. J. Pharmacol. & Exper. Therap., 30:217-250 (Jan.) 1927.
30. Wiggers, C. J.: Some factors controlling the shape of the pressure curve in the right ventricle. Am. J. Physiol., 33:382-396 (Feb.) 1914.
31. Wiggers, C. J., and Katz, L. N.: The contour of the ventricular volume curves under different conditions. Am. J. Physiol., 58:439-475 (Jan.) 1922.
32. Wolf, A. V.: Demonstrations concerning pressure-tension relations in various organs. Science, 115:243-244 (Feb.) 1952.

REGULATORY MECHANISMS RELATING TO BLOOD VOLUME

(Continued from Page 15)

6. Kjellberg, S. R.; Lönroth, H., Rudhe, U., and Sjöstrand, T.: Acta med. Scandiav., 140:446-453, 1951.
7. Kjellberg, S. R.; Rudhe, U., and Sjöstrand, T.: Acta radiol., 31:113-122, 1949.
8. Kjellberg, S. R., Rudhe, U., and Sjöstrand, T.: Acta physiol. Scandinav., 19:136-145, 1949.
9. Kjellberg, S. R.; Rudhe, U. and Sjöstrand, T.: Ibidem, 19:152-169, 1949.
10. Lind, J.: Acta radiol. Suppl., 82, 1950.
11. Menely, G. R.; Segalaff, A., and Wells, E. B.: J. Clin. Investigation, 26:320-324, 1947.
12. Petrén, T.; Sjöstrand, T., and Sylvén, B.: Arbeitsphysiol., 9:376, 1936.
13. Sjöstrand, T.: Physiol. Rev., 33:202-228, 1953.
14. Taylor, H. L.; Erickson, L.; Henschel, A., and Keys, A.: Am. J. Physiol., 144:227-232, 1945.

SYSTEMATIC ERRORS IN FLOW DETERMINATIONS BY THE FICK METHOD

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IN MANY PHASES of cardiovascular investigation, it is essential to make measurements of the rate at which blood flows through an organ. The mounting demand for precision in quantitative physiology causes us to develop new methods of measurement, and to re-examine the accuracy of long-accepted methods. For many years the application of the Fick principle has provided the classical indirect means of measuring blood flow. Other methods have been judged on the basis of their agreement with Fick measurements. Careful workers have recognized the importance of "the steady state" as a prerequisite to reliable determinations. However, either "the steady state" has escaped rigorous definition, or at least the definition has not been widely appreciated.

Until recently, apparently few doubts have been entertained regarding the validity of the conventional application of Fick's law. Lately provocative articles have appeared in the literature which should stimulate a reconsideration of the assumptions underlying the Fick method of determining cardiac output and of the justification for making these assumptions.[2,7]

First, several questions can be asked concerning the usual application of the Fick principle; second, the lines can be indicated which future experimentation might take to throw light on unanswered questions. The discussion may proceed by examining a statement of Fick's law in several forms, and by describing a model to which the law unambiguously applies. The difficulties that obtain in applying the principle to the real physiological system will be demonstrated by considering the discrepancy between the ideal model and its physiological counterpart.

The law of Fick states that the rate of uptake of a reference material by an organ must equal the net rate at which the material is removed from the organ by the blood flowing through it (other paths of removal being excluded). The statement is conveniently expressed as an equation:

$$\frac{dM}{dt} = F_oC_o - F_iC_i,$$

where $\frac{dM}{dt}$ = rate of uptake of reference material by organ in question
F = rate of blood flow
C = concentration of reference material in blood
Subscripts
$_i$ and $_o$ refer respectively to blood flowing into or out of the organ

This form of the law is applicable to infinitesimal intervals of time, and since means are not available for instantaneous measurement of uptake and blood contents, the equation as it stands is not in a usable form. Uptake and blood contents are measured over an extended period. Thus, in practice, the integrated form of the equation (second equation) is employed.

$$M = \int^{T} (F_oC_o - F_iC_i)\, dt$$

If the rate of flow, F, and the concentrations of the reference material in the blood supplying and draining the organ are independent of time: i.e., constant during the interval of observation, T, these terms may be taken from under the integral sign and the equation reduced to the following simple statement. During the observation the uptake of reference material is equal to the product of: the rate of blood flow, the arteriovenous difference in the concentration of the reference material, and the time interval involved. The equation is then:

$$M = F(C_o - C_i)T$$

In order to arrive at this simplification it was necessary to assume that the flow and concentration were constant. Also, it may be emphasized that the assumption is implicitly made that the measured uptake reflects the net amount of reference material added to the blood flowing through the organ. This is the familiar form of Fick's law which is used for the calculation of flow from these data. In the restricted area of the appli-

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cation of the law of Fick to the determination of flow, it might be asserted that the "steady state" is one in which this usable expression of the principle may be applied without error.

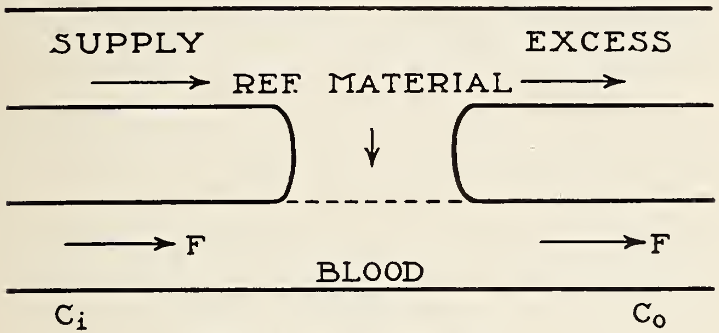

Fig. 1. Idealized organ.

In Figure 1 is shown a simple model which would strictly conform to the restrictions imposed by the assumptions made in deriving the last equation. The essential features are: (1) vessels supplying and draining the organ, (2) a supply of reference material to the organ, (3) a "dead space" or reservoir in the organ of fixed volume both for blood and for the reference material, and (4) constant blood flow and blood concentrations during an observation.

In the actual case, for instance in determining cardiac output by the pulmonary transport of oxygen, it is obvious that these conditions are by no means fulfilled. The cardio-pulmonary system is much more complex than this simple idealized model. In principle, the physiological system is represented in Figure 2, which illustrates some of the pertinent details by which it differs from the simple model.

Under the influence of the cardiac cycle, the respiratory cycle, and from other causes, the lung volume and the pulmonary blood volume change. It is well known from the work of various investigators; for example Greene and Swanson,[3] and Heymans and Jacob,[4] that as a perturbation to the basic respiratory excursions, the mean lung volume undergoes considerable periodic fluctuation with a period equal to several respiratory cycles. Plethysmographic studies indicate the existence of cardiac, respiratory and relatively long term fluctuations in the volume of the extremities. These are consistent with the notion that the pulmonary blood volume undergoes periodic change.

Since in practice the oxygen uptake can only be measured at the mouth or nose, the variable buffer capacity for oxygen of the alveolar space and the pulmonary blood may interfere with the accurate short term measurement of the uptake as referred to the blood flowing to and from the lungs. This question has been raised by Fishman and his colleagues.[2] In order to avoid this type of error it is essential that the amount of oxygen within this buffer reservoir be the same at the beginning and at the end of the period of observation.

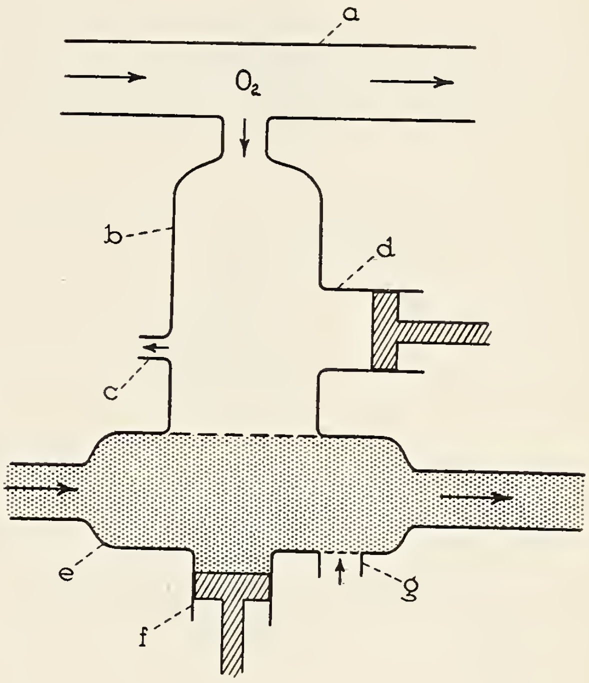

Fig. 2. Schematic representation of cardio-pulmonary system. (a) external airway, (b) total lung capacity, (c) route for direct nonvascular pulmonary, consumption of oxygen, (d) variable part of lung capacity, (e) central blood volume, (f) variable part of central blood volume, (g) drainage of venous blood to left ventricle, through Thebesian veins.

Recently, Drs. Visscher and Johnson have initiated consideration and discussion of another important aspect of the total problem.[7] They have pointed out that the usual procedure of sampling blood at constant rate may not yield a sample of the same oxygen content as the average content of all the blood which flows past the sampling site in the collection period. This undesirable situation can occur whenever both the rate of flow and the oxygen content of the blood, passing the sampling site, vary during the sampling procedure.

While this error is *per se* a sampling error, the circumstances of variable flow and concentration, which give rise to it, may also impart an error directly referable to the discrepancy between the ideal model and the real system. For, the buffer reservoir of the central circulation and lung volume may temporarily absorb, or give up, oxygen, even without an alteration in its volume if the concentration of oxygen in the blood or gas in this space undergoes fluctuation. This factor could be expected to contribute a systematic non-random error in the period of changeover from one "steady state" to another, as for instance, in the transient period of adjustment to exercise or to an alteration in the inspired oxygen tension.

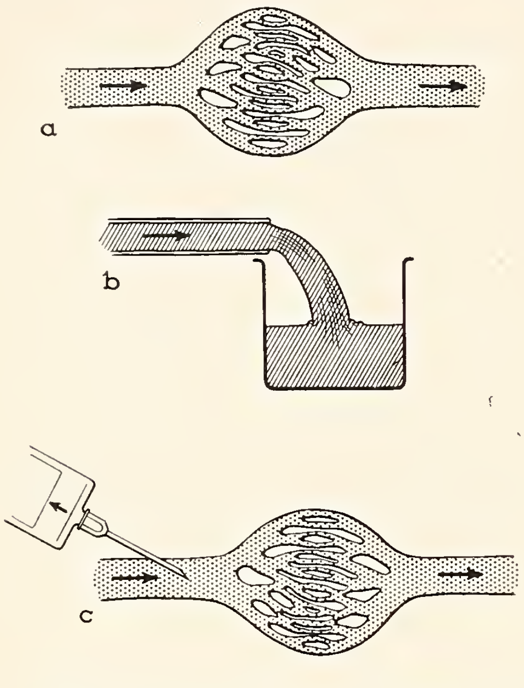

Fig. 3. Hypothetical and actual methods of sampling blood supplying an organ under investigation. (a) the organ with its artery and vein, (b) the hypothetical collection of blood for an arterial sample, (c) the actual collection of an arterial sample in a syringe.

To return to the sampling error, its basis can be understood by considering in detail the relation between the collection of the sample and the flow of blood and of oxygen past the sampling site. In Figure 3a, the organ in question is illustrated with the artery and vein which serve it. Focussing attention on the blood in one particular vessel, the flow in the artery may be examined. If, without disturbance, the artery could be severed and the blood flowing through it caught in a beaker for the same length of time that the sampling procedure would ordinarily require, after this interval the beaker would contain the same volume of blood and the same amount of oxygen as otherwise would have entered the organ. This hypothetical procedure is shown in Figure 3b. This would be true regardless of variations in flow or oxygen content during the collection. The average content of the blood in the beaker, multiplied by the volume of blood, would equal the quantity of oxygen which would have entered the organ in the collection interval. It is precisely this concentration that is required in the calculation of blood flow from the simplified equation expressing Fick's law for extended periods of time.

In actual practice a needle or a catheter is inserted in the artery and blood is withdrawn at approximately constant rate (Fig. 3c). In the presence of variable flow and oxygen content, the average content of the sample thus obtained might not be the same as that of the blood in the beaker. The discrepancy between the two occurs due to the fact that the flow into the syringe, , *is not* always proportional to the flow in the vessel, F. The syringe sample would yield the *time average concentration,* whereas the sample from the beaker would have the *volume average concentration.* The time average concentration is equal to the quantity of oxygen in the syringe divided by the volume of blood in the syringe. The equivalent equation is:

$$\overline{C^t} = \frac{\int^T \mathcal{F} C dt}{\int^T \mathcal{F} dt} = \frac{\int^T C dt}{T}$$

where

$\overline{C^t}$ = the time average oxygen content
$\mathcal{F}$ = constant rate at which the syringe is filled
C = instantaneous oxygen content
t = interval of observation.

The volume average concentration is equal to the quantity of oxygen in the beaker divided by the volume of blood in the beaker. Or expressed as an equation:

$$\overline{C^v} = \frac{\int^T F C dt}{\int^T F dt}$$

where

$\overline{C^v}$ = volume average concentration
F = instantaneous rate of flow from the artery.

An exact expression can be obtained for the difference between these two average concentrations. Let the flow, at any instant, be represented as the sum of the average flow over the entire interval of observation, plus the instantaneous deviation from the average. Likewise, let the oxygen concentration at any instant be similarly represented as the sum of the average (this will be the time average), and the instantaneous deviation from the average. Thus:

$$F = \overline{F} + \delta F$$

$$C = \overline{C^t} + \delta C$$

By substituting these expressions for flow and concentration in the formulas for the time and volume average concentrations, the difference between these two quantities, $\triangle C$ is obtained.

$$\triangle C = \overline{C^t} - \overline{C^v} = -\frac{1}{\overline{F}T}\int^{T} \delta C \cdot \delta F dt.$$

This formula is quite general and it is applicable to any specific situation involving a discrepancy between time and volume average concentrations. Unfortunately, from a practical point of view, this result indicates that detailed knowledge regarding the flow and the concentration of the reference material is needed in order to estimate the magnitude of the error incurred by using time average rather than volume average concentrations.

In order to illustrate the meaning of this expression, a calculation has been made for a hypothetical case of sinusoidal variations of the same frequency in concentration and flow. Figure 4 shows by means of a graph the assumed patterns of flow and concentration. The abscissa is proportional to time, the ordinate scale at the left gives the instantaneous flow corresponding to the upper line, while the ordinate scale at the right represents the instantaneous value of the concentration. The amplitudes are designated A and B and the phase angle by which the concentration wave leads the flow wave is given as α. For this case:

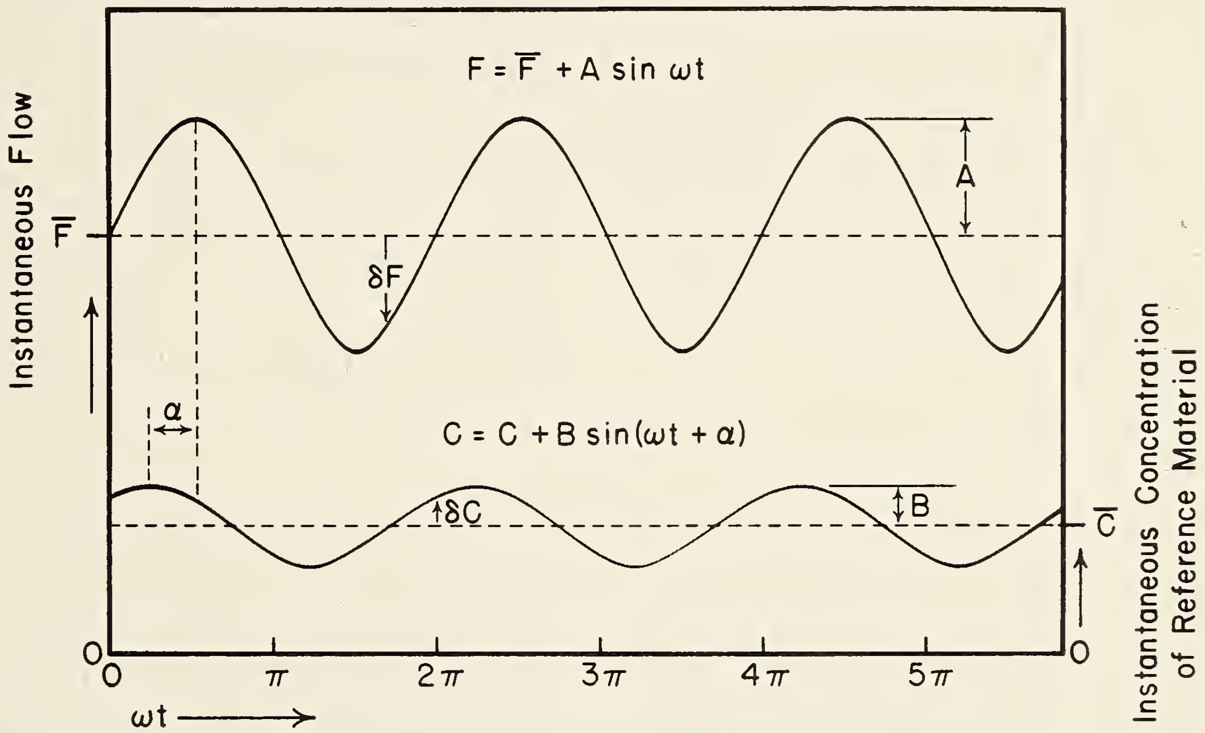

Fig. 4. Assumed sinusoidal variations in flow and concentration.

$$\triangle C = -\frac{AB\cos\alpha}{2\overline{F}}.$$

Since the discrepancy between the time and volume average concentrations is proportional to the cosine of α, it is evident that the time average concentration may be larger, smaller or the same as the volume average concentration depending on the value of α.

A general expression can be obtained for the fractional error in the calculated flow or output by considering the error due to the use of time average concentrations for both arterial and venous blood. The correct formula for average flow is:

$$\overline{F} = \frac{M}{T(\overline{C_o^v} - \overline{C_i^v})}.$$

The incorrect formula utilizing time average concentrations is:

$$\overline{F}^{*} = \frac{M}{T(\overline{C}_o^{\,t} - \overline{C}_i^{\,t})}.$$

The fractional error in flow is obtained by combining these expressions with $\triangle C$ as defined above.

$$\frac{\overline{F}^{*} - \overline{F}}{\overline{F}} = \frac{\int^{T} (\delta F_o \cdot \delta C_o - \delta F_i \cdot \delta C_i)\, dt}{M + \int^{T} (\delta F_o \cdot \delta C_o - \delta F_i \cdot \delta C_i)\, dt}$$

Only the sampling error has been considered in arriving at this result. The last formula cannot of course be applied in practice. However, it may serve as a guidepost in estimating the limiting error of this type when some of the details of flow and concentration at the sampling sites are known.

Within the framework of these formal conclusions, it is of interest to examine some of the factors which may produce fluctuations in flow and concentration and some of those which tend to smooth out or otherwise annul the effects of the variations. Although insufficient data are available to assess the importance of the various factors, it is perhaps justified to speculate briefly concerning them.

Arborization is one of the cardinal features of the vascular system. Both a branching in the arterial tree, and a confluence in the venous system, are possible sources of difficulty as related to the subject of the sampling error. First, consider a confluence in the venous system. If the oxygen contents of the blood flowing in the tributary vessels are different, any variations in the relative contributions of the small veins to the flow in the main vessel will produce fluctuations in the oxygen content of blood flowing in the larger vessel. Thus it is possible that such a site is the origin of concentration differences in the venous circulation. This situation is apt to occur in the case of the vena cavae and coronary veins supplying blood to the right atrium.

At a bifurcation in the arterial tree, the same process in reverse may be operative. If concentration waves are present in the parent artery, any time variation in the relative flow into the daughter vessels would result in differences in the average saturations of the blood flowing in the two smaller arteries. In this way, it is seen that a sample withdrawn from a peripheral artery might not be equivalent to one obtained from the main arterial stream.

In addition to this effect, systemic arterial blood may be subjected to variations in oxygen content in the process of becoming arterialized in the pulmonary capillaries. Information is lacking regarding fluctuations of the frequency of the heart rate, but in earpiece oximetry, undulations in oxygen saturation of respiratory frequency are sometimes observed even in normal subjects at rest breathing air. This finding is substantiated in the theory of respiratory gas exchange which indicates that substantial variations of this frequency may occur in arterial gas contents.[1,6]

Turning now to the question of the inconstancy of flow, recently, careful dynamic flow measurements made in animals have been reported. For example, McDonald[5] presents data obtained by high speed cinematography of the flow pattern in the abdominal aorta of the rabbit. He made simultaneous measurements of the velocity and pressure just distal to the origin of the renal arteries. His data clearly show that at the site of observation the pressure and velocity waves differ both in phase and form. Thus, in general the sampling error could not be avoided by merely allowing the variable pressure head to deliver the sample. Elimination of the error would require collection at a rate always proportional to the flow at the sampling site.

Thus far only those factors have been considered which emphasize the sampling error. There are, however, several ways in which the causes for error are dissipated. As the blood moves away from the source of time variations in composition, it travels with different velocities in the various lamina. The more peripherally the blood moves, the less pronounced the inequalities in composition become due to longitudinal mixing. Also, for instance, in the case of systemic arterial fluctuations in oxygen content, the differences in the path length from various pulmonary capillaries to the sampling site, tend to randomize the initial inhomogeniety.

A final point is worthy of mention. Previously attention was drawn to the importance of the separation in phase between undulations in flow and concentration in determining the magnitude and direction of the sampling error. This difference in phase is principally determined by two factors: (1) the velocity with which the flow and concentration waves travel (the concentration wave moves with the mean blood velocity while the flow wave moves considerably faster at approximately pulse wave velocity); (2) if the basic causes for the existence of these waves were of constant frequency, then the phase angle between the two would be constant at any particular site. However, the periods of physiological oscillations including the heart beat and the respiratory excursion are never precisely constant. Thus the phase angle even at one site would vary. This factor would also aid in randomizing the sampling error and hence in reducing its over-all effect.

Unfortunately, the status of this subject at present is decidedly uncertain. Whether there is appreciable error in the usual applications of the Fick principle is a matter of conjecture. Obviously, there are possible causes for error. An answer to the questions that can be raised may come from either of two directions. It would be helpful to compare the results of Fick measurements with a reliable direct method of determining blood flow at various levels of activity and with different inspired gas mixtures. Direct assessment of the significance of the sampling error can only be made on the basis of simultaneous dynamic measurements of flow and oxygen content at various places in the vascular system.

References

1. Dubois, A. B.; Britt, A. G., and Fenn, W. O.: J. App. Physiol., 4:535-548, 1952.
2. Fishman, A. P.; McClement, J.; Himmelstein, A., and Cournand, A.: J. Clin. Investigation, 31:770-781, 1952.
3. Greene, J. A., and Swanson, L. W.: Arch. Int. Med., 61:720-725, 1938.
4. Heymans, C., and Jacob, J.: Arch. internat. de pharmacodyn. et de therap., 75:382-391, 1948.
5. McDonald, D. A.: J. Physiol., 118:328-339, 1952.
6. Stow, R. W.: Thesis, University of Minnesota, 1952.
7. Visscher, M. B., and Johnson, J. A.: J. App. Physiol., 5:635-638, 1953.

CONGENITAL CARDIAC DEFECTS—REFERRALS AND RECOMMENDATIONS

The general practitioner will not see many cases of congenital heart defect in his practice. When he does see one he is confronted with these questions: Is the murmur indicative of a serious defect? Is this an operable condition? Should the patient be referred for consultation? What shall the parents be told? How much activity should the child be permitted?

Any cyanotic type of heart lesion is serious, but only certain types may be operated on at present with the expectation of good clinical results.

In general, intense cyanosis, or an enlarged heart or an abnormally shaped heart on fluoroscopic examination is indicative of a serious congenital heart defect. These cases should be referred for consultation to a cardiologist familiar with hear disease in childhood, and the decision as to the necessity for special diagnostic procedures should be left to him. Parents should be told of the presence of a congenital cardiac defect, informed that it is not a disease, and a definite effort should be made to explain to them the nature of the defect, serious or otherwise. If informed by the family doctor whom they know and trust, the parents will be far less upset than if they learn of the defect through a routine school or club examination. The general practitioner is advised not to bring up the necessity for surgery before consultation unless he is sure of the exact diagnosis.

If the family physician plans to refer the child to a specialist, he has to be on his guard when he discusses his diagnosis with the parents or mentions the possibility of surgery to them. Because he wishes to persuade the parents to consult a specialist at once, he may be tempted to make a strong statement, but he will do much harm to the patient and the family if he tells them bluntly that the child has a heart condition and must have an operation or he will die. Cases of congenital heart defects are rare in his practice and the family physician often has no means of verifying the diagnostic impression gained from the history and the physical findings. There is always the chance that the diagnosis may be wrong and, even if it is correct, operation may not be indicated or may not be feasible.

Younger children with congenital heart defects rarely need to be limited in their daily activities by the physician or parents, as they will automatically limit themselves; this is especially true of children with cyanotic lesions. They need not be told to sit down and rest; the development of dyspnea will make them rest of their own accord. Older children with congenital heart defects, resulting in an enlargement of the heart, should not be permitted to take part in competitive sports, since their pride may force them to continue beyond their physical capacities.

Children with minor cardiac defects, such as a small ventricular septal defect, may be as active as they wish. Far greater harm will be done to the child if he is continually admonished to "be careful—go slowly" than if he is left on his own. These children do not need monthly physical check-ups except under special circumstances. School health slips should be carefully filled out, and emphasis laid on the degree of activity permitted; otherwise the teachers will tend to refuse to permit the child to join in any play.

If tooth extractions or operations such as tonsillectomy are indicated, they need not be avoided. Anesthetics may be administered safely to these children. Prophylactic medication should be given before and after the surgical procedure to avoid subacute bacterial endocarditis to which these children are especially susceptible.

The child with a congenital cardiac defect has been handicapped by nature. Every effort should be made to avoid creating an additional handicap by unnecessary restrictions. He should be allowed to live as active a life as possible.—From "Diagnosis of Congenital Cardiac Defects in General Practice," American Heart Association.

THE PHYSIOLOGY OF CONGESTIVE FAILURE OF THE CIRCULATION

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Augusta, Georgia

I AM GREATLY HONORED to be asked to come to the University of Minnesota to deliver a talk on the Physiology of the Circulation. It is like carrying coals to Newcastle because work in this field has been very important in both the Rochester and the Minneapolis branches of the University.

I am particularly pleased that this lecture is dedicated to Dr. Fahr. For many years it has been my pleasure to look for him in the Federation meetings, to greet him as a friend, and then proceed to reap the riches of this great mind. I salute him as a great teacher, one who delights to share his very sound ideas and is ever patient and helpful to his younger associates.

My topic, the Physiology of Congestive Failure of the Circulation, is one about which there is much debate at the present time. Particularly is this true about edema. Our textbooks would have it that a high venous pressure will *ipso facto* result in edema by raising capillary pressure and augmenting filtration of fluid from the vascular system into tissue spaces. This oversimplified idea is attributed to Starling—though, in his original description of "Physiological Factors in the Causation of Dropsy,"[28] he stresses the unbalance between ingested water in excess of that excreted by the kidney. This produces hydremic plethora, which increases venous and capillary pressure. The resulting excessive transudation, according to Starling, leads to dropsy.

Since edema is constituted of large quantities of interstitial saline solution, we must agree with Starling that its accumulation of necessity results from intake of water and salt being greater than output. In heart disease it would seem that this unbalance is due to lessened renal output rather than to increased intake.[23]

Three ideas are ascendent as to the cause of renal retention of salt and water: (1) pre-renal deviation, (2) alteration of renal hemodynamics, and (3) hormonal disturbances. It may be that the adrenal gland or the pituitary gland secretes the key hormone.

In thinking of pre-renal deviation of salt and water, one meets the suggestion that the fluids are sequestered in the interstitial spaces so that they cannot get to the kidney. The cause is said to be high venous and capillary pressure resulting in transudation which not only causes the accumulation of dropsical fluids but also prevents their return to the bloodstream and kidney.

However, one does not need a high venous pressure in order to sequester fluids in the interstitial spaces. When one drinks a glass of water, or submits to an infusion, the fluid passes at once out into the interstitial spaces, and expands either the total body water (H_2O, D_2O) or the extracellular fluid (saline). It does this at a very rapid rate and with no measurable increase in venous pressure. Extra salt water is all that is needed to increase the extracellular fluid volume, and the reduction of this fluid volume is the simple result of getting rid of the salt water. This happens in heart disease by bed rest, digitalis, or diuretics in spite of a venous pressure persisting above normal levels. It happens in nephrosis spontaneously with the venous pressure normal during the accumulation and disposal of the dropsy.

The effect of increasing renal venous pressure upon sodium resorption is a temporary sodium retention when a ligature is placed around the cava just above the renals,[18] but this is permanent if the constriction is made above the liver,[5,27] indicating that renal venous pressure *per se* is not the key stimulus to edema formation. Temporary oliguria with sodium retention also results from exercise,[30] venous congestion of the extremities,[29] and pressure on the abdomen.[2] This last has been looked upon as illustrating the effect of increased venous pressure[2,26] but, since the kidney and vena cava are subject to the same ambient pressure, it is hard to see how the effective renal venous pressure can be increased by this maneuver. The ef-

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The experimental work outlined in this paper is a co-operative venture of the Departments of Physiology and Thoracic Surgery of this school. Participating were R. G. Ellison, R. W. Pickering, E. E. Hague, and J. T. Rucker, Jr. It was made possible through grants from the U.S.P.H.S., and from the Life Insurance Medical Research Fund.

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Dr. Hamilton is Professor of Physiology at the Medical College of Georgia.

fective arterial pressure, however, is lowered by the amount of the externally applied pressure as the arteries enter the abdomen.

Alternatively, retention of salt and water is said to be the necessary result of changes in renal hemodynamics. Thus, it is more difficult to get rid of sodium when there is lowered renal blood flow. Lowered blood flow lowers the filtration rate, ultimately, though the filtration fraction is increased. When the lowered filtrate formation displays sodium before the tubules in less amount than is their obligate reabsorptive function, a minimal amount of sodium is cleared from the body.[20]

As a general rule the glomerular filtration rate is reduced in congestive failure. However, in patients regaining compensation, sodium may be released with no consistent change in filtration rate. This is true whether the sodium load is high[3] or low[25]—whether filtration rate is measured by sodium thiosulfate,[3] inulin, or creatinine.[26] Tubular resorption of sodium (and water), through it is often more complete with low filtration, may be entirely independent of filtration.[3,7,12,13,25] This indicates that the resorptive mechanism is under direct physiological control and is not an obligate function of filtration rate. The fact that sodium excretion may be independent of filtration rate is further evidenced not only by the fact that subjects with low filtration rate will release sodium as they recompensate under the influence of bed rest, as well as after digitalization, and after the immediate influence of mercurial diuretics have passed off,[3] but also by the fact that circulatory handicap will lead to sodium retention even though filtration is normal.[5,13]

The evidence that the retention of salt and water is due to either prerenal deviation or to exclusively hemodynamic factors is not conclusive. Tubular function and the renal circulation seem, moreover, to be remote in their control from neurogenic factors—at least in the steady state.[30]

This leaves hormonal factors. The pituitary gland may play a key role. The posterior pituitary gland seems to be controlled by osmoreceptors[30] in its liberation of antidiuretic hormone promoting tubular resorption of water. Sight should not be lost of a possible mechanism involving the liberation of adrenocorticotrophic hormone to control the resorption of salt.

Conclusive evidence that the posterior lobe plays a role in the edema of circulatory failure is lacking. Presumptive evidence of its own activity is that the plasma sodium is often low in congestive failure and yet the patient suffers from chronic oliguria with antidiuretic substance in the plasma.[19] This would imply that some factor in heart disease stimulates the hypothalamo-pituitary mechanism to conserve water in greater amount than that needed to preserve the isotonic state.

That the adrenal does play a rôle is suggested by the fact that, when desoxycorticosterone acetate is given in excessive amounts to Addisonians, there result all of the signs of congestive failure of the circulation.[4] Remission of these signs occurs as soon as the drug is withdrawn.

It has also been shown by Davis and Howell,[5] that chronic passive congestion of the abdominal viscera, produced by partial obstruction of the thoracic inferior vena cava, caused retention of sodium and water. Upon adrenalectomy,[6] and the administration of cortisone, there was a prompt loss of sodium and water. Addition of desoxycorticosterone acetate in larger amounts than for ordinary maintenance, caused retention at a rate similar to that shown before adrenalectomy.

From the foregoing it would seem that a circulatory handicap may result in retention of salt and water with the formation of edema, and that the function of the adrenal gland, and possibly the pituitary, are important factors in this response.

In the hope of gaining insight into the physiology of the ascites of mitral stenosis, experiments outlined below were made on dogs. It was found, in brief, that when the mitral valve was gradually narrowed to a size comparable with that seen in severe clinical mitral stenosis, the animal became oliguric, excreted small amounts of sodium, and developed ascites. This occurred with normal venous pressures and could be reversed by withholding sodium or removing the adrenals, or simply by loosening the ligature. We have been unsuccessful so far in getting an animal to become ascitic, even with extreme stenosis, while afflicted with diabetes insipidus.[11,15,16,21,22]

The surgical operations described below were carried out by Dr. R. G. Ellison, whose work was a keystone in the investigation. Indispensable also were the constructive ideas and careful, painstaking work of R. W. Pickering, E. E. Hague, and J. T. Rucker, who took major responsibilty in the cardiorenal studies detailed herein.

For a long time it has been known that the mitral valve can be constricted sufficiently to give evidence of stenosis (early literature[9]), but without any clinical deterioration of the animal. It was found by Haddy and colleagues[14] that in animals in this condition, even though the mitral valve was constricted enough to increase the pulmonary venous pressure, the cardiac output and clinical condition remained normal. It has been our experience,[9] confirmed by Haddy et al,[14] that if an acute constriction of the mitral valve was produced, so that its aperture was of the order of 1 cm²/m², the animal died promptly of pulmonary edema.

We were interested in the occurrence of congestive failure after mitral stenosis. It seemed possible that the signs of this condition might appear if (1) the valve was not constricted at the time the animal was faced with the added handicap of deep anesthesia and post surgical shock, and (2) if the valve were constricted gradually and in such a manner that the constriction could be loosened when the signs of pulmonary edema appeared.

Operations

In order to do this, an operation was devised which is described elsewhere[9,10,11,15,16,21,22] and may be briefly outlined here. A special ligature was prepared, consisting of 28G stainless steel wire encased in 18G nylon tubing. Two 18G sharp needle tubes were pushed snug against the nylon tube and crimped in place on the wire.

The dogs were anesthetized under 10 mg/Kg. morphine and 20 mg/Kg. pentobarbital. The chest was opened aseptically by a rib spreading incision in the fifth interspace. The pericardium was opened and the ligature was threaded through the right auricle as near the septum and the floor as possible. (Fig. 1). It was drawn around the mitral ring and held in place by passing it under the epicardium in two or three places. The ends of the ligature were then drawn through a telescoping adjustable silver tube, one end of which was placed against the heart to hold this organ in place, and the other end between the fifth and sixth ribs and under the skin. The ligature was drawn snugly about the valve ring. The needles were cut away and a silver clip was bent around the ligature ends to hold them in place. The incisions were all closed with the usual surgical precautions.

When all wounds had healed and the animal seemed in good shape, the skin over the end of the tube was anesthetized, or light general anesthesia given, and the ligature ends exposed. They were then drawn tight enough so that a persistent (two or three minutes) weakening of the femoral pulse occurred. The ligature ends were re-clipped in the new location. This tightening was repeated at weekly intervals. The narrowing was 2 or 3 mm. above the mitral ring and left the leaflets functional. The mitral orifice was reduced in size comparably to that seen in severe clinical mitral stenosis (Fig. 2) and showed a murmur typical of the clinical disease (Fig. 3). The table indicates the pertinent facts relating size of the stenosis with the condition of the animal. The conclusion seems fair that when the mitral aperture is narrowed gradually it may be reduced to a very small size without producing acute pulmonary edema, whereas, as seen above, when it is constricted at the time of the operation, pulmonary edema and prompt exitus result even though the mitral orifice is

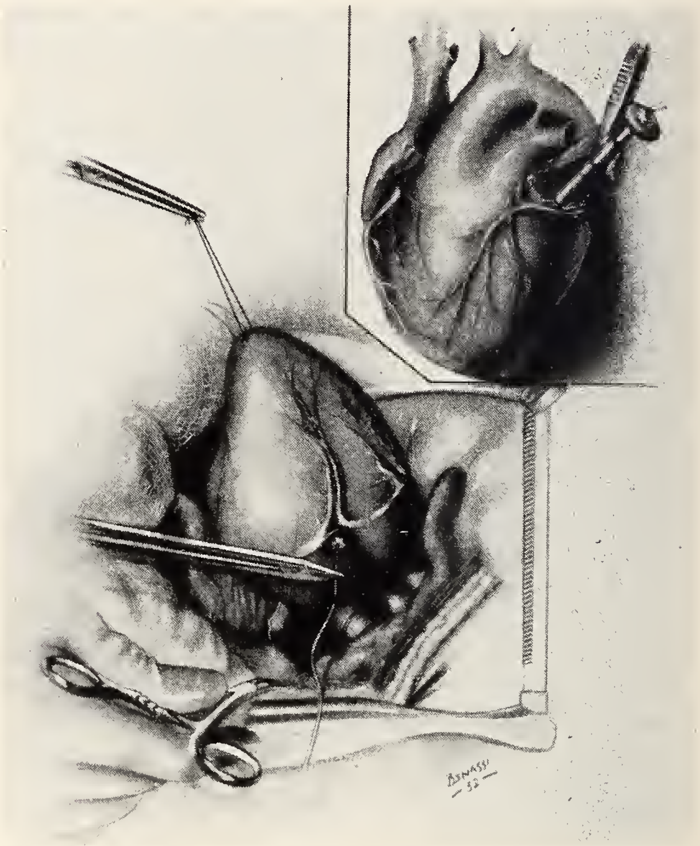

Fig. 1. Exposure of heart and placement of stenosing ligature (See text). Reprinted from Surgical Form.[10]

relatively large. The animals whose valves were gradually constricted developed a chronic state resembling, in some aspects, congestive failure of the circulation. After each tightening, there was an evanescent sodium retention and occasionally the temporary presence of ascites could be demonstrated. After a variable number of tightenings, urinary sodium excretion diminished to very low levels, and ascites accumulated prodigiously at a rate of sometimes 1.4 liters per week (Fig. 4). The ascites could be relieved by regular paracentesis or its formation prevented by administration of ion exchange resins and the animals kept alive from three to thirteen months.

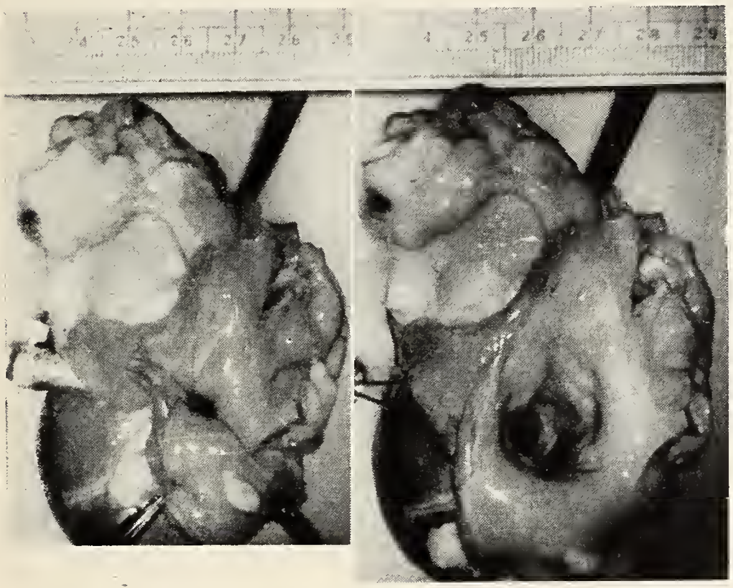

Fig. 2. The mitral orifice in mitral stenosis of controlled degree (right) as compared with a normal orifice. Reprinted from Surgical Form[10].

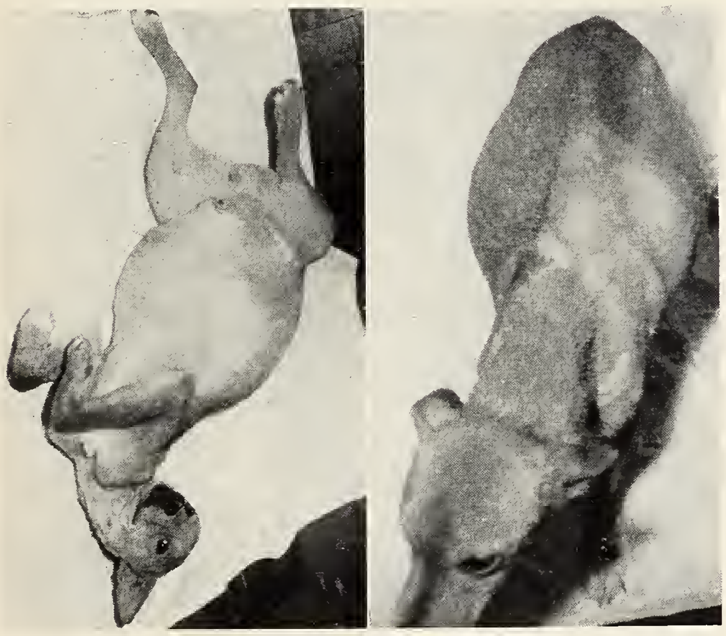

Fig. 4. Ascitic dogs with experimental mitral stenosis.

Except for those that were sacrificed, nearly all of the dogs died of terminal pulmonary edema. In two cases, a clot was found at autopsy causing an obstruction of mitral orifice further than that indicated by its anatomical measurement. The others may have had such a clot which was lost at autopsy. Another fact may explain the pulmonary edema. During the chronic course of the experiment, the plasma proteins gradually fell to low levels. This, by reducing the coloidal osmotic pressure, may have rendered the animals vulnerable to pulmonary edema at pulmonary vascular pressures not ordinarily dangerous.

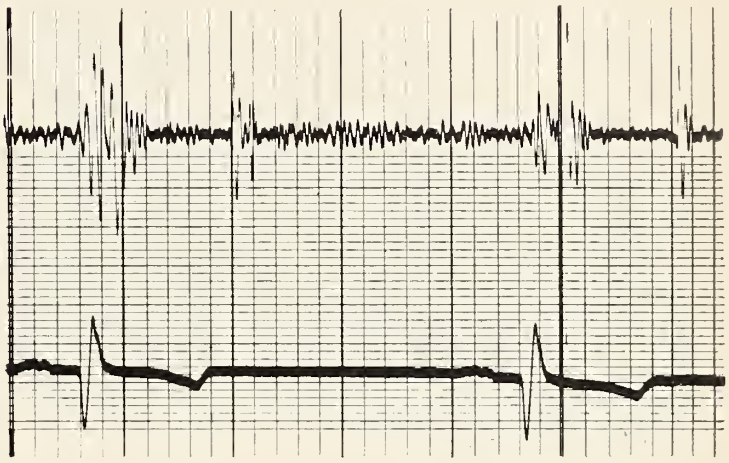

Fig. 3. Murmur typical of mitral stenosis from experimental dog, with simultaneous electrocardiogram.

Attention should also be called to the dog (H-9) whose pituitary stalk had been cut with probable injury to the hypothalamus. This animal had severe diabetes insipidus. It received a protracted series of seven tightenings, reducing the mitral orifice to a figure well below that associated with ascites in most of the other dogs. It may be that ascites is incompatible with diabetes insipidus.

The most obvious suggestion as to the cause of the ascites is that it results from simple filtration caused by a high venous (capillary) pressure. The venous pressure at the portals of the heart and the end-diastolic (filling) pressure in the right ventricle were not, however, above normal limits (Table I).

Not only was the central venous pressure normal, but there were none of the classical signs of chronic passive congestion. The spleen, kidney and liver were of normal appearance and size, grossly. The only microscopic abnormality was an obscure spotty hyperemia of the hepatic sinusoids and a complex convolution of the paralob-

TABLE I. CIRCULATORY AND RENAL EFFECTS OF EXPERIMENTAL MITRAL STENOSIS

Dog		No post-op. tightenings	Area mitral orifice mm^2/m^2	Cardiac index	Ascites duration days	Pressure mm Hg			Clearance cc/mm				Plasma Protein G %		Urinary Sodium mEg/day	
									Creatin		PAH					
No.	Wt. K					RA diast.	RV-PA syst.	PA diast.	Control	Failure	Control	Failure	Control	Failure	Control	Failure
59	—	4	65	—	150	0	38	15	—	—	—	—	—	—	—	—
74	14	1	21	—	90	0	28	10	—	—	—	—	—	—	—	—
75	15	2	21	—	60	3	40	16	—	—	—	—	—	—	—	—
76	14	2	47	2.2	20	2	34	12	—	—	—	—	—	—	—	—
82	13	3	35(clot)	2.6	30	0	30	—	31	22	119	73	—	4.1	51.0	1.7
83	15	3	55†	2.0	60	1	24	7	41	32	130	141	—	4.6	57.0	1.9
86	12	2	90	—	48	‡	—	—	48	10	120	50	—	3.4	34.0	1.5
94	8	1	—	3.2	365†	0	32	16	17	16	101	65	5.4	4.2	161.0	3.0
99	10	2	94	2.4	37	3	31	15	21	29	137	107	6.9	3.6	66.0	2.3
108	13	3	47(clot)	—	6	‡	—	—	29	35	164	154	5.7	5.4	42.0	1.1
110	13	1	86	2.0	30	0	26	8	27	18	137	97	6.1	4.0	4.2	0.7
111	15	4	99†	—	14#	‡	—	—	23	29	88	91	5.7	4.5	79.0	0.7
112	14	3	300*	—	12#	‡	—	—	35	33	157	147	6.2	4.8	41.4	0.4‡
H9	15	7	19	—	0	‡	—	—	33	—	128	—	6.2	—	61.3	—

†Tricuspid stenosis also. *Suture broken. ‡Jugular pressure normal. #Adrenalectomy.

Dog No.	Remarks
59	Terminal pulmonary edema
74	Terminal pulmonary edema
75	Interstetial edema cachexia TPE
76	Gangrene (sacrificed)
82	PE (due to mitral clot)
83	Gut hemorrhage after paracentesis
86	Cachexia TPE

Dog No.	Remarks
94	Alive and well. Ascites controlled by carboresin
99	Sacrificed
108	TPE (mitral clot)
110	Sacrificed
111	Sacrificed Adrenalectomy
112	Sacrificed Adrenalectomy
H9	Experimental diabetes insipidus—no ascites

ular venous channels. This affected only a few among many quite normal lobules. When ascites is produced by clamping the thoracic inferior vena cava and raising the abdominal venous pressure, the abdominal organs show clear signs of chronic passive congestion.[31]

On the other hand, when the dog was in the supine position the femoral venous pressure was definitely elevated. Nevertheless, edema of the feet and legs was not conspicuous.

The elevated femoral venous pressure is hard to explain except as the result of an increased pressure in the abdominal vena cava. The likelihood of this is enhanced by the fact that the superficial network of veins in the abdominal wall were greatly dilated, serving no doubt as a collateral drainage of the hind quarters.

To measure the pressure in the abdominal vena cava, a Cournand catheter was passed via the jugular vein, beyond the diaphragm, and the pressure was recorded. The abdominal caval pressure responded to manual pressure against the abdomen and, with the animal in supine position, varied from 12 to 14 cm. H_2O above the level of the catheter tip in the vena cava.

Now when the catheter was drawn into the thoracic part of the vena cava, the pressure fell to the normal venous pressure value (the height of the apex beat) or 6 cm. above the vena cava. There was a fall in pressure between that in the abdominal and thoracic inferior vena cava. It was caused, no doubt, by the fact that the contents of the distended abdominal cavity transmitted a pressure nearly equal to that in the vena cava, collapsing its walls about the contained blood, and contributing largely to the pressure measured in the great vein. As the blood approached the diaphragm the down stream pressure was reduced, the velocity increased, and the loss of lateral pressure (Bernoulli effect) caused further collapse of the vein around a fast jet of blood entering the inferior thoracic vena cava which was not subject to high outside pressure. Back pressure from this jet is responsible for the fact that pressure throughout the abdominal vena cava is slightly above that in the abdomen.[8,17] In the thoracic vena cava, blood pressure is not subject to conspicuous changes from abdominal manipulation as is the case in the abdominal vena cava.

The same experiment was performed on another dog whose belly was very tense. The pressure in the abdomen was 13 to 16 cm. H_2O of which 10 cm. could be accounted for by hydrostatic factors. The abdominal vena cava and abdominal pressures responded similarly to abdominal manipulation. The vena cava pressure (13 to 17 cm.) was not greatly different from that in the abdominal cavity, allowing for the hydrostatic weight of the abdominal contents. The difference between the thoracic and abdominal caval pressures was greater than in the first animal persisting in the natural position. Thoracic inferior vena cava pressure responded less than abdominal to pressure against the abdominal wall.

In whatever position, the caval pressure became nearly equal to that in the auricle as soon as the catheter tip passed the diaphragm, indicating that the ligature in no way interfered with the return of blood along the inferior vena cava, and that the high inferior vena cava and femoral pressures were due to the outside pressure from the abdominal contents.

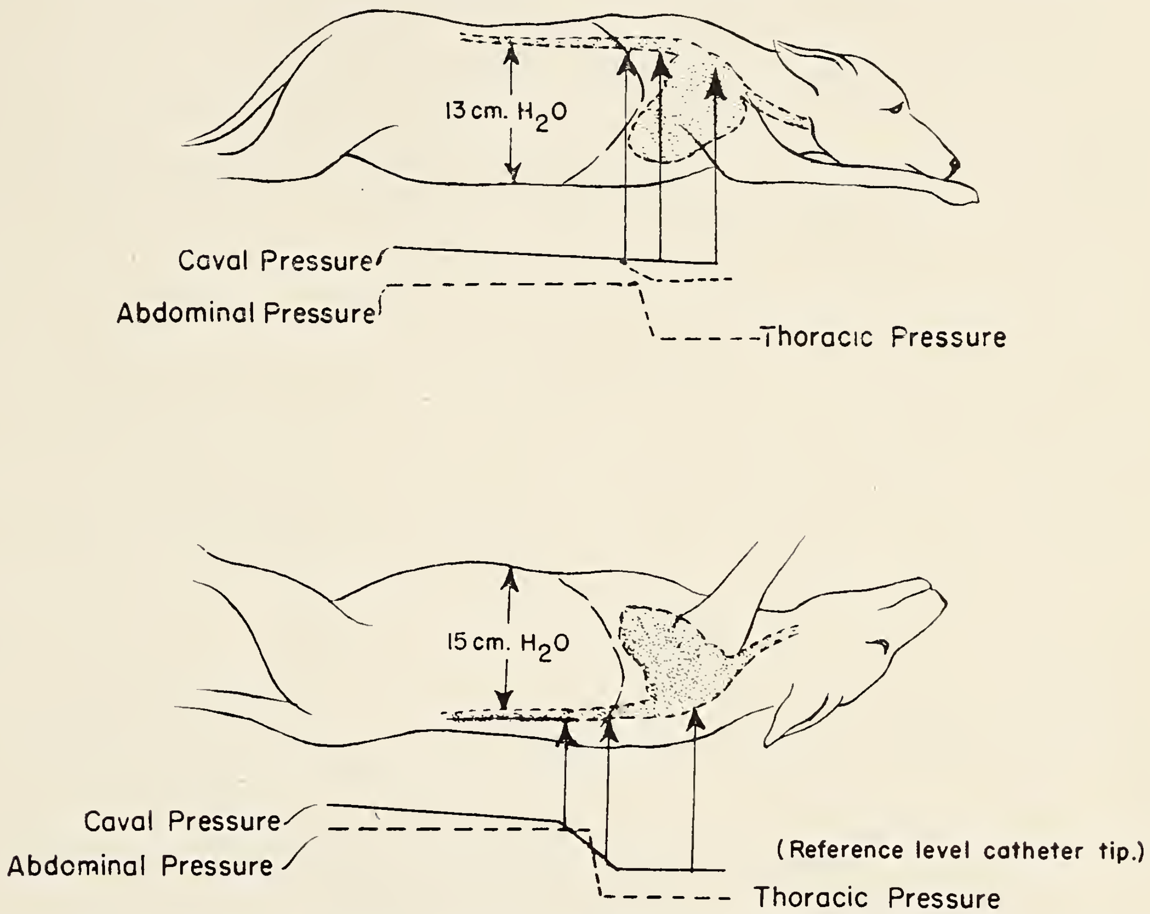

Fig. 5. Diagramatic representation of the fact that, though the pressure in the vena cava is high in ascitic dogs, it does not exceed by more than a few cm. H_2O the pressure in the abdominal cavity and, hence, cannot be responsible for the formation of ascites by simple filtration. Above animal in natural position; below, in prone position.

The table illustrates other hemodynamic findings. Although the cardiac filling pressure is low, the pulmonary vascular pressures are somewhat elevated. The cardiac output is at, or slightly below, the low normal level (pentobarbital anesthesia). It is hard to see anything in the hemodynamic picture to cause the ascites. The fact that the arteriovenous oxygen difference is wide, and the mixed venous O_2 tension low, may play a rôle.[3]

Without venturing an hypothesis as to how the direct effects of the ligature produce it, we have evidence that there are changes in renal function which occur with the development of ascites.

The renal blood flow and filtration rates were measured by the clearance of paraaminohippuric acid and of endogenous creatinine. The dogs were trained to lie quietly on the table while samples were being taken. As will be seen from the table, both of these measurements tended to be low in dogs with ascites, but rarely did they fall below the lowest measurements seen in normal control. The kidney seems to have handled blood and filtrate in an essentially normal manner. The reabsorption of sodium (and water) seem to be the renal functions which are most greatly changed. The renal tubule, in response to an unknown mechanism, has been stimulated to reabsorb sodium (and water) from the glomerular filtrate almost completely. Water and sodium seem to have been handled by the kidney in a coordinated manner because, whether the animal was gaining his ascitic pool or losing it, the plasma sodium remained within normal limits. Only the adrenalectomized animal had a low plasma sodium.

The chronically ascitic animal can be made to lose his pool of ascitic fluid. Attempts to cause this by the administration of digitalis or of mercurical diuretics were of no avail. The diversion of dietary sodium into the feces by means of ion exchange resins (carbo-resin, Lilly) often resulted in a dramatic drying out of the animal. The protocol in Figure 6 illustrates some of the things that happened in our experience.

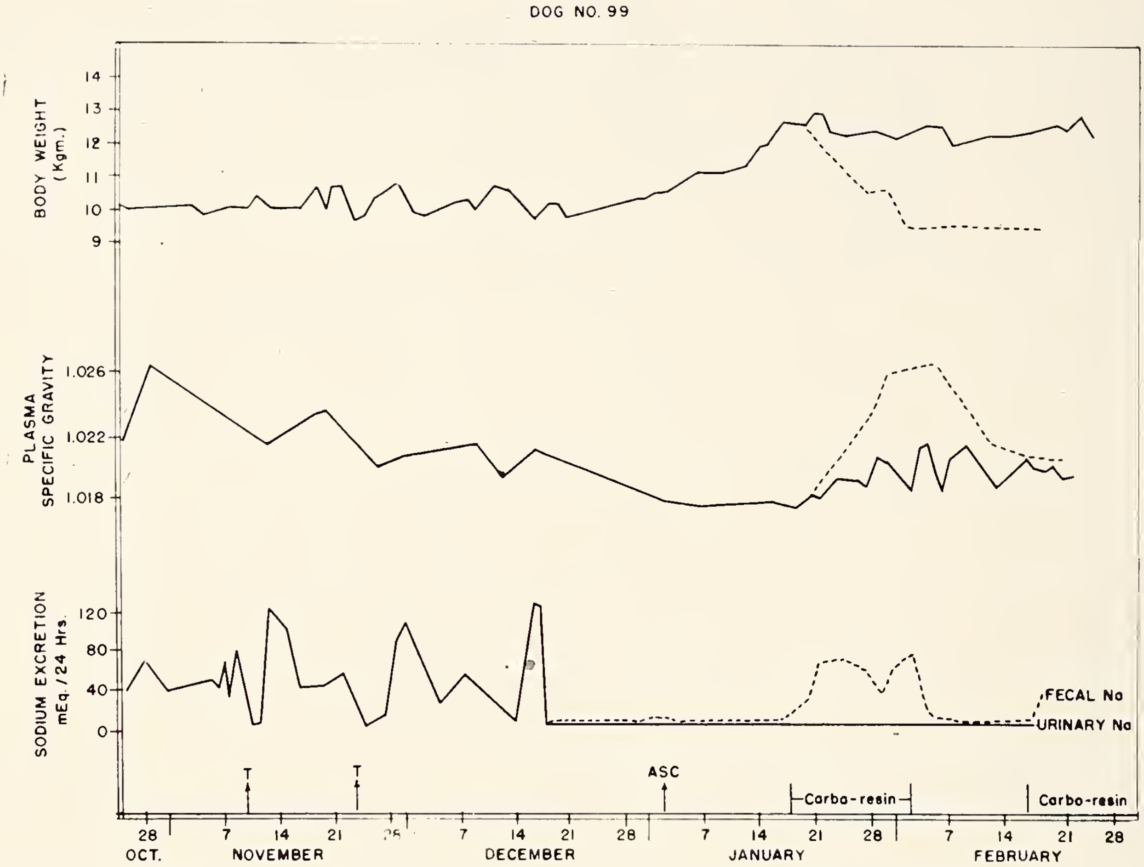

Fig. 6. The course of body weight, plasma specific gravity and sodium excretion in response to the administration of carbo-resin (Lilly). The dog indicated by the solid line was followed through the whole experiment. The dog indicated by the dotted line responded better to the resin (See text).

When the ligature was first tightened, the animal retained sodium for a day or so and then excreted it in excess. A second tightening resulted in the same response, followed by erratic urinary levels which finally eventuated in almost complete sodium retention and the formation of ascites. The body weight increased, the plasma protein level decreased, and the abdomen swelled conspicuously. As carbo-resin was administered, the sodium output (fecal) came into balance with the intake, the body weight leveled off, and the plasma protein returned to normal levels. Another dog whose response to carbo-resin was better (broken line), lost weight on the exhibition of the ion exchange resin, her ascitic pool was evacuated and her plasma proteins, after an overshoot, returned to normal values, which persisted as long as the resin was administered. Withdrawal of the resin caused this animal to become ascitic with low plasma protein levels. Animals can be induced to go through this cycle several times.

A similar cycle occurred when two dogs frayed their ligatures in two, releasing the mitral stricture. The sodium balance was restored by urinary excretion of sodium rather than fecal loss. The clinical picture was similar in that both animals lost their ascitic fluid and had an episode of concentrated plasma protein, which soon returned to normal. The animals became in all respects normal except for the hardware in their chests.

A very curious event occurred in the case of three dogs. In two animals, after two or three tightenings, no effect on the femoral pulse resulted from further tightening. On a later attempt the ligature came out in our hand. We were amazed to find it unbroken. It had evidently sawed its way through the auricle, much as a weighted string pulls through a piece of ice. The heart healed behind the ligature as it worked through the auricular walls and septum. The animal became quite normal and showed no signs of discomfort. In the case of the third animal the unbroken ligature was found free of the heart at autopsy.

The ascites began to form when plasma protein and venous pressure were within normal limits. Moreover, when carbo-resin was displayed, or when the ligature broke, the animals had an ascitic fluid whose protein concentration (normal AG ratio) was only 1 to 1.5 per cent lower than that in the plasma. The relation between hydrostatic and osmotic pressure was such as might be expected to lead to filtration. Nevertheless, the ascitic fluid was promptly evacuated as soon as the effective sodium intake was lowered or, in the broken string animals, as soon as the hemodynamic picture was made more nearly normal. This would seem to be brought about by a lessening of the stimulus (whatever it is) which causes the tubule to resorb water and sodium.

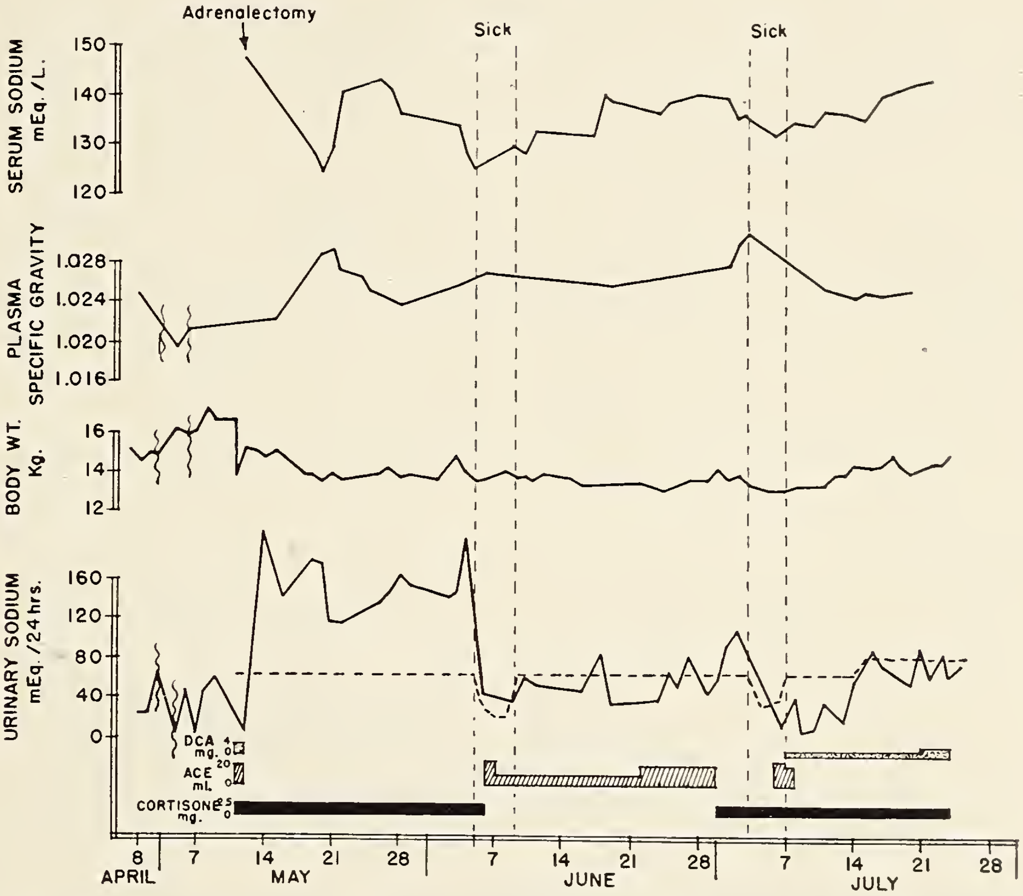

Fig. 7. Effect of adrenalectomy and substitution therapy in dogs with ascites from experimental mitral stenosis. (For details, see text.)

The immediate cause of the change in tubular resorption is probably hormonal. The amorphous part of the adrenal secretion is usually regarded as controlling the resorption of salt, and the posterior lobe hypothalamic mechanism probably controls the resorption of water. Neither salt nor water can be resorbed alone, but excess of adrenal hormone may cause the resorption of sodium followed by water, and excess of the pituitary hormone may cause resorption of water followed by salt. The pituitary mechanism has been implicated when plasma sodium levels, in congestive failure patients, are low and the plasma antidiuretic titer high.[19] The adrenal mechanism has been implicated by experiments that show that disoxycorticosterone acetate will produce edema, pulmonary congestion, and enlarged heart, making up the syndrome of congestive failure.[4]

Our dogs with mitral stenosis had normal sodium levels in their plasma. It was therefore decided to test both the pituitary mechanism and the adrenal mechanism.

After several attempts to get diabetes insipidus with hypothalamic injury and stalk section, by means of an operative approach with direct vision and by means of a stereotaxic instrument, two dogs, one of which died before the experiment was complete, were prepared with clinical evidence of the disease. On the surviving dog, the ligature, previously placed around the mitral valve, was tightened seven times at intervals of a week or more. She finally succumbed to pulmonary edema. At autopsy the mitral aperture was only 8 sq. mm. (19 mm^2/m^2), a size much smaller than those found in most of our other ascitic dogs. During nine weeks the tightening was repeated to produce sustained weakening of the femoral pulse, and no retention of sodium or formation of ascites occurred. Less than four such tightenings always produced ascites in our normal animals. We are left with the hypothesis that diabetes insipidus protects against sodium retention and ascites formation. During the last three days of the animal's life, urinary sodium was low but the belly was dry post mortem.

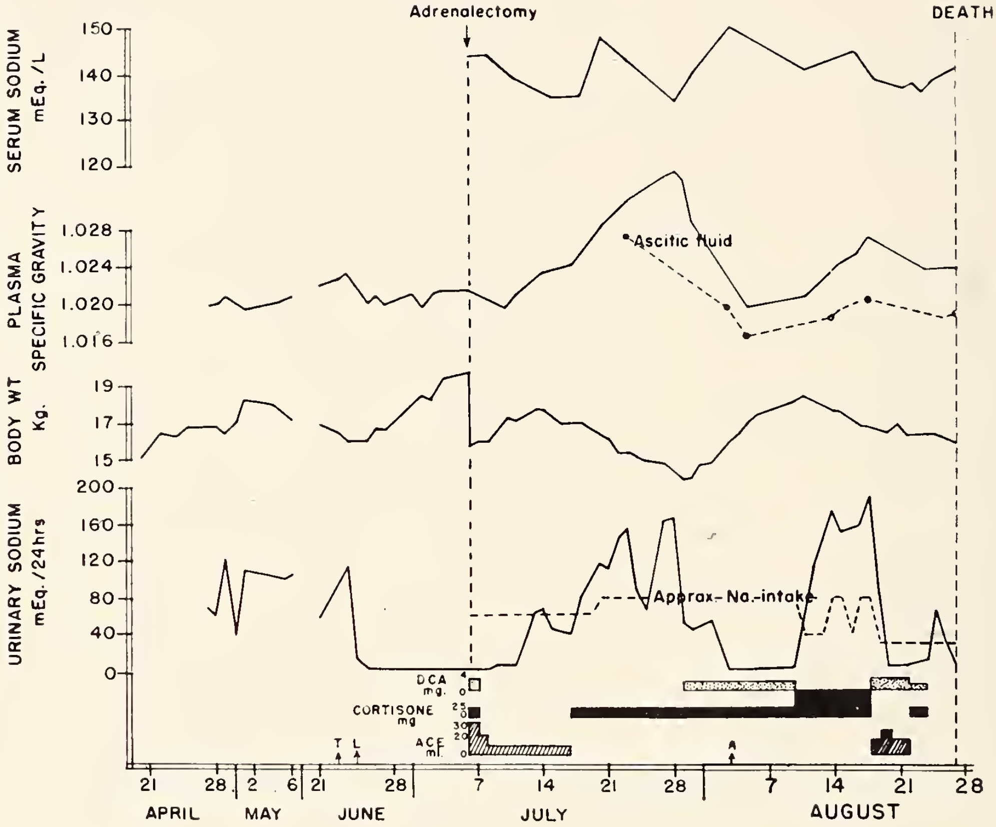

Fig. 8. Effect of adrenalectomy and substitution therapy in dogs with ascites from experimental mitral stenosis. (For details, see text.)

An attempt to produce diabetes insipidus in a dog already ascitic failed because of cellulitis and edema at the site of the operation.

To test the rôle of the adrenal gland, three animals were adrenalectomized on one side, the ligature placed and tightened until ascites appeared. One animal died. The other two became ascitic, and the second adrenal was removed. The first animal (Fig. 7) was treated for two postoperative days with whole adrenal extract, 25 cc. per day. When she was in good shape, extract was withdrawn and cortisone, 25 mg. per day was administered. What ascitic fluid had not been spilled during the operation was promptly evacuated and urinary sodium was markedly increased. The specific gravity of the plasma overshot, and then returned to normal. The animal was very vigorous. As soon as the ascitic fluid was gone, the plasma sodium decreased, the animal lost her appetite and showed other signs of adrenal deficiency. The cortisone was discontinued and she was given 10 cc. per day of whole adrenal extract (Upjohn). She came promptly into sodium balance and good health. Twenty cc. per day—four times the dose for normal adrenalectomized animals—did not cause any salt retention. Under the stimulus of mitral stenosis, the adrenal gland must have secreted an amount of the salt retaining hormone in excess of that contained in four maintenance doses of extract.

Cortisone treatment was then repeated. The animal again showed low plasma sodium levels and became ill. She was restored to vigor with a few doses of whole extract and maintained at that level with cortisone, 25 mg. per day and desoxycorticosterone acetate, 2 mg. per day. At autopsy the ligature was broken.

The second animal was adrenalectomized while in failure (Fig. 8). She was given 10 cc. per day of adrenal extract while she was still ascitic and came into approximate sodium balance without loss of ascites. Substitution of cortisone caused her to lose sodium and ascites in a profuse diuresis. The plasma sodium level remained normal and the animal vigorous as long as the store of ascitic fluid was available. When it was gone, the plasma sodium declined, and the animal became ill. Addition of desoxycorticosterone acetate, 2 mg. per day, put the plasma sodium back to normal and caused her to become vigorous again. The sodium excretion soon became minimal and the ascites returned. The desoxycorticosterone acetate was withheld, the cortisone dosage was increased to 75 mg. per day. The ascites was evacuated and the sodium excretion increased very much. The animal deteriorated at this point and died while we were attempting to improve the substitution therapy.

Conclusion

In concluding, it may be said that the mitral stenosis produced in these dogs resembles closely, in the size of the mitral orifice, and in the nature of the murmur, the condition seen in severe clinical cases. It leads to congestive failure (ascites) but this condition in the dog differs from congestive failure in man in that the fluid is collected in the abdomen rather than in the interstitial spaces. The effects of posture and gravity can be assigned a rôle in this difference.

The condition of our dogs lacks another of the usual signs of human congestive failure—*viz.* a high venous pressure. However, there are many clinical cases on record of cardiac edema with low venous pressure and many with high venous pressure and no edema.[1] Patients will gain or lose edema fluid regardless of whether their venous pressure is high or low if maneuvers are undertaken to correct renal errors in water balance.

The low venous pressure, and the absence of signs of passive congestion in the abdominal viscera, minimizes the rôle of possible forced filtration in these dogs. The amelioration of the ascites with sodium deviation by means of carbo-resin emphasizes the rôle of sodium in the disturbance. The fact that renal blood flow and glomerular filtration were not unduly restricted, indicates that the primary disturbance is a stimulation of the resorbtive function of the tubules.

The result of adrenalectomy implicates this gland and the fact that ascites would not increase under four times the maintenance dose of cortical extract implies either that the secretion of the gland was very excessive as a result of the stenosis, that the adrenal secretion was abnormal in character, having an undue amount of the salt retaining (amorphous) fraction or else that, in addition to the adrenal disturbances, there may be a disturbance in the function of the posterior pituitary.

Evidence that the pituitary is involved is very tenuous. It is only that the dog with experimental diabetes insipidus did not become ascitic under conditions that made other dogs ascitic. Further work is going on in an attempt to elucidate this problem.

It is interesting to speculate as to the rôle which this endocrine mechanism may play in health. Can it be decided whether we are working with a homeostatic mechanism that is being over stimulated, or an emergency response that is being continuously activated, either of which might lead to the vicious cycle seen here.

Gauer has presented evidence in this symposium that experimental procedures which tend to increase the effective local pressure of the blood within the great veins, right heart, and lungs, cause an increase in the output of salt and water. This is evidently a homeostatic mechanism. The stimulus, an increased distention of the low pressure blood reservoir, being the resultant, naturally, of an increase in the intravascular volume and the diuresis which eventuates, would decrease fluid and blood volume.

However, Gauer's mechanism is not the one that causes dogs with mitral stenosis to form ascites. Both the Gauer and the present procedure cause elevation of the effective pulmonary vascular, and right heart pressures. Gauer's dogs have diuresis; the mitral stenosis dogs have oliguria. It seems that we must think that the mitral stenosis dogs are making use of an emergency rather than a homeostatic mechanism. The oliguria of exercise,[30] which conserves salt and water, may be overstimulated and continuously activated until sodium practically disappears from the urine and ascites develops. The circulatory handicap may act like chronic exercise and set the mechanism off. The post pituitary is undoubtedly involved in the oliguria of exercise and may, perhaps, be over active here. An atypical stress reaction[24] may involve the adrenals. The picture is not clear and much further work should be done.

References

1. Altschule, M. D.: Physiology in Diseases of the Heart and Lungs. Cambridge: Harvard Univ. Press, 1949.
2. Bradley, S. E.; Mudge, G. H.; Blake, W. D., and Alphonse, P.: The effect of increased intra-abdominal pressure upon renal excretion of water, sodium, and potassium in normal human subjects and in patients with diabetes insipidus. J. Clin. Investigation, 28:772, 1949.
3. Briggs, A. P.; Fowell, D. M.; Hamilton, W. F.; Remington, J. W.; Wheeler, N. C., and Winslow, J. A.: Renal and circulatory factors in the edema

formation of congestive heart failure. J. Clin. Investigation, 27:810, 1948.

4. Currens, James H., and White, Paul D.: Congestive heart failure and electrocardiographic abnormalities resulting from excessive desoxycorticosterone acetate therapy in the treatment of Addison's disease. Am. Heart J., 28:611, 1944.
5. Davis, J. O., and Howell, D. S.: Mechanisms of fluid retention in experimental preparations in dogs. II. With inferior thoracic vena cava constriction. Circ. Research, 1:171, 1953.
6. Davis, J. O.; Howell, D. S., and Southworth, L. L.: Mechanisms of fluid and electrolyte retention in experimental preparations in dogs. III. Effect of adrenalectomy and subsequent desoxycorticosterone acetate administration on ascites formation. Circ. Research., 1:260, 1953.
7. Davis, James O., and Shock, Nathan W.: The effect of theophylline ethylene diamine on renal function in control subjects and in patients with congestive heart failure. J. Clin. Investigation, 28: 1459, 1949.
8. Dumarco, J.; Rimini, R., y Recarte, P.: La presion intraabdominal y la presion en la vena cava inferior. Revista argent. de Cardiol., 11:273, 1944.
9. Ellison, R. G.; Major, R. C.; Pickering, R. W., and Hamilton, W. F.: Technique of producing mitral stenosis of controlled degree. J. Thoracic Surg., 24: 154, 1952.
10. Ellison, R. G.; Major, R. C.; Pickering, R. W., and Hamilton, W. F.: Further experiences with a technique of producing mitral stenosis of controlled degree. p. 305. Surgical Forum, Philadelphia: Saunders, 1953.
11. Ellison, R. G.; Major, R. C.; Pickering, R. W., and Hamilton, W. F.: Surgical production of mitral stenosis of controlled degree. Am. J. Physiol., 167:781, 1951.
12. Farnsworth, E. B., and Krakusin, J. S.: Electrolyte partition in patients with edema of various origins. Qualitative and quantitative definition of cations and anions in cardiac decompensation. J. Lab. & Clin. Med., 33:1534, 1948.
13. Fishman, A. P.; Stamler, J.; Katz, L. N.; Miller, A. J.; Silber, E. N., and Rubenstein, L.: Mechanisms of edema formation in chronic experimental pericarditis with effusion. J. Clin. Investigation, 29: 521, 1950.
14. Haddy, F. J.; Ferrin, A. L.; Hannon, D. L.; Alden, F. F.; Adams, W. L., and Baronofsky, I. D.: Cardiac function in experimental mitral stenosis. Circ. Research, 1:219, 1953.
15. Hague, E. E.; Ellison, R. G.; Hamilton, W. F.; Pickering, R. W., and Rucker, J. T. Jr.: Renal and electrolyte changes in experimental mitral stenosis. Am. J. Physiol., 171:731, 1952.
16. Hamilton, W. F.; Ellison, R. G.; Pickering, R. W.; Hague, E. E., and Rucker, J. T. Jr.: Electrolyte hemodynamic and endocrine responses in experimental mitral stenosis. Am. J. Physiol. In press.
17. Holt, J. P.: The collapse factor in the measurement of venous pressure. Am. J. Physiol., 134:292, 1941.
18. Hwang, W.; Akman, L. C.; Miller, A. J.; Silber, E. M.; Stamler, J., and Katz, L. N.: Effects of sustained renal venous pressure on sodium excretion in unanesthetized dogs. Am. J. Physiol., 162:649, 1950.
19. Leaf, A., and Mamby, A.: An antidiuretic mechanism not regulated by extracellular tonicity. J. Clin. Investigation, 31:60, 1952.
20. Merrill, A. J.: Mechanisms in salt and water retention in heart failure. Am. J. Med., 6:357, 1949.
21. Pickering, R. W.; Hamilton, W. F., and Ellison, R. G.: Responses to mitral stenosis. Am. J. Physiol., 167:817, 1951.
22. Rucker, J. T., Jr.; Ellison, R. G.; Hague, E. E.; Hamilton, W. F., and Pickering, R. W.: Further observations on experimental mitral stenosis. Am. J. Physiol., 171:762, 1952.
23. Schroeder, H. A.: Studies in congestive failure. 1. The importance of restriction of salt as compared to water. Amer. Heart J., 22:141, 1941.
24. Selye, H.: Stress. Acta, Inc., Montreal.
25. Sinclair-Smith, B.; Kattus, A. A.; Genest, J., and Newman, E. V.: The renal mechanism of electrolyte excretion and the metabolic balances of electrolytes and nitrogen in congestive cardiac failure. The effects of exercise, rest, and aminophylline. Bull. Johns Hopkins Hosp., 84:369, 1949.
26. Smith, H. W.: The Kidney. New York: Oxford Press, 1951.
27. Stamler, J.; Goldberg, H.; Gordon, A.; Weinshel, M., and Katz, L. N.: Relationship of elevated renal venous pressure to sodium clearance and edema formation in unanesthetized dogs. Am. J. Physiol., 166:394, 1951.
28. Starling, E. H.: Physiological factors involved in the causation of dropsy. Lancet, 1:1405, 1896.
29. Wilkins, R. W.; Culbertson, J. W.; Burrows, B. A.; Tinsley, C. M.; Judson, W. E., and Burnett, M. C.: Antidiureses and renal vasconstriction following venous congestion of the limbs in normal and splachnicectomized subjects. J. Clin. Investigation, 28:819, 1949.
30. Verney, E. B.: Antidiuretic hormone and factors which determine its release. Proc. Roy. Soc., London, s. B., 135:25, 1947.
31. Zimmerman, H. M., and Hillsman, J. A.: Chronic passive congestion of the liver. An experimental study. Arch. Path., 9:1154, 1930.

COARCTATION OF THE AORTA

Coarctation of the aorta will vary from a partial narrowing of a considerable portion of the aortic arch to a severe localized constriction. Mild cases of coarctation will be asymptomatic and may be discovered only at necropsy. The chief clinical finding is *evidence of hypertension in the upper extremities* with decreased to absent pressures in the lower extremities, as manifested by faint or absent femoral pulsations. (Normally the pressure in the leg is greater than that in the arm.) Murmurs may or may not be present. The classic x-ray finding of notching of the ribs is rarely found before the age of fourteen years, as it takes time for the collateral circulation to cause pressure erosion of the ribs. Operation is indicated if the hypertension is severe or increasing.

The optimum time for surgery is under fourteen years of age because of the early appearance of arteriosclerotic plaques in the aorta, with consequent technical difficulties in suturing after excision of the narrowed portion.—From "Diagnosis of Congenital Cardiac Defects in General Practice," American Heart Association.

Laboratory Aids to Medical Practice

Sponsored by
The Minnesota Society of Clinical Pathologists

LABORATORY DIAGNOSIS OF HEMOLYTIC DISEASE OF THE NEWBORN

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THE laboratory diagnosis of hemolytic disease of the newborn is usually based on the demonstration of four general requirements: (1) Rh incompatibility between mother and father; (2) maternal Rh antibodies for erythrocytes of the father's Rh type; (3) presence of maternal antibodies on the infant's erythrocytes and (4) objective evidence of increased destruction of the infant's erthrocytes.

Demonstration of Rh Incompatibility

Proof of Rh incompatibility between parents depends on the reliability of tests to determine the presence or absence of Rh factor in erythrocytes. In general, such tests are reliable. However, shortcomings in routine Rh testing do exist. Occasional batches of testing serum with low potency are obtained from manufacturers. Increased demand for serum and decreased production have caused dangerous shortage of supplies at times.

Errors in Rh testing sometimes are made because tests are performed too soon after transfusion of blood of unknown Rh type. Errors occur because abnormalities in the patient's serum proteins may affect the results. Such errors are less common in obstetric patients than in patients who have chronic and complicated illnesses, such as lupus erythematosus, leukemia or malignant tumors. The only obstetric patients in whom this error might arise are those recently recovered from primary atypical pneumonia who have autoagglutinins that might give false positive results in an Rh negative person.

Demonstration of Maternal Antibodies

When the Rh factor was first discovered, antibodies could be found in only about half of the patients who had babies with hemolytic disease. Later, with the discovery that Rh antibodies differed from other antibodies, it became possible to increase the sensitivity of these tests so that antibodies could be found in 98 per cent of mothers who later had affected infants. At present, tests for antibodies have fallen into some disrepute because workers developed overly sensitive tests and reported antibodies in many patients who had normal babies. Also, faith was lost in the tests because it was found that the titer of antibody did not have the prognostic significance originally claimed for it. For example, increasing titers during gestation were not invariably associated with severely affected infants.

Methods of testing for antibodies at the Mayo Clinic have not been changed during the past six years; these methods have given satisfactory correlation between the presence or absence of antibodies and the occurrence of disease in the infant. The sole object of such tests should be the prediction of disease in the infant so that all necessary arrangements can be made for proper management at birth. The ideal time to do these tests is as late in gestation as possible and yet in time to make arrangements for handling the problem at birth.

Demonstration of Maternal Antibodies on the Infant's Erythrocytes

Maternal antibodies on the infant's erythrocytes usually are detected by the antiglobulin, or Coombs', test. Experience with this test indicates that when antibodies are found consistently in the mother results of the Coombs test at birth fre-

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The Mayo Foundation is a part of the Graduate School of the University of Minnesota.

This is the twelfth in a series of editorial reports sponsored by the Minnesota Society of Clinical Pathologists and designed to foster closer relationships between clinicians and pathologists.

quently are positive and other evidence of hemolytic disease is found. Negative results are obtained occasionally when other evidence of disease is present. Thus, positive results indicate trouble but negative results may be misleading. The Coombs' test should not be relied on as a sole means of diagnosis but should be performed along with other tests to meet the fourth laboratory requirement for diagnosis of hemolytic disease, namely evidence of destruction of the infant's erythrocytes at birth or within the first 24 hours of life.

Demonstration of Hemolysis

The best indicator of abnormal destruction of the infant's erythrocytes is an increase of indirect serum bilirubin to values of more than 4.5 mg. per 100 ml. of serum obtained from cord blood. The next best test is a decrease in erythrocytes to less than 3,500,000 per cubic millimeter of blood and next an increase in the incidence of reticulocytes to more than 10 per cent and of normoblasts to more than 15 per cent.

Therefore, the following laboratory tests are ordered and obtained immediately on babies born of Rh negative mothers who have Rh antibodies: serum bilirubin (cord blood), test for Rh factor, antiglobulin test and counts of erythrocytes, reticulocytes and normoblasts.

Comment

The diagnosis of hemolytic disease of the newborn is sometimes made although all four requirements are not met. The pathogenesis in these cases is not fully understood. Some investigators explain these cases on the basis of ABO rather than Rh incompatibility. Thus, one theory assumes that natural anti-B isohemagglutinins in the circulation of a group A mother may traverse the placenta and react with B substance on or in the infant's erythrocytes, leading to their abnormal destruction.

Death from hemolytic disease appears to be closely allied to toxic effects related to sudden breakdown of erythrocytes. Therefore, treatment now is being directed mainly at immediate replacement of the infant's blood with normal blood when any evidence of increased destruction is present. Thus, the presence of clinical jaundice within the first twenty-four hours, confirmed by values for indirect serum bilirubin of more than 4.5 mg., is a positive indication for replacement transfusion. Replacement transfusion using 500 ml. of blood usually causes about 50 per cent reduction in the value for serum bilirubin. It also eliminates from the infant's circulation any sensitized erythrocytes that have not yet been destroyed. If values for bilirubin continue to increase and go above 20 mg., further replacement transfusion is done.

Summary

The most important and vital need for successful management of hemolytic disease is the detection of jaundice during the first 24 hours of life. The explosive suddenness with which the hemolytic process sometimes develops without warning evidence emphasizes the fact that no substitute exists for almost constant observation of newborns for the appearance of jaundice, confirmed by measurement of serum bilirubin. The appearance of jaundice is an indication for immediate treatment.

LYON LABORATORIES DEDICATION

The new Elias P. Lyon Laboratories which include laboratories for cancer and heart research among others, will be dedicated February 11 in ceremonies to be conducted at the Coffman Memorial Union, University of Minnesota. Featured speakers at the dedication ceremonies will include President J. L. Morrill speaking for the University of Minnesota, representatives of the Public Health Service, and Dr. M. R. Runyon, executive vice president of the American Cancer Society.

The Minnesota Division, American Cancer Society, which supplied a major share of the funds for the construction of the new laboratory building, will conduct a workshop for state volunteer cancer workers in the Hotel Normandy, Minneapolis, February 10 and 11. Speakers who will help bring cancer information up to date for the field workers will include Dr. Bernard Zimmermann, cancer co-ordinator, University of Minnesota, and Dr. David Anderson, Austin, president of the Minnesota Division, American Cancer Society. Dr. John Bittner will conduct a tour of the University's cancer research facilities and Dr. Claude Hitchcock will be in charge of cancer exhibits in Coffman Union.

The Dean's Page

THE CANCER DETECTION CENTER

Intensive clinical and basic research during the past several decades has failed to provide a simple, direct, method of diagnosing cancer. At the present time many avenues of approach to this problem are being explored in various medical centers. While we wait and hope, it is imperative that we exhaust the possibilities of known methods for establishing a correct and early diagnosis of malignant disease.

It is becoming more apparent that earlier diagnosis and immediate treatment are the most significant procedures by which real improvement in long-term cancer patient survival will result. In order that this may come about for larger groups of people, increased emphasis must be directed to precancerous conditions and suspicious lesions. It is essential that all members of the medical profession join actively in this effort. In truth, "Every Doctor's Office Should Become a First Line of Defense as a Cancer Detection Center."

"Cancer detection" carries a unique significance as compared with "cancer diagnosis." Diagnosis implies the establishment of the cause of symptoms existing in a given patient. "Detection," on the other hand, indicates the finding of malignancies, pre-malignant conditions, or suspicious lesions or in symptom-free patients undergoing periodic check-ups.

The Cancer Detection Center at the University of Minnesota was established five years ago to test whether it would be feasible to examine large numbers of asymptomatic people for the earlier detection of malignancies in all organs of the body. Other Cancer Detection Centers have dealt primarily with organs readily accessible to the examining physician. This Center has placed emphasis on internal organs. It is now possible to review this five years' work.

During this five-year period, 5,352 patients were given a total of 11,343 examinations. A total of ninety cancers have been found. Precancerous lesions were present in 1,300 of the examinees.

In spite of our efforts to do so, it has been impossible to limit examinations at the Detection Center to individuals completely free of symptoms. We have divided, therefore, our cases of cancers into two groups: those with some symptoms referable to the organ or organ system in which cancer was found, and those who were without symptoms for the organ or organ system involved. On this basis, cancer has been diagnosed in forty-four patients with symptoms and in forty-six patients without symptoms.

An immediate point of interest was whether there has been a significant difference in the survival of patients who had lesions detected in the absence of symptoms as compared with those who had symptoms which, in all likelihood, would soon have taken them to their family physician. In the gastrointestinal tract, including the stomach, large bowel, and rectum, there were twenty-nine cancers diagnosed. Ten of the lesions were in persons with symptoms and nineteen were in persons free of symptoms of gastrointestinal disease. At the present time, there are only two survivors in the group with symptoms of gastrointestinal disease, while fifteen of the asymptomatic group of patients, are living and well. All of these patients received therapy through customary channels of medical practice throughout the state of Minnesota. It appears that the most significant factor in the difference of survival has been the early diagnosis of the lesions in the asymptomatic phase of the disease.

The Detection Center at the University Hospitals has used a routine, comprehensive type of complete examination composed of standard diagnostic practices. We have found achlorhydria or extreme hypochlorhydria to be an accurate aid in selecting patients who should have gastrointestinal x-ray examinations. The proctosigmoidoscopic examination of the rectum and sigmoid has been extremely

(Continued on Page 53)

Justus Ohage, M.D.
President, Minnesota State Medical Association

President's Letter

NEW YEAR, NEW CENTURY

It is with great pleasure that I begin the year as president of the Minnesota State Medical Association. I am honored to have been chosen to represent the medical profession of Minnesota in the highest position its organization can confer on a doctor.

Traditionally, the office of president of the state medical association has carried with it the esteem of medical men in the state, the inherent dignity of any such position of leadership, and the dedication and selflessness of the men who have held the position in the past.

The men who have preceded me as president have exuded those qualities in great measure; they have been exemplary figures representing the purposes of medicine; they have deserved the trust placed in them by their colleagues; and they have been able leaders, strong and forthright in their beliefs.

With such an inspiring tradition, I pledge myself to carry on their noble ideals, for I have a deep respect for their work and for the profession's central purpose—to promote the science and art of medicine; to elevate the standard of medical education; and to promote public health.

Furthermore, 1954 marks the beginning of a new era for the Minnesota State Medical Association. With this year we are inaugurating a new century of medical organization in the state—having celebrated the centennial milestone last year.

Thus, I feel doubly proud to head this fine organization as it embarks on its second century of vigorous existence. What this new century will bring, none of us can tell. But one thing is certain: the underlying strength of the profession will increase its stature, broaden its outlook, and spur it on to even greater accomplishments.

During the coming year, I intend to work with the profession in Minnesota to bring that ideal one step closer. Working together with Association committees, the Council, the county medical societies, the House of Delegates and individual members, we shall make continued forward progress as an organization.

Thus, it is an honor and privilege for me to serve the medical profession of the state as its president. In spite of probable setbacks and pitfalls, with such a fine organization as ours, one cannot help but see that improvements will be forthcoming, and achievements will be more numerous.

J. Ohage.

President, Minnesota State Medical Association

◆ Editorial ◆

Arthur H. Wells, M.D., *Editor;* Henry G. Moehring, M.D., and John F. Briggs, M.D.

BOARD OF EDITORS

THE COUNCIL created a Board of Editors of MINNESOTA MEDICINE at its September session, and appointed ten members to the Board at the December meeting. This group is entrusted with the solicitation, selection, editing and publishing of scientific papers and news items of our journal. The organization and its functions appear in certain respects to be unique. Each member has specific and indispensable editorial duties.

Acting on the theory that one of the principal functions of the State Medical Association journal is to help develop self-expression and professional authority within the state, Dr. Glenn J. Mouritsen of Fergus Falls will seek out desirable scientific presentations in the county and regional medical society meetings throughout the state. A second Scientific Assembly Editor, Dr. Tague C. Chisholm of Minneapolis will cover meetings of state-wide medical organizations and those in the Twin Cities. Dr. L. E. Steiner of Albert Lea will edit a Business Assembly Section of the journal. Drs. Henry G. Moehring of Duluth, and John F. Briggs of Saint Paul will be responsible for the Editorial Section. Miss Olive V. Seibert, the publishers' editor, will represent a fundamental link with the Bruce Publishing Company. Dr. Alex E. Brown with Dr. George Stilwell, of Rochester, and Dr. Robert B. Howard with Dr. Stuart W. Arhelger, of Minneapolis, will represent the two great scientific fountains of our state. Among other things, they hope to develop a section of the journal which will compare with the popular brief reviews of several commercial magazines. Their slogan is "MINNESOTA MEDICINE, the Bedtime Companion of Every Physician."

Any alterations in the editorial organization or the published journal are not to be considered as criticisms of the past, but are based on the theory that a trial of variations in all of man's activities is essential to progress. Please keep in mind that the standing of a state medical journal is principally limited by the professional attainments and support of the membership.

CARDIAC SYMPOSIUM

THE scientific section of MINNESOTA MEDICINE in the January and February issues will be devoted to the presentations of the Symposium on Recent Advances in Cardiovascular Physiology and Surgery, held at the University of Minnesota on September 15, 1953. This meeting of outstanding authorities from the scientific centers of the world was made possible through the unusual efforts of Dr. Grace M. Roth, Dr. Robert B. Howard, Dr. John F. Briggs and many other physicians of our state and by the financial support of the Minnesota Heart Association.

You can keep up-to-date by reading MINNESOTA MEDICINE.

FORECASTS

THE first month of a year is the time for giving that year a prognosis. Some people call this forecasting and make it a favorite sport during January. From our friends, we have gleaned some general rules about these forecasts; to wit, (1) If you want to be spectacular, go ahead and make definite predictions about our economic future, the outcome of the next elections, or whatever you prefer to forecast about. But don't forget that we herewith advise you to limit your forecast to several paragraphs which commit you to nothing—the type of verbose ambiguity which sounds good, but actually boils down to an incongruous jumble of irrelevant considerations. (2) On the other hand, when the accuracy of your comments cannot be questioned, be (and in this connection we use the word with fear and only after fasting) precise. A known statistic or one that can't be checked should be quoted to the last decimal place, not rounded off to an easily remembered number; for example, reports of 1880 showed 275,545 cows in the state of Minnesota.

Our Forecast.—Some experts are predicting that the outlook for 1954 is as glum as a bad mistake. We don't hold with that. Adjustments will occur, surely, but in proper perspective, a moving economy and society require such healthy changes. People will continue to try to get something for nothing, but will gradually become enlightened enough to settle for getting the highest quality something for the lowest price. And if physicians continue a patient and kindly program of enlightening the public about how to get the best medical care for the lowest cost to society, the year's end should show a real gain for the medical profession in the eyes of the public.

H.G.M.

MEDICAL CARE OF DEPENDENTS OF THE ARMED FORCES

When peace is gone and war is nigh,
God and the soldier is the cry.
When war is o'er and peace perfected,
God and the soldier are neglected.

THIS quatrain, so old it must be classed as "traditional" or "anonymous," touches on basic considerations in making up one's mind about medical care for dependents of those in the Armed Forces. It is recalled by the report from the Washington Office of the American Medical Association that a committee of admirals and generals recommend more medical care of dependents.

The report says a five-man committee believes that a serious deterioration of career service can be halted only by such things as higher military pay and more medical and dental care for dependents. Further, Assistant Secretary of Defense John Hannah warns that the services are losing career men in numbers "so great as to be disturbing." Secretary Hannah also cites low military pay and a "continual nibbling away at fringe benefits."

The immediate reaction may well be that military pay is already too high; as are taxes. What with this high military pay, why can't members of the Armed Forces pay for medical care like civilians? Well, we can only urge to look at the other side of the coin before you make any decisions:

If military pay is too high, why was, and is, everyone so eager to get out of the services into civilian life "where you can really make some money?" The fact remains that desirable members of the Armed Forces are leaving the services because pay is not high enough. Patriotism, loyalty to a cause, a high sense of duty—these are all well and good as statements of ideals, but day-in-and-day-out pay scale and benefits cut a more realistic and tangible figure to anyone objectively evaluating the pros and cons of making the Armed Forces his career.

The response that the "regulars" are no good anyway requires little rebuttal. One of the reasons that some career service men gave a poor impression during World War II was that the pay scale had been adequate for those men; their competence was a reflection of the pay scale rather than the pay scale's being a result of their ability.

You can't pick and train first rate chiefs of staff, surgeons general and top non-commissioned officers from a crowd of second rate men. You can't expect the crowd from which you intend to pick top-ranking career service men to be anything but overloaded with second raters if you don't pay them adequately.

It's the old story—you get what you pay for. If you want top quality men forming the "regular" framework of the Armed Forces, you must either pay for superior ability or put your hope and trust in finding a group of idealists who are above anything as mundane as pay and benefits.

H.G.M.

THE CANCER DETECTION CENTER

(Continued from Page 49)

valuable. Seventeen per cent of our examinees (all of whom are aged forty-five or over) have had adenomas or polyps of the rectum and lower sigmoid. Among patients who are asymptomatic the determination of occult blood in the stool has not been worth the laboratory expense and effort entailed. Careful visual examination of the vagina, and cervix, and bi-manual palpation of the pelvis have been rewarding in finding female pelvic malignancies. Five ovarian carcinomas have been detected and all were localized and confined to the ovary at the time of surgery. These women are all well at this time.

For the general public, the practicing physician is and always will be the key person in the diagnosis of cancer. A most important aspect of the Cancer Detection Center program is the evaluation and standardization of procedures which can be applied by physicians generally. We believe that physicians should continue to strive for wider application of cancer detection examination plans. Our Cancer Detection Center is eager to cooperate with them in so doing.

Harold S. Diehl
Dean, Medical School,
University of Minnesota

Medical Economics

Edited by the Committee on Medical Economics
of the
Minnesota State Medical Association
George Earl, M.D., Chairman

IMPORTANT ACTIONS TAKEN AT AMA SESSION

The House of Delegates of the American Medical Association, meeting at the Jefferson Hotel in St. Louis, during the Seventh Annual Clinical Session, took important policy actions on social security, voluntary health insurance, medical ethics and unethical practices, medical education, hospital accreditation, military affairs and a wide variety of subjects affecting both physicians and the public.

G. P. of the Year

Highlight of the opening session of the House on December 1 was the announcement that Dr. Joseph I. Greenwell of New Haven, Kentucky, had been selected as the 1953 "General Practitioner of the Year."

The first day of the meeting also featured addresses by several Association officers, and annual reports from the Board of Trustees, the Secretary and General Manager and by standing and special committees of the House.

Compulsory Coverage Hit

Approving a recommendation by its Reference Committee on Legislation and Public Relations, the House passed a resolution reaffirming its opposition to the compulsory coverage of physicians under the Old Age and Survivors Insurance provisions of the Social Security Act, and advocating passage of the Jenkins-Keogh bills now pending in Congress. These bills were described as providing for "the development of a voluntary pension program which is equitable, free from compulsion, and satisfies the retirement needs of physicians."

The reference committee report adopted by the House said:

> "The purpose of these bills is to eliminate the discrimination, and inequities which exist under present tax laws by extending the tax deferment privilege to the country's ten million self-employed and also to millions of employes who are not covered by pension plans. The purpose of the resolution is to reaffirm our support of the voluntary pension program provided in the Jenkins-Keogh bills and to reaffirm our strong opposition to the extension of compulsory coverage of physicians and other self-employed persons under Title II of the Social Security Act."

Bricker Amendment Supported

The same committee report urged continued action to obtain passage of the Bricker Amendment (S. J. Res. 1) and approved the principle of legislation which would reduce or remove the limitation on the deduction of medical and dental expenses for income tax purposes. The Bricker Amendment would outlaw any treaties which supersede the Constitution of the United States. The report also opposed any further extension of the "Doctor Draft" Law beyond the present expiration date of June 30, 1955.

On the "Doctor Draft" Law, the report stated that "your Committee feels strongly that there should be no further extension of the 'Doctor Draft' Law. We feel that the legislation is discriminatory and urge the Committee on Legislation and the Board of Trustees to actively oppose any further extension." AMA had recommended that the law be extended for only one year, when it expired on June 30, 1953.

Health Insurance Discussed

The House acted to accelerate the development of voluntary health insurance by passing a resolution requesting the Council on Medical Service to proceed immediately with a special study of the problems of catastrophic coverage and coverage for retired persons. The Council was asked to present its findings and recommendations to the House not later than the 1954 Clinical Meeting. The resolution pointed out:

> "There are two large groups of citizens for whom improved coverage could be offered under present pre-

paid medical care plans, namely: (a) those individuals who suffer catastrophic or long-continued and highly expensive illness and whose financial resources are not adequate to meet the cost thereof and (b) those citizens who have retired and are living on small incomes and who are not eligible under presently existing public or private plans."

The resolution emphasized the medical profession's "responsibility to make every effort to promote such prepaid medical coverage for all citizens whose circumstances make them eligible."

Another resolution on voluntary health insurance, adjudged to be emergency business by the Reference Committee on Insurance and Medical Service and then passed by the House, stated that "the American Medical Association condemns all insurance contracts which classify any medical service as a hospital service."

The resolution was the result of information received that Health Service, Inc., a capital stock casualty insurance company owned by the national Blue Cross Commission, had issued insurance contracts containing medical benefits as well as hospital benefits, to employes of the meat packing industry on a national basis. The resolution stated that this was in violation of principles set forth by the House which define pathology, radiology, anesthesiology and physiatry as medical services.

A further statement in the resolution said: "this continued disregard becomes even more disturbing in view of the fact that Medical Indemnity of America, Inc., an insurance company said to be owned and controlled by Blue Shield Medical Care Plans, Inc., and Health Service, Inc., are operated under an agreement whereby each insurer assumes half of the risk underwritten by the other."

Ethics' Problems Studied

A resolution introduced by the Iowa State Medical Society, calling for approval of a joint-billing procedure involving services rendered by two or more physicians, was referred to the Judicial Council, at the suggestion of the Reference Committee on Miscellaneous Business, with the recommendation "that the Judicial Council investigate the factors involved in the matter as presented and determine if there are new factors or new facets that would cause it to change the opinion" determined in 1952.

In an effort to solve the publicity problems resulting from unethical practices by a small minority of doctors, the House referred to the Board of Trustees a resolution calling for appointment of a special committee with broad professional representation to study all aspects of the problems. The Board was asked to study and implement the intent of the resolution and to report its findings to the House at the June, 1954, meeting in San Francisco.

Dues Picture Clarified

Because of confusion on AMA dues which has resulted since the House began the practice several years ago, the delegates passed the following resolution, which clarifies the situation considerably:

> "Resolved, that any active member of the American Medical Association who failed to pay dues for the year 1950, and who was suspended for such delinquency, may be reinstated during the first six months of 1954 by the payment of 1954 dues only.
>
> "Should such an individual fail to pay his 1954 dues by July 1, 1954, he shall continue to be considered delinquent."

This resolution was substituted for one on the same subject which was introduced by Minnesota's delegates in June, 1953.

To clarify misunderstandings among physicians regarding the rules and regulations of the Joint Commission on Accreditation of Hospitals, especially as they concern the role of the Department of General Practice in a hospital, the House adopted the following resolution:

> "That this House of Delegates of the American Medical Association request the Joint Commission on Accreditation of Hospitals to publish an article, or series of articles, in the *Journal of the American Medical Association* and other official publications circulating among the medical and hospital professions, to acquaint the medical-hospital profession with the regulations, by-laws and their interpretations, and
>
> "That the Commission clarify the methods by which an aggrieved hospital or its staff may appeal a decision with which they are not in agreement."

Education Fund Bolstered

The House was "pleased to note" that a fourth grant of $500,000 had been made by the American Medical Association to the American Medical Education Foundation for financial aid to the nation's medical schools. The Foundation reported that its 1953 income now totals $1,174,000 and that the number of contributors is now more than double the total in 1952.

Dr. Louis H. Bauer of New York, immediate past president of the AMA, was elected president of the Foundation just prior to the opening of the Clinical Session.

PR CONFERENCE DRAWS MANY

The Public Relations Conference, held the day before the Clinical Session of the AMA in St. Louis, was well attended by doctors interested in public relations and medical society personnel from all over the country. Those present heard many interesting and informative discussions on such topics as making a PR program work, what motivates the public's feelings toward medicine, selling the American economic system and how others do it, and mending PR fences.

One of the many talks presented that day was one by Mr. Roger Fleming, Secretary-Treasurer of the American Farm Bureau Federation. Mr. Fleming spoke on how the American Farm Bureau Federation informs the public about its program and its position on price controls.

He emphasized that simply because an organization happens to have taken a negative position on a matter, there is no reason to be ashamed and defensive about it. Illustrating, he stated that one of the most effective "programs" of all time has been the Ten Commandments, and that seven out of the ten begin with "thou shalt not."

Mr. Fleming also pointed out that Paul Revere and the rest of the early Americans were against something—they were against anything that was against freedom. "And that is the position of the Farm Bureau," he stated.

In conclusion Mr. Fleming sited the simile of the fruit growers in a certain state spraying their apples to prevent coddling moths from devouring them completely. It was not a matter of disliking moths so much, he stated, as it was a matter of liking apples so well.

FEDERAL SCHOLARSHIP LEGISLATION READY

The Defense Department's draft legislation for medical and other federal scholarships has received final checking over before presentation to the Budget Bureau for approval, according to a recent issue of the AMA Washington Letter. Budget Bureau approval is necessary if the plan is to be presented as an administration bill, but regardless of the bureau's action, the proposal could be offered by any member of Congress.

The plan provides:

1. Any medical, dental, nursing, or veterinary student accepting a scholarship would be obligated for one year of federal service for each scholarship year.

2. Payment would be made directly to the schools for tuition and other incidentals and to the student to cover living expenses during the school year.

3. Scholarships, limited to four years, would not be offered to pre-medical students or others preparing for professional courses.

4. Deans would make recommendations, but final selection would be by the Defense Department.

Department spokesmen explain that there are two objectives of the plan: first, to meet armed forces' needs after expiration of the doctor draft in 1955, if the regular draft obligation does not produce enough officers. Second, to interest enough young officers in regular military careers to maintain the regular corps at the necessary level. Currently regular medical officers make up only about 25 per cent of the medical corps total; it is hoped to reverse this ratio.

In regard to these federally subsidized scholarships, the AMA House of Delegates, at the Clinical Session in December, considered an emergency resolution which would have endorsed the principle of the scholarships. The resolution was discussed by a House reference committee, which recommended that the matter be referred to the Board of Trustees for study and action. The delegates approved this action unanimously.

HEPATITIS INCREASING

Infectious hepatitis (jaundice) is increasing in Minnesota as well as throughout the United States. To mid-December, 1953, 875 cases had been reported to the State Department of Health. Apparently no part of Minnesota is free from the disease, but it has been reported more frequently from some areas than from others. This difference may represent better reporting by physicians in the areas from which most cases are reported.

Early symptoms of infectious hepatitis include fever, nausea with or without vomiting, loss of appetite, fatigue, lassitude, headache, and abdominal discomfort.

Gamma globulin is reported to be effective in preventing infection in contacts where there is prolonged exposure. The Minnesota Department of Health supplies this material in limited quantities for family contacts of cases. A comparatively small dose is required for this protection—From *Minnesota Health*, January, 1954.

History of Medicine In Minnesota

MEDICAL HISTORY OF THE COUNTY OF YELLOW MEDICINE

MILDRED B. LEE
Granite Falls, Minnesota

(Continued from December, 1953, issue)

John Thomas Gill was born February 26, 1858, at Monroeville, Ohio, the son of Edward and Esther (Young) Gill. His father was a native of the Isle of Man, and had followed the trade of ship's carpenter. He had also helped in surveying the first railroad to be built on the Isthmus of Panama. Esther Gill was a native of Ohio.

John Thomas Gill was the fifth child in a family of nine children, and attended the public schools of Monroeville, Ohio. On completing his high school course he entered Western Reserve College at Cleveland, Ohio, then went to New York Medical College for his medical training, graduating in 1886. Two brothers, William and George, and a sister, Mary Gill, also chose medicine as a profession.

After receiving his degree in medicine Dr. Gill returned to Monroeville where he practiced medicine for four years. At the end of that time he went to Kansas City and practiced for a time in connection with the Cherry Street Hospital there. Following this experience he practiced in Chicago for two years. In 1895 he decided to look for a location in the west, and with Appleton, Minnesota, as his destination he chose a route through Yellow Medicine County over the Minnesota and St. Louis railroad. It was midwinter, and when the train reached Echo it was stalled by blizzard which tied up all rail traffic in the area. The passengers secured what accommodations were offered by the tiny settlement, Dr. Gill securing a room at the Nicollet House.

Word that a doctor was in town soon spread throughout the community, and before many hours had passed Dr. Gill was sought out by a man who was the father of two very sick children. At the farm home Dr. Gill found two little girls ill with diphtheria, one already beyond his help; from that time on Dr. Gill was in constant demand, and by the time the epidemic of diphtheria was past he had made up his mind to remain in Echo.

On October 9, 1896, Dr. Gill was united in marriage with Blanche McClure, who was a native of Seaforth, Ontario, Canada, but had been in Yellow Medicine County since 1894. After their marriage they resided in a small frame house in the business district of Echo and he had his office there for many years.

In appearance Dr. Gill was a slender, wiry, small-boned man of medium height with dark hair and dark eyes which were his most arresting feature. He was friendly, soft-spoken, but nevertheless a man of strong convictions; quick in his movements, decisive in action. His manner was unassuming, quiet, and kindly. He was everybody's friend, devoted to his patients, maintaining a high standard of medical practice and at the same time finding opportunities to take part in civic affairs for the betterment of the community.

In the course of his life in Echo he served as a member of the village council, and as a health officer, and as a member of the school board. He also became a member of the Masonic Order.

Dr. Gill never refused a call. In his first years in Echo he hired a livery rig to take him on his country calls. Later he had his own team and buggy or cutter, and when automobiles came into general use he became the owner of a small car. Thus equipped he would brave any kind of weather, at any cost to himself, and often his pay for this gruelling service would be in the form of a quarter of beef, a load of wood, a dressed goose, or just a word of thanks. Dr. Gill had great sympathy for the needy; he gave freely not only of his medical skill but of whatever he could of material help. Many times Mrs. Gill and her daughters made baby clothes and bought infant's shirts for some needy expectant mother. His daughter Dorothy says, "I can still see him going off with a bundle of baby clothes under one arm, and a toy for an older child in another. He taught his own children that to get the most out of life you had to share with your neighbor. He personally loved every baby he delivered, and there were hundreds of them."

Dr. Gill was, in his quiet way, intensely patriotic. At the beginning of World War I he took an active part in arranging patriotic meetings. His son, Edward, was one of those who were killed in action in that war, and brought home for burial in native soil.

As the years passed, nine children were born to Dr. and Mrs. Gill, grew up, and were educated in the local schools. As Dr. Gill grew older there was no slackening of his efforts to take care of the health problems of the community. During World War I he bought a house in Echo which he equipped as a sort of hospital, or nursing home, for his patients. In later years he took many of his patients to Redwood Falls Hospital. His daughter Esther became a nurse, the only one of his children showing his own inclination to service in the field of medicine.

In February, 1938, some of the people of Echo undertook to make a public show of appreciation for the doctor who had served them so tirelessly and devotedly for forty-three years. They planned a surprise party of grand proportions to be held at the auditorium on a Sunday afternoon, with a suitable gift to be presented to the guest of honor. But on February 21 Dr. Gill received a call from a country patient. Unable to locate his son Robert, who now drove for him, he decided he would go alone, as the Koecher home to which he was called was only two miles from Echo. He reached the home, attended to the needs of his patient, and started back again in his little car. The roads were partially blocked with snow, and Mr. Koecher therefore followed the doctor's car with team and wagon to see that he got safely through the bad places in the road; and when the car stalled he went to help the doctor put additional "snow links" on the wheels. Mr. Koecher had finished putting them on one side, then walked around the car to find the doctor lifeless in the snow.

It was found that death came as a result of a heart attack.

The doctor was survived by his wife and seven children. One child, a son, had died in infancy; Edward was killed in the first World War. The children surviving were James, Crystal (Mrs. P. E. Long), Margaret (Mrs. Fred Norton), Almona, Robert, Esther, Dorothy (Mrs. Carl Sorenson); also eight grandchildren.

The following obituary appeared in the February 25 issue of the *Echo Enterprise,* of Echo, Minnesota.

JOHN THOMAS GILL

His Death Came Suddenly, Without Warning,
Taking from Us Our Best Beloved Citizen.
The Entire Community Mourns.

Our village was stunned and shocked on Monday when word flashed around town that our best beloved citizen, Dr. John T. Gill, had suddenly passed away while making a call to the Fred Koecher home two miles southwest of Echo. The doctor was very busy that morning, and not being able to locate his son, Robert, when ready to start, decided to make the short drive alone. He had reached the Koecher home but before starting back they had placed two snow links upon each of the wheels. Mr. Koecher accompanied the doctor but just before reaching the highway 67 the car stalled. The two men got out to put an extra link upon the wheels. Mr. Koecher had got his link on and upon walking around to the other side of the car saw the doctor slumped over, dead.

Mr. Koecher who had followed the car with his team was obliged to drive back home to summon his son, Fred, Jr., who in turn contacted Alfred Bode and Robert Gill and drove to the scene of the trouble.

The hearse was then secured and the body brought to the mortuary rooms.

The doctor, as the most familiar figure in Echo over this wide span of years, will be sadly missed. Throughout his long residence here as a country doctor he had always stood ready, in all kinds of weather, to answer sick calls and administer to the need. He alone had visited more homes than any other resident, and was always on hand when needed. We join with citizens in general in paying just tribute to this worthy man and doctor. His will be a link in the lives of Echo citizens that will be hard to fill.

In the early days when this country was sparsely settled, and it was necessary to make long trips over the prairie, even in the winter time, with a cutter or bob sled, he never refused to respond to a call.

John T. Gill was born at Monroeville, Ohio, February 26, 1858, where he grew to manhood. After finishing the high school course in his home town, he attended the Western Reserve College at Cleveland and the New York Medical College at New York, graduating in 1886. He returned to his home town and practiced four years. He then went to Kansas City, Missouri, and practiced two years in the Cherry Street hospital. After two years in Chicago he came to Echo in 1895, where he has practiced his profession for the past forty-three years.

Dr. Gill was married October 9, 1895, to Miss Blanche McClure, the ceremony taking place at Redwood Falls. Mrs. Gill came to Echo in 1894 from Seaforth, Ontario, Canada. The widow and seven children survive, namely, James, Mrs. P. E. Long (Crystal) and Mrs. Fred Norton (Margaret) all of Minneapolis, Almona and Robert of Echo, Esther of Detroit, Mich., Mrs. Carl Sorenson (Dorothy) of Tyler. Edward Gill was a victim of the World War, dying in France with burial in Echo cemetery. One son died in infancy. Eight grandchildren also survive. Dr. Gill was a member of the Masonic order. He had served on the village council and school board and was health officer. The parents of Dr. Gill were Edward and Esther Young Gill. The father was born in the Isle of Man, and the mother in Ohio. They passed away in 1920 and in 1912. Mr. Gill Senior was a ship carpenter by trade, and helped survey the first railroad on the Isthmus of Panama. John T. Gill was the 5th child of a family of nine children. Surviving brothers and sisters were Dr. Mary Hunter and Miss Ruth Gill, of Grand Junction, Colo., Miss Bertha Gill, Ypsilanti, Mich., Dr. William Gill, Norwalk, Ohio, Dr. George Gill, Elyria, Ohio, Ernest Gill, Redondo Beach, California. Three sisters preceded him in death.

Dr. W. R. Alexander, pastor of the Presbyterian Church at Vesta, will officiate at the service at one-thirty at the auditorium.

Edward Henry Kerkhoff was born on May 13, 1872, on a farm in Spring Hill Township in Stearns County, Minnesota, about eight miles from Melrose. He was the son of Conrad Kerkhoff who was a native of Lippstadt, Westphalia, Germany, and Catherine Horner Kerkhoff, a native of Stuttgart in Wurttemberg, Germany. The parents had come to the United States and settled first in Wisconsin, then in Minnesota. In 1884 they settled on a farm near Melrose and Edward attended school there. After finishing his studies he took up telegraphy, but about 1894 he made up his mind to study medicine, enrolling in the old Hamline University Medical Department (Minneapolis College of Physicians and Surgeons). He completed his studies and graduated in 1899, but he had been licensed to practice medicine on June 16, 1898.

Early in June, 1898, Dr. Kerkhoff went to Clarkfield with the intention of locating there, as shown by the following announcement of the *Clarkfield Advocate,* on June 2, 1898:

> "Dr. E. H. Kerkhoff came up from Minneapolis last Friday and is now permanently located in Clarkfield. The doctor comes very well recommended, is a very pleasant gentleman to meet, and we predict success for him in our midst. He can be found at the drug store or Commercial House for the present. For the benefit of those who do not speak English we will state that the doctor can converse in both Skandinavian and German."

At the time of Dr. Kerkhoff's arrival in Clarkfield there were two other doctors in residence there; Dr. Albert R. Torgersen, a pioneer physician of the county, and Dr. J. Woodbury Donnell who was county coroner and also county physician. Dr. Donnell was a single man, often depressed and moody. He drank heavily at times, and his manner was not one to inspire confidence. Consequently he was not very successful in establishing a practice in Clarkfield, and spent most of his time helping out in the drug store.

On June 16, Dr. Kerkhoff was asked to make a professional call upon Dr. Donnell. On arriving at the doctor's rooms over the Clarkfield Drug Store Dr. Kerkhoff found Dr. Donnell lying in bed, bleeding profusely from a wound in the throat which he had inflicted upon himself with a pair of scissors. He was also in the throes of convulsions due to a dose of strychnine, also self-administered. In an attempt to save Dr. Donnell's life Dr. Kerkhoff quickly summoned help and endeavored to sew up the wound in the throat, but the patient died within a few hours.

In November Dr. Kerkhoff decided to leave Clarkfield. The *Clarkfield Advocate* of November 10, 1898, announced:

> "Dr. E. H. Kerkhoff left last Saturday for Minneapolis where he will enter the City Hospital for the purpose of improving himself in his chosen profession. During his stay in Clarkfield the doctor made many fast friends, all of whom will greatly miss him. It is hoped that he will again locate in this village when he has finished his hospital service."

After completing his internship at the Minneapolis City Hospital Dr. Kerkhoff was for a short time in Kasson, then in Plainview (Wabasha County). On December 19, 1899, he settled in Pierz, Minnesota, where he continued to practice until his death.

In 1904, Dr. Kerkhoff was married to Miss Rose Vorath, of Agram Township in Morrison County, near Pierz. There were two children born of this marriage.

In 1906 Dr. Kerkhoff practiced for a time in Richmond, Stearns County, but returned to Pierz within a few months. In addition to his medical practice he edited the *Pierz Journal* and was very much interested in farming.

Dr. Kerkhoff was known as a physician who was devoted to his patients and tireless in their service. He was exceptionally well read and interested in many fields outside the practice of medicine. His devotion to his patients caused him to continue caring for them during the pandemic of influenza which followed the first World War, although he himself had been ill for several weeks and was not well enough to resist the disease. He became ill with influenza, complicated by pneumonia, and he died in Pierz on November 15, 1918, at the age of forty-eight years.

John S. Kilbride, son of Michael and Mary (Quinn) Kilbride, was born in the village of Zwingle in Jackson County, Iowa, on December 23, 1865. His father and mother were born in County Roscommon, Ireland, where the Kilbride family had lived for many generations and worked in the trades of weaving and spinning in the Belfast district. Mary Quinn Kilbride was born in County Mayo, Ireland. Michael Kilbride and Mary Quinn came to the United States as young people, and met and were married at Chilicothe, Ohio. After their marriage they moved to eastern Iowa, and then to Havana, Illinois. They came to Jackson County, Iowa, about 1865. Three brothers of Mary Quinn had been killed in the Civil War. In Jackson county the Kilbrides prospered, and they acquired a farm of four hundred acres. Four children were born to them, the oldest of whom was John S. Kilbride. Michael Kilbride died at an early age, when the children were small. Mrs. Kilbride married again, and the stepfather was highly esteemed and respected by the children.

John S. Kilbride grew up in Jackson County, living on a farm and attending country schools. He also had a course at the State Academy at Iowa City, then went to Minneapolis where he attended Central High School, working for his board and other expenses. In the summer he did farm work, or any kind of work he could get.

For his medical training Dr. Kilbride entered the University of Illinois School of Medicine in Chicago, and graduated in 1893. He began his medical practice at Dawson, in Lac qui Parle County, Minnesota.

In the fall of 1894, a pretty young woman by the name of Jennie Spragg came from Milwaukee to teach school at Dawson, and shortly became ill with pneumonia. Dr. Kilbride was called to attend her, and it soon became apparent that she was a very sick girl. Dr. Kilbride was determined to save her; he was almost constantly at the house, neglecting all other calls to devote his efforts to her recovery. His efforts were rewarded; not only did she recover, but she became his wife, the mother of four children: Stephen, who died in infancy, Edward A., Linnae, and Zylpha.

In 1897 Dr. Kilbride took additional medical training, then practiced for a short time in Sleepy Eye. In 1900, he located in Canby.

From the first years of his practice Dr. Kilbride showed exceptional skill and aptitude for the practice of medicine. In his first years at Dawson he earned a reputation for quick and correct diagnosis and treatment. An instance is related of a young man suddenly stricken with appendicitis; Dr. Kilbride lost no time, but operated on the patient on a kitchen table, and removed an appendix already ruptured; the patient recovered. Again, cholera morbus which had claimed many small lives in Dawson, was promptly treated by Dr. Kilbride with unfailing success. His devotion to his patients in that community is still gratefully remembered.

A misfortune that befell the second son, Linnae, in Canby, weighed heavily on the doctor's mind for many years. The railroad had a turntable at Canby; in legal parlance, classed as an "attractive nuisance" in that children were so often drawn to them as a playground. Returning from a professional call one evening the doctor espied his two young sons playing around, and he took them home, and warned the maid and their mother about letting them play away from home. Then he left for his office. Within a short time the little boys were out of the house and back at play; this time they ventured on to the turn table, and Linnae's leg was so crushed that it became necessary to amputate it. Dr. Kilbride was a man of deep feeling, Irish temper and temperament, and he never got over feeling that the accident should have been prevented.

Mrs. Kilbride died August 16, 1908.

Dr. Kilbride was a general practitioner, but he also was a specialist in eye, ear, nose and throat surgery and skin diseases. He kept up with all developments in the field of medicine and attended clinics in medical centers both at home and abroad. In 1910 he went to Germany and studied for a year at the Zeugniss University, afterward touring Europe and visiting the birthplaces of his parents in Ireland. In 1911 he returned to Canby and sold his practice there to Dr. Field, then located in Watertown, South Dakota, where he practiced for four years.

During his residence at Watertown he married again, his second wife Marie being a former resident of Hastings, Minnesota. Of this marriage there was one child, a daughter, Kathleen. The second Mrs. Kilbride was a stately, handsome woman and a Catholic.

In 1915 Dr. Kilbride returned to Canby and formed a partnership with Dr. L. J. Holmberg. This association continued until 1936, when Dr. Kilbride sold his interest to Dr. Holmberg and went to Worthington to practice with his son, Dr. Edwin. In 1943 Dr. Kilbride returned to Canby and established an office in his home, and practiced until the end of World War II, when he returned to Worthington.

In December, 1949, he was severely injured in an automobile accident, and never recovered from it. As a result of his injuries he suffered kidney trouble with complications; loss of vision, and other difficulties. He was treated at Rochester, Minnesota, but rapidly failed and died May 26, 1950, at the Kilbride Hospital in Worthington.

Dr. Kilbride was survived by his four children, Dr. Edwin A. Kilbride of Worthington, Attorney Linnae of Hastings, Zylpha, wife of Stanford Clinton, an attorney, of Chicago, and Kathleen, wife of Nelton Knoop, of Des Plaines, Illinois.

Dr. Kilbride was a Democrat, a member of the Catholic church, and of the Holy Redeemers Council of the Knights of Columbus at Marshall, Minnesota. He was a member of the Minnesota State and American Medical Associations, and was at one time President of the Camp Release Medical Society. He was interested in agriculture, and owned several farms in Yellow Medicine County.

Dr. Kilbride's obituary appeared in the *Canby News*, June 2, 1950.

> "Dr. John S. Kilbride, 84, physician and surgeon in Canby for 32 years, died in the Kilbride Hospital at Worthington Monday night.
>
> Dr. Kilbride, who had been living with his son, Dr. Edwin Kilbride in Worthington since 1945, had been seriously injured in an automobile accident shortly before Christmas, 1949, sustaining a partial rupture of his stomach, and never fully recovered, and had failed rapidly recently. He had lost much of his vision before his death.
>
> Funeral services took place at a Worthington funeral home this morning (Thursday) and burial was made in the Worthington cemetery.
>
> Born in Iowa, December 23, 1864, he grew up in Dubuque and following his graduation from medical school began practicing in Dawson in 1898. After a year there he went to Sleepy Eye and practiced there for a year before coming to Canby in 1900.
>
> In 1910 he went to Germany and studied for a year, and on his return sold his practice here to Dr. Field, and established an office at Watertown, South Dakota, where he practiced for four years. In 1915 he and Dr. L. J. Holmberg formed a partnership here which continued until 1936, when Dr. Kilbride sold his interest to Dr. Holmberg and formed a partnership with his son in Worthington. In 1943 Dr. Kilbride returned to Canby and established an office in his home and practiced until the war ended, returning to Worthington in 1945.
>
> Surviving are four children; Dr. Edwin in Worthington, Attorney Linnae in Hastings, Zylpha in Evanston, Ill., and Kathleen (Mrs. Nelton Knoop) in Des Plaines, Ill."

Dr. Thomas McKey graduated from the Chicago Medical College in 1878. He then took a postgraduate course at the State University of Iowa City, Iowa, in surgery, under Professor W. F. Peck. He completed his course and received another diploma from that institution in March, 1882.

Dr. McKay's medical practice was begun in Merrill, Wisconsin. He was appointed surgeon of the Waupun Prison in 1883, and remained there until 1885 when he moved to Luverne, Minnesota. After a residence of six months in that place he removed to Clear Lake, South Dakota, and practiced there for ten years. In 1896 he located at Canby, Minnesota.

Dr. McKey was a man of average height and very corpulent, weighing in the neighborhood of 250 pounds. He was a pleasant man to meet, friendly and jovial, and made many friends. He was considered to be a very good doctor and he enjoyed a good practice while in Canby. Both the Doctor and Mrs. McKey were very well liked, and they had several children. So far as is known they had no church affiliations in Canby, but Dr. McKey was one of the first officers of Cassiopea Camp, Royal Neighbors, organized in Canby in 1899.

Dr. McKey remained in Canby until after 1900, and then went to Albert Lea, Minnesota.

Dr. M. G. McLean. Nothing is known about Dr. M. G. McLean aside from the fact that he was located in Canby for a few years prior to 1881, and served as coroner in 1878. His period of residence covered about the same period as that of Dr. Billington but there are few who remember anything about him, and as there is no record of his practice it is possible that he was not a graduate with a medical degree. He was, however, recognized as a physician by the county board, which at its meeting in March, 1877, allowed a bill of Dr. M: G. McLean for $2.00 for medical attention to the poor.

Dr. McLean took into parnership Dr. Herman E. Farnsworth in 1880, and the firm is mentioned in Rose's History of Yellow Medicine County in connection with the case of Michael J. Dowling, victim of the storm of October 15, 1880.

James McPeek was born in Indianapolis, Indiana, on March 5, 1852. He was the son of Thomas McPeek, a native of Indiana, and Sahara Barto McPeek who was born in Maine of French Huguenot stock. The family removed to Olmsted County, Minnesota, about 1855 and settled on a farm near Byron, where the boy grew up and attended the public schools of the county.

On December 24, 1875, James McPeek married Miss Ruth Ann Stevens. Some years later he decided to study medicine, and enrolled in the Keokuk College of Physicians and Surgeons in Iowa, graduating in 1889. Following this, and possibly for some time prior to his graduation, he practiced medicine in Gary, South Dakota.

When Dr. McPeek decided to locate in Canby, Yellow Medicine County, there were five McPeek children: Maude, George, Earl, DeEtte, and Sadie. In the spring of 1891 Dr. McPeek purchased, in partnership with O. M. Eichinger, the Adolph Gilberson drug store in Canby, and moved his family into the O. N. Lund house. At this time Mrs. McPeek was in poor health, and accompanied by daughter Maude she went to the home of her mother at Owatonna. In October it was found that her ailment was incurable, and she died in January, 1892, and was buried at Owatonna.

For a time the McPeek family was broken up, some of the younger children being placed with relatives. During this period Dr. McPeek was away on business for the drug store and personal affairs demanded much attention, so his brother Dr. E. A. McPeek "from the eastern part of the state" came to Canby with the intention of remaining there permanently; but he stayed only a few months.

In the latter part of 1892, the drug firm of McPeek and Eichinger bought out the stock of Dorr and Jenson and consolidated the two businesses. This store was one of those destroyed in the great fire in Canby on September 5, 1893; also it was one of the very few to be insured.

Dr. McPeek continued as a member of the Board of Pensions which met in Gary, South Dakota. He had been licensed to practice in that state on May 25, 1889, and made frequent trips there; the older children also kept their connections there. On September 16, 1893, Dr. McPeek was united in marriage with Mrs. Bessie Eakins, of Gary, a widow who had a son by her former marriage. This boy joined the family at Canby and attended school there, and in 1894, a son was born to Dr. and Mrs. McPeek.

Dr. McPeek was in early middle age when he came to Canby, and was of medium height, inclined to fat. He was genial and likeable, a very friendly man. The second Mrs. McPeek was a woman of striking good looks.

About 1895 Dr. McPeek left Canby to locate at St. Charles, Minnesota. Thereafter he moved to Kasson, about 1898, and in 1899 he moved to Sleepy Eye in Brown County. In 1903 he was located in International Falls, where he became health officer of the town. In 1906 he returned to Gary. In 1912 he was practicing at Breckenridge and was the local surgeon for a railway company there.

In his later years Dr. McPeek lived at Ranier, on Birch Point, Rainy Lake, Minnesota. He died of erysipelas of the face at the home of his daughter, Mrs. Stephen Trageser, of Minneapolis, on December 24, 1922, and was buried at Byron, Minnesota.

(To be continued in the Februrary issue)

RHEUMATIC FEVER AND THE SCHOOL CHILD

The school gives an unusual opportunity to attack rheumatic fever. Certain improvements in school health services are needed to make the best use of this opportunity. These recommendations of the Academy will not only be a better approach to rheumatic fever but to other health problems of children. The alertness of teachers and school nurses can bring to medical attention children with signs and symptoms suggestive of rheumatic fever whose condition might otherwise be overlooked. It is also an opportunity through periodic medical examinations to discover unrecognized damage to the heart and to keep under medical supervision known cases of the disease and to make family studies of rheumatic children. It is an opportunity to teach the principles of healthful living to children who have the disease or who are susceptible.

To aid school health authorities to develop a more rational approach in the control of disease, the Committees on School Health and Rheumatic Fever of the American Academy of Pediatrics recommend: (1) that the school medical examination be improved to aid in more accurate recognition and supervision of rheumatic children, (2) that more emphasis be placed on referral by teachers and nurses of pupils believed to be below-par for medical review, (3) that less emphasis be placed on restricting the physical activity of rheumatic children and more attention given to daily observation of pupils for signs or conditions suggestive of rheumatic disease, (4) that there be available, to school health services and the practitioners, diagnostic and consultation services to establish diagnosis, (5) that these services be developed in co-operation with, and by utilization of, existing medical and public health resources in the community. —From Report of the Committee on School Health and the Committee on Rheumatic Fever of the American Academy of Pediatrics, 1948.

THE MINNESOTA STATE BOARD OF MEDICAL EXAMINERS

230 Lowry Medical Arts Building
Saint Paul 2, Minnesota
E. M. JONES, M.D., *Secretary*

MINNEAPOLIS WOMAN MUST SERVE SIX MONTHS FOR ABORTION

Re. State of Minnesota vs. Annabelle Keith

On December 7, 1953, Mrs. Annabelle Keith, aged twenty-seven, 2805 St. Mary's Place, Minneapolis, was sentenced by the Hon. Theodore B. Knudson, Judge of the District Court of Hennepin County, to a term of not to exceed four years in the Women's Reformatory at Shakopee on an abortion charge, the sentence being stayed, however, and the defendant placed on probation on condition that she serve the first six months of her sentence in the Minneapolis Women's Detention Home. Judge Knudson further provided that Mrs. Keith should begin serving her term on December 29, 1953. The defendant had previously, on October 13, entered a plea of guilty before Judge Knudson to an information charging her with the crime of abortion.

Mrs. Keith was arrested by Minneapolis Police Officers on October 2, 1953, when it was discovered that she had aborted a twenty-four-old Saint Paul divorcee about three weeks previously. The defendant performed the abortion upon the patient who was three and a half months pregnant, by using a catheter and was paid the sum of $50.00 for her services. She is not licensed to practice any form of healing in this state and further admitted that she has had no medical training but claimed to have learned how to do abortions from a Milwaukee, Wisconsin, woman in 1942.

ONE MINNEAPOLIS MAN GIVEN PRISON TERM, ANOTHER PROBATION FOR ABORTION

Re State of Minnesota vs. Paul Schwede and Julius J. Capra

On October 1, 1953, Paul Schwede, forty-nine, 127 West 26th Street, Minneapolis, a meat market operator, was sentenced by the Hon. Theodore B. Knudson, Judge of the District Court of Hennepin County, to a term of not to exceed four years at hard labor in the State Prison at Stillwater for the crime of abortion. Because he was not placed on probation Schwede must serve his sentence

On November 9, 1953, Judge Knudson sentenced Julius J. Capra, thirty-five, a meter reader who resided at 239 Clifton Avenue, Minneapolis, to a three-year prison term on the same charge, the sentence being suspended, however, and the defendant placed on probation for the same period.

Both defendants were arrested in connection with the same case. An investigation disclosed that Capra had given the name and phone number of a twenty-eight-year-old Minneapolis divorcee who was seeking an abortionist, to the defendant Schwede. The abortion was later performed by Schwede, who holds no license to practice any form of healing in Minnesota. Of the $285 that was paid for the abortion Schwede received $200 and Capra was paid $85 for his services in the case. The investigation revealed that Capra had referred other women to Schwede for the same purpose in the past.

The defendant Schwede has a previous conviction for abortion, having been sentenced on June 20, 1950, by the Hon. Rolf Fosseen, Judge of the District Court of Hennepin County, to a term of not to exceed four years in the State Prison at Stillwater on that charge. However, the sentence was stayed for three years and Schwede was placed on probation after he had served three months in the Minneapolis Workhouse. The records of the United States District Court in Minneapolis disclose that on November 4, 1946, Schwede was fined $200 following a plea of nolo contendere to an information charging him with a conspiracy against the United States in connection with the illegal use of counterfeit sugar stamps.

MINNEAPOLIS WOMAN SENTENCED FOR ABORTION

Re State of Minnesota vs. Lois Townsend

On October 30, 1953, the above-named defendant was sentenced by the Hon. Theodore B. Knudson, Judge of the District Court of Hennepin County, to a term of not to exceed four years in the Women's Reformatory at Shakopee on an abortion charge, the sentence being stayed, however, and the defendant placed on probation for the same period of time. She had previously on September 16 entered a plea of guilty before Judge Knudson to an information charging her with that crime.

Miss Townsend, twenty-nine, was charged with having taken medicine, drugs or other substances, and used certain instruments with intent to produce her own miscarriage. She was brought by ambulance on August 21, 1953, from her apartment at 1325 Hawthorne Avenue, Minneapolis, to General Hospital because she was hemorrhaging as a result of an abortion, which medical opinion held to be an induced abortion.

When the police searched Miss Townsend's apartment, they found a bone knitting needle, which had the appearance of a catheter, some quinine, and also a wire such as those that are inserted inside of catheters. In addition they discovered a medical book that had a marker in it on a page which dealt with the subject of abortion. The ownership of the book was traced to a man who had been convicted of abortion in Hennepin County in 1941. When Miss Townsend was questioned by the authorities she denied that an abortion had been performed upon her by anyone, but when it was learned that she had previously stated that she had aborted herself the charge referred to above was placed against her.

◆ Reports and Announcements ◆

INTERNATIONAL ACADEMY OF PROCTOLOGY

The International Academy of Proctology announces its annual cash prize and certificate of Merit Award contest for 1953-1954. The best unpublished contribution on proctology or allied subjects will be awarded $100 and a Certificate of Merit. Certificates will be awarded also to physicians whose entries are deemed of unusual merit. This competition is open to all physicians in all countries, whether or not affiliated with the International Academy of Proctology. The winning contributions will be selected by a board of impartial judges, and all decisions are final.

The formal award of the first prize, and a presentation of other certificates, will be made at the annual convention dinner dance of the Academy in April, 1954. The Academy reserves the right to publish all contributions in its official publication, *The American Journal of Proctology*. All entries are limited to 5,000 words, must be typewritten in English, and submitted in five copies. All entries must be received no later than the first day of February, 1954. Entries should be addressed to the International Academy of Proctology, 43-55 Kissena Blvd., Flushing, New York.

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Physicians are cordially invited to attend the sixth annual convention of the International Academy of Proctology to be held at the Palmer House, Chicago, Illinois, April 8 to 11, 1954.

An extensive motion picture seminar of proctologic surgery (including office techniques) will be held on April 11. All scientific papers will present the latest developments in proctology and gastroenterology.

Because general practitioners as well as gastroenterologists and proctologists, face proctologic problems in their daily practice, much of the program has been planned to answer their questions.

The program will be available upon request to the Executive Office of the International Academy of Proctology, 43-55 Kissena Boulevard, Flushing, New York.

MISSISSIPPI VALLEY MEDICAL SOCIETY 1954 ESSAY CONTEST

In its thirteenth annual essay contest the Mississippi Valley Medical Society will offer a cash prize of $100, a gold medal, and a certificate of award for the best unpublished essay on any subject of general medical interest (including medical economics and education) and of practical value to the general practitioner of medicine. Certificate of merit may also be granted to the physicians whose essays are rated second or third best. Contestants must be members of the American Medical Association and residents and citizens of the United States. The winner will be invited to present his contribution before the nineteenth annual meeting of the Mississippi Valley Medical Society to be held at Chicago, September 23 and 24, 1954. The Society reserves the exclusive right to first publish the essay in its official publication—the *Mississippi Valley Medical Journal.* All contributions shall be typewritten in English in manuscript form, submitted in five copies, not to exceed 5,000 words, and must be received not later than May 1, 1954. The winning essays in the 1953 contest appear in the January, 1954, issue of the *Mississippi Valley Medical Journal.* Further details may be secured from: Harold Swanberg, M.D., secretary, Mississippi Valley Medical Society, 209-224 W. C. U. Building, Quincy, Illinois.

INTERNATIONAL GROUP OF DOCTORS IN ALCOHOLICS ANONYMOUS

The fifth annual International Group of Doctors in Alcoholics Anonymous will meet at the Mayflower Hotel, Akron, Ohio, May 14 to 16, 1954. All physicians interested in this meeting are cordially invited to attend. For information and reservations, address: Doctors, Mayflower Hotel, Akron, Ohio.

MINNESOTA OBSTETRICAL AND GYNECOLOGICAL SOCIETY

New officers of the Minnesota Obstetrical and Gynecological Society were named at a meeting held December 5, at the Hotel Lowry, Saint Paul. Dr. Arnold O. Swenson, Duluth, vice president, succeeds Dr. Arthur B. Hunt, Rochester, as president; Dr. Leonard Lang, Minneapolis, was elected vice president, and Dr. Rodney F. Sturley, Saint Paul, was re-elected secretary-treasurer. Scientific papers were presented during the day sessions, and Dr. LeRoy A. Calkins, professor of obstetrics and gynecology at the University of Kansas Medical Center, Kansas City, Kansas, addressed the group in the evening.

POSTGRADUATE COURSE IN DISEASES OF THE CHEST

A postgraduate course sponsored by the Council on Postgraduate Medical Education of the American College of Chest Physicians will be conducted February 15 to 19, 1954, at New Orleans, Louisiana. The course will be held in conjunction with the Tulane University of Louisiana and the Louisiana State University. Dr. J. Winthrop Peabody is chairman of the postgraduate course committee. For further information, address American College of Chest Physicians, 112 East Chestnut Street, Chicago 11, Illinois.

FAMILY DOCTORS' DAYS

University of Minnesota Hospitals

All physicians are cordially invited to attend the following Family Doctors' Days which will be presented under the direction of the department indicated.

February 17	—	Neurology
March 10	—	Dermatology
April 7	—	Pediatrics

Activities begin with luncheon at 12:00 noon with the staff of the department in the Hospital Dining Room and will continue throughout the afternoon.

For further information, write the head of the department concerned or the Director, Department of Continuation Medical Education, 3330 Powell Hall, University of Minnesota.

(The initial Family Doctors' Day of the year was presented under the direction of the Department of Radiology, January 6, 1954.)

CONTINUATION COURSES

A *Conference on Coroners' Problems* will be presented by the University of Minnesota from March 8 to 10, 1954, at the Center for Continuation Study on the University Campus. Intended primarily for coroners, the course will also be of interest to law enforcement officers and to pathologists. Procedures of help in the investigation of all types of accidental or suspicious deaths will be discussed. The faculty for the course will include Dr. Walter W. Jetter, professor of legal medicine, Boston University, and the program will be presented under the direction of Dr. James McCartney, professor, department of pathology, University of Minnesota Medical School.

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A continuation course in *Eye, Ear, Nose, and Throat for General Physicians* will be presented April 5 to 7 by the University of Minnesota at the Center for Continuation Study. The management of common problems involving the eyes, ears, and upper respiratory tract will be stressed. The course will be presented under the direction of Drs. L. R. Boies, Clinical Professor and Head, Department of Ophthalmology and Otolaryngology, and Erling W. Hansen, Clinical Professor and Director, Division of Ophthalmology. They will be joined by members of the clinical faculty of the University of Minnesota.

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The University of Minnesota will present a continuation course in *Urology for General Physicians,* April 8 to 10, 1954, at the Center for Continuation Study. Common urological problems encountered by the physician in general practice will be discussed in detail. The course will be presented under the direction of Dr. C. D. Creevy, Professor and Director, Division of Urology, who will be joined by members of the faculty of the University of Minnesota Medical School and the Mayo Foundation.

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Dr. Samuel A. Levine, Clinical Professor, Harvard University Medical School, Boston, will take part in a continuation course in *Cardiovascular Diseases for General Physicians* which will be presented at the Center for Continuation Study on the University of Minnesota Campus, March 22 to 24, 1954. The course, sponsored jointly by the University and the Minnesota Heart Association, will stress management of practical problems in the cardiovascular field. Dr. Levine will also present the annual George E. Fahr Lecture on March 23. The course will be presented under the direction of Dr. C. J. Watson, Professor and Head, Department of Medicine, University of Minnesota Medical School, and the remainder of the faculty will include clinical and full-time members of the faculty of the University of Minnesota Medical School and the Mayo Foundation.

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A continuation course in *Emergency Surgery for General Physicians* will be presented at the Center for Continuation Study, April 1 to 3, 1954. Intended primarily for the physician in general practice, the course will stress the emergency management of various types of trauma and of acute abdominal conditions. The faculty for the course will include Dr. J. Garrott Allen, Professor of Surgery, University of Chicago School of Medicine. He will also deliver the annual Clarence M. Jackson Lecture on the evening of April 1. The program will be presented under the direction of Dr. O. H. Wangensteen, Professor and Chairman, Department of Surgery, with clinical and full-time members of the staff of the University of Minnesota Medical School.

BLUE EARTH VALLEY MEDICAL SOCIETY

Doctors of Martin, Faribault and Watonwan Counties (Blue Earth Valley Medical Society) held their annual meeting at the Presbyterian Church at Blue Earth, November 19, with the following officers elected: Dr. E. J. Bratrude, St. James, president; Dr. R. S. Armstrong, Winnebago, vice president; Dr. H. Boysen, Madelia, secretary-treasurer; Dr. M. P. Virnig, Wells, delegate; Dr. Harold Coulter, Madelia, alternate delegate, and Dr. William J. Hruza, Madelia, member of the board of censors.

RICE COUNTY MEDICAL SOCIETY

Officers of the Rice County Medical Society for 1954 were elected October 20 at Faribault, as follows: Dr. Norman Lende, Faribault, president; Dr. Paul F. Meyer, Faribault, vice president; Dr. Burton A. Orr, Faribault, secretary-treasurer; Dr. James J. Kolars, Faribault, delegate; Dr. R. R. Moses, Kenyon, chairman of censors, with Dr. F. W. Stevenson, Faribault, and Dr. R. F. Mears, Northfield, as the other members of the board of censors.

Committee chairmen for 1954 are: Dr. Burton A. Orr, Faribault, Committee on Military Affairs and Civil Disaster Medical Services; Dr. Heinz H. Bruhl, Faribault, Committee on Education; Dr. Paul F. Meyer, Faribault, Chest Pilot Study and Follow-up; Dr. E. J. Engberg, Faribault, County Medical Advisory Committee on Medical Service; Dr. Paul H. Weaver, Faribault, Committee on Public Health; Dr. Carl A. Traeger, Faribault, Committee on newspaper publications; and Dr. Bernard Street, Northfield, County Medical Advisory Committee to the Welfare Department.

Society meetings will be held regularly on the third Tuesday of each month during 1954.

In Memoriam

LEE WILLIS BARRY

Dr. Lee Willis Barry, long a distinguished Minnesota obstetrician and gynecologist, died at the age of sixty-seven in Fort Lauderdale, Florida, Thursday, December 3, 1953.

Dr. Barry had practiced for nearly thirty-four years in Saint Paul, but had moved his offices to the Minneapolis Medical Arts Building shortly before he died.

As assistant clinical professor of obstetrics and gynecology at the University of Minnesota and as a practitioner, Dr. Barry was a leader for many years in his specialty. He was the first to receive a Ph.D. degree in obstetrics and gynecology at the University of Minnesota.

He was born at Elbridge, Michigan, and received his pre-medical training and medical education at the University of Michigan. After a year's general internship at the St. Francis Hospital in Pittsburgh, Pennsylvania, he spent two years in postgraduate study in pathology at the University of Indiana. After a two-year interval, three additional years of obstetrics and gynecology study were spent at the University of Minnesota, from which he received his advanced degree in 1919. He located in Saint Paul in 1920.

Besides his University affiliation, Dr. Barry was a member of the Ramsey County Medical Society, the Minnesota State Medical Association, the American Medical Association, the Minnesota Academy of Medicine, the Saint Paul Surgical Society and the Minnesota Obstetrical and Gynecological Society. He was a fellow of the American College of Surgeons and the International College of Surgeons, and a diplomate of the American Board of Obstetrics and Gynecology and the American Academy of Obstetrics and Gynecology. He held the post of chief of staff at St. Joseph's Hospital in Saint Paul and was a member of Saint Paul Rotary Club, Sigma Xi and Phi Rho Sigma.

He is survived by his wife, Nora W. Barry; two daughters, Mrs. Harry Skornia, Champaign, Illinois, and Miss Margaret Barry, Minneapolis; one son, Dr. Lee W. Barry, Jr., Saint Paul, a dentist; one sister, Mrs. Russell Thomas, Owasso, Michigan, and three grandchildren. Honorary pall bearers were Drs. Joseph F. Bicek, G. D. Brand, Ben A. Weis, Earl Lowe and C. N. Hensel, all of Saint Paul.

PHILIP FRANCIS DONOHUE

Dr. Philip Francis Donohue, prominent Saint Paul urologist and 1953 president of the Ramsey County Medical Society, died at Miller Hospital in Saint Paul, Thursday, December 17, 1953, at 9:30 p.m., following a heart attack suffered a short time earlier at the St. Paul Curling Club.

Dr. Donohue was fifty-nine years old and had been active in his practice and in his favorite sports up to the time of his death.

He was born in 1895 in Saint Paul, where he attended grade schools, the St. Thomas Academy and Mechanic Arts high school. He took his premedical training at St. Thomas College and the University of Minnesota and received his medical degree from the St. Louis School of Medicine in St. Louis, Missouri. He served his internship at St. John's Hospital in St. Louis and at Miller Hospital in Saint Paul and took his postgraduate training in urology in the Department of Urology at Ancker Hospital, Saint Paul. He practiced his specialty as surgeon and diagnostician for thirty years in Saint Paul, where he served as staff urologist at Ancker, Miller, St. John's, St. Luke's and St. Joseph's Hospitals. He also served as associate professor of urology at the University of Minnesota.

Dr. Donohue was a fellow of the American College of Surgeons and a diplomate of the American Board of Urology, on which he also served as member of the board. He was a member of the North Central Section of the American Urological Association and of the Saint Paul Surgical Society, the Saint Paul Clinical Club, the Saint Paul Medical Forum and the Twin City Urological Society. He was a member of the Ramsey County Medical Society which he served in many capacities besides president, the Minnesota State Medical Association, and the American Medical Association.

Dr. Donohue occupied the old family home at 1481 Summit Avenue. He was an enthusiastic golfer and a member of Saint Paul Rotary Club, and of the Town and Country Club.

He is survived by one brother, John H. Donohue, of Saint Paul, and three sisters, Mrs. Grace Lavery, Evanston, Illinois; Mrs. Edith Dolan, Saint Paul, and Miss Florence Donohue, Pittsburgh, Pennsylvania.

ROBERT CYRUS FARRISH

Dr. Robert Cyrus Farrish died Friday, December 11, 1953, at his home in Sherburn after more than forty years of general practice in the area. He was eighty-six years old and still served occasionally as a consultant at the time of his death.

A community celebration marked his fortieth year of practice in Sherburn in 1950, and hundreds of residents from the entire district, most of whom he had delivered as infants, attended. It was the occasion for many recollections about him as the beloved and trusted country doctor. He was said to have cared for 1,600 cases during the influenza epidemic of 1917 and 1918 during which period he wore out four sets of tires, lost 44 pounds in weight, and drove to exhaustion no less than seven drivers for his car.

Dr. Farrish was born in Rockwood, Ontario, where his father, William Farrish, a Dumfries Scot, had located in the early 1850's. A friend of Empire-builder James

J. Hill, the elder Farrish moved to Saint Paul soon after Dr. Farrish was born in 1869. Dr. Farrish attended Rockwood Academy and then went to work for Hill in Saint Paul to help finance his medical education. He enrolled in the medical school of the University of Minnesota in 1897 and received his degree in 1901. He served his internship at St. Mary's Hospital in Rochester and became surgical assistant there to Dr. Charles H. Mayo. At the time of his death, he was the oldest living intern from St. Mary's.

He was a charter member of the Blue Earth County Medical Society, a member of the Minnesota State Medical Association, of which he became a life member in 1947, and of the American Medical Association. He served as vice president of the Minnesota Association in 1930.

Dr. Farrish went to Sherburn to practice with his brother, Dr. Malcolm Farrish, also a physician, in 1910, and together they maintained offices in Sherburn and Ceylon. After the death of his brother in 1915, Dr. Farrish practiced alone and also operated the Farrish Hospital in Sherburn for many years. In 1920, he married Mrs. Laura Bettenhausen Farrish, his brother's widow, and adopted her three children, the eldest of whom, Mrs. William Luwe, is now a practicing attorney in Mankato. A second daughter married Dr. L. O. Johnson, a Sherburn dentist. The third child is Dr. Robert Malcolm Farrish, a dentist practicing in Litchfield.

Dr. Farrish was a regional medical examiner for the 4-H clubs, and a 32nd degree Mason and member of the Shrine.

He is survived by his wife, three children, a sister, Mrs. L. G. Foley of Saint Paul, and two grandchildren.

HILLARD HERMAN HOLM

Dr. Hillard Herman Holm, for many years a leading surgeon of Glencoe, Minnesota, died following surgery for a stomach ailment at Asbury Hospital in Minneapolis, Thursday, November 19, 1953. He was sixty years old.

Dr. Holm had been active in his practice at Glencoe until a month before he died. He moved there on Valentine's Day of 1920, the day after his marriage to Blanche Laura Rucker of Howard Lake, and remained there continuously for thirty-three years.

He was born at Carver, April 11, 1893, and went to school there. He attended Macalester College where he received his Bachelor of Arts degree in 1915. He received his medical education at the University of Minnesota medical school and served a two-year internship in surgery at Minneapolis General Hospital.

During the earlier years of his practice, Dr. Holm operated his own hospital in Glencoe with the assistance of Mrs. Holm, a registered nurse. He was health officer of Glencoe and McLeod County for a number of years and served as coroner for several terms. He was a leader in civic as well as medical affairs in the community and was a member for many years of the First Congregational Church of Glencoe, the American Legion, the Izaak Walton League and the University of Minnesota Alumni Association.

Among physicians, he was recognized as a skilled surgeon, and he is remembered especially for a successful operation separating Siamese twins which was performed in January, 1927.

Dr. Holm was a fellow of the American College of Surgeons, a member of the Minnesota State Medical Association, the American Medical Association, the McLeod County Medical Society, the American Association of Railway Surgeons and the Milwaukee Road Company Surgeons.

He is survived by his wife; a daughter, Mrs. R. H. Rantala, Fort Worth, Texas; a brother, Harold C. Holm, St. Paul; and two grandchildren, Neil and Erick Rantala of Fort Worth.

Eleven fellow physicians from Glencoe and surrounding communities acted as honorary pallbearers. They were Drs. J. D. Selmo, Norwood; J. J. Smyth, Lester Prairie; A. M. Jensen and G. L. Griebie, Brownton; W. W. Klima, Stewart; Edward G. Olsen, Minneapolis; Charles G. Sheppard, Donald Brink, Walter G. Sahr and E. W. Lippman, Hutchinson; and C. W. Truesdale, Glencoe.

HENRY J. SHELVER

Dr. Henry J. Shelver, who practiced medicine first at Appleton and then in Ortonville for nearly thirty years, died Saturday, January 2, 1954, in the United States Veterans Administration Hospital in Fargo, North Dakota, where he had been a patient for the last three years. Dr. Shelver was sixty-nine years old.

He was educated at Concordia College in Moorhead and at Hamline and Northwestern Universities. He was graduated from the latter in 1910 and served his internship at Swedish Hospital in Minneapolis. After a further period of postgraduate work in Chicago, he opened his practice in Appleton where he remained for eight years before moving to Ortonville.

Until his retirement from active practice, Dr. Shelver was a member of the West Central Medical Society, the Minnesota State Medical Association and the American Medical Association.

Survivors are Mrs. Shelver and a daughter, Mrs. Glenn Crane of Morristown, New Jersey.

◆ Of General Interest ◆

Willow River, without a resident doctor since the death of Dr. W. C. Ehmke in 1948, has secured the services of **Dr. Martin S. Munson,** Barnum, for Tuesdays and Thursdays of each week. Willow River Community Club and Willow River Commercial Club have spent time and effort working out the details of the plan and securing office space for Dr. Munson.

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Dr. Fred Soper, New York City, director for the World Health Organization for the Western Hemisphere, in speaking at the University of Minnesota recently, cited the tremendous strides in helping backward countries help themselves to better health. Most significant has been the development of teams consisting of doctors, engineers, nurses, educators, sanitary inspectors and others who have worked together to fight perennial diseases such as malaria and yellow fever, and solve problems of mother and child health.

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The quarterly new Fellows dinner, November 19, 1953, was held at Balfour Hall of Mayo Foundation House, with more than one hundred physicians in attendance. The dinner was given for new Fellows, and physicians recently returned from military service to complete their fellowships. Speakers were: **Dr. Fredrick A. Willius,** senior consultant in cardiology at the Clinic, who spoke of the heritage and traditions of the Mayo Clinic, and described experiences he had had with the late Dr. Henry S. Plummer; **Dr. Jesse Edwards,** consultant in pathological anatomy at the Clinic, who spoke of the opportunities for Fellows at the Mayo Foundation with regard to research activities, meetings and seminars, and the opportunities of obtaining postgraduate degrees.

Fellows were greeted by **Dr. Clayton H. Klakeg,** vice president of the Fellows Association and master of ceremonies for the program. The following officers were introduced: **Dr. E. V. Johnston,** president; **Dr. J. O. Godden,** secretary, and **Dr. E. B. Waldmann,** treasurer. **Dr. William Manger,** past president of the association, greeted the group also.

A standing ovation was given by **Dr. Donald C. Balfour, Sr.,** emeritus director of the Mayo Foundation, as he expressed his greetings.

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Dr. Paul V. Cumiskey of Mankato, is temporarily working at the Oliver Clinic until a new clinic with which he will be associated in Minneapolis, is completed. Dr. Cumiskey is a graduate of the University of Minnesota, serving his internship at the Fresno County General Hospital.

Dr. M. H. Bauer, Winthrop, was injured by flying glass particles when a stray bullet hit the windshield of the car in which he and his wife were driving through Chaska, November 23. The shatterproof windshield protected him, probably saving his life. Mrs. Bauer was driving at the time of the accident.

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The regents of the University of Minnesota authorized thirty-five new staff appointments, fifty terminations of staff personnel, eighteen promotions and transfers, twenty salary adjustments, twenty-eight leaves of absences, and accepted $28,744 in gifts. Two medical research scientists headed the list of appointments: **Dr. Francisco Grande,** citizen of Spain, will become an associated professor in the laboratory of physiological hygiene at the University, and **Dr. Richard W. Anderson,** psychiatrist from Saint Paul, will become an associated professor in the department of psychiatry and neurology.

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The merits of the Blue Cross and Blue Shield plans were discussed at the Stearns-Benton Medical Society meeting held at St. Cloud, November 19, 1953. **Dr. O. L. M. Nelson,** Minneapolis, a specialist in internal medicine, described cases of psychosomatic factors in treatment of diseases. Attendance prizes were won by **Dr. R. P. Koenig,** Paynesville, **Dr. M. McMahon,** St. Cloud, and **Dr. E. Sherwood,** Kimball.

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The new Seifert-Dupont-Dornbach medical clinic, Excelsior, opened the first of November. The clinic contains some twelve consultation rooms and laboratories, and offices for the three physicians: **Dr. M. H. Seifert, Dr. J. A. Dupont,** and **Dr. Robert Dornbach.**

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Dr. Walter A. Reiling, chief of staff of the Good Samaritan Hospital, Dayton, Ohio, spoke at a general meeting of all Optimist clubs in Minneapolis, December 2. Dr. Reiling is president of Optimist International.

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Dr. John O'Hanlon has opened offices in Edina with **Dr. Harry C. Jensen.** Dr. O'Hanlon is a graduate of Marquette University. He has been in practice in rural areas as well as Minneapolis and Duluth.

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Dr. James Reinhardt, of Detroit Lakes, has been promoted to the rank of captain in the army. Dr. Reinhardt, stationed at James Connally Air Force Base, was surgeon and physician at Alexandria before going into the army.

Dr. Louis Katz, director of cardiovascular research at Michael Reese Hospital, Chicago, and professional lecturer in physiology at the University of Chicago, spoke on the brighter outlook for the victim of blood pressure at Saint Paul Society for Internal Medicine held at the University Club, December 2. **Dr. D. R. Gillespie,** of Saint Paul, is president of the group.

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Dr. Wallace Ritchie, Saint Paul, presented a discussion on head injuries with particular reference to neuro-surgical treatment which the general practitioner can render, at a dinner meeting of the Southwestern area of Minnesota Academy of General Practice. The meeting was held in the Municipal Lounge at Willmar, November 30.

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Dr. M. H. Larson, Nicollet, spoke at a Cancer Workshop held in St. Genevieve's Church, Lake Benton, November 18. Dr. Larson stressed that the doctor's office is the first line of defense against cancer.

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The fiftieth anniversary of the Hennepin County Tuberculosis Association which was organized December 2, 1903, as the Anti-Tuberculosis Committee of the Minneapolis Associated Charities, was celebrated at the Citizens Aid building, Minneapolis, December 2, 1953. **Dr. S. Marx White,** member of the association's executive committee, presided. He also gave a pictorial record of early tuberculosis work in Minneapolis and Hennepin County. "Tuberculosis led all other diseases as a cause of death. One child in every nine died before its first birthday," said Dr. White in commenting on the early work.

Dr. Stewart C. Thomson, professor and assistant director of the University of Minnesota School of Public Health, reviewed the work of a half century in fighting tuberculosis in a talk entitled, "The Building of a Wall."

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Dr. Martin S. Munson, Barnum, has "adopted" Kim Kyong Ja, an eleven-year-old Korean girl, under Foster Parents' Plan for War Children, Inc. The "adoption" is not legal, of course, only financial. The foster parent promises to contribute $15 monthly toward the child's support for one year, receiving in turn a brief history of the child and a photograph as well as correspondence, if possible, to help the child feel that he belongs to someone. "Home" to Kim Kyong Ja is a crowded institution in Kunsan.

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Dr. Herman E. Hilleboe, son of P. S. Hilleboe, Mankato, has been named president-elect of the American Public Health Association. Dr. Hilleboe is state commissioner of health in New York. He will take office as president at the association's annual meeting to be held in Buffalo, New York, October, 1954.

Dr. Frederick W. Wittich, Minneapolis allergy specialist, has been named trustee and member of the executive committee of the newly-formed American Foundation for Allergic Diseases, Inc. This is a nonprofit organization to help promote understanding of allergic diseases, of which, studies have shown, there are 3,900,000 Americans suffering. Dr. Wittich is managing editor of *Annals of Allergy* and editor of the *Quarterly Review of Allergy and Applied Immunology.*

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Dr. and Mrs. Arthur B. Williams, Saint Paul, celebrated their golden wedding anniversary in October. They were married in Rock Rapids, Iowa, October 14, 1903, and lived in Wilmont, Minnesota, where Dr. Williams practiced medicine until he was called into service in World War I. Following the war, the family moved to Worthington, where Dr. Williams practiced for six years, before moving to Saint Paul in 1926.

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Dr. N. R. Kippen has become associated with the Breckenridge Clinic, Breckenridge. Dr. Kippen was graduated from the University of Manitoba in 1948. During the last five years he had further studies in surgery, orthopedic surgery and pathology in Canada.

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Dr. Clinton C. Berg, Dr. Melvin G. Oppen and **Dr. Roger S. Johnson** are engaged in group practice with offices in both Excelsior and Deephaven. The association was formed December 1. Dr. Berg was previously associated with Dr. Lewis Reid of Excelsior. All three doctors are graduates of the School of Medicine, University of Minnesota.

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Dr. Irvine McQuarrie, president of the State Heart Association, and **Dr. Harold S. Diehl,** dean of the University of Minnesota Medical School, were speakers at a luncheon in Coffman Memorial Union, December 5, for volunteer workers in the 1954 Heart fund. The annual fund campaign, to be held in February, with a quota of $210,000, is sponsored by members of the Minnesota State Association of Life Underwriters. A check of $30,000 was presented by the Minnesota Heart Association to Dr. Diehl at the luncheon. This is the second allocation for heart research at the university for 1953.

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Dr. Joseph Brennan, Duluth, spoke at the Park Point Community Club, of Duluth, November 27. Dr. Brennan, born on the island of Malta, was studying medicine in Rome when he was imprisoned by the Italians during World War II. After escaping, he worked with underground groups. He completed his education in Europe and America.

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The new Chisholm Memorial Hospital opened November 2, 1953, with **Dr. P. H. Macfarlane** as head of the staff; **Dr. Clarence Jacobson,** vice president, and **Dr. A. D. Klein,** secretary. Organized on an "open staff" basis, the following doctors are registered with the hospital: **Dr. Joseph L. Arko, Dr. George Doxsee, Dr. George Erickson, Dr. Frank Farley, Dr. Bernard Flynn, Dr. Bayard French, Dr. Bernard Hapler, Dr. Robert Hansen, Dr. H. R. Irwin, Dr. Luverne Johnsrud, Dr. Stanley Lipinski, Dr. Robert Murray, Dr. Wilbert Parker, Dr. Andrew Sinamark, Dr. Carlyle Tingdale,** and **Dr. Wesley Tomhave.**

The thirty-bed hospital has twelve bassinets, operating and delivery room, and laboratory and x-ray facilities.

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Dr. B. J. Kennedy, University of Minnesota, spoke on "Medical and Chemotherapy Treatment for Cancer," at a luncheon meeting of the Ramsey County Cancer Society held at the Athletic Club, Saint Paul, November 18.

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Dr. H. J. Just was named chief of staff and **Dr. Carl A. Weiss,** secretary of the staff, at a recent meeting to name officers for the Hastings Memorial Hospital staff.

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Dr. Malcolm M. Hargraves, member of the Mayo Clinic staff, known for his interest in soil conservation activities, was made an honorary member of the Soil Conservation Society of America at its national convention at Colorado Springs, Colorado, in December.

In 1948, Dr. Hargraves discovered the cell in human blood that made possible the diagnosis of lupus erythematosus.

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Dr. Henry Ulrich, who helped found the Hennepin County Tuberculosis Association fifty years ago, was recently presented with a plaque in the form of the well-known emblem of the Anti-Tuberculosis Committee of the Associated Charities. The presentation was made by **Dr. S. Marx White** at a luncheon held in Dr. Ulrich's honor at the Citizens Aid Building, Minneapolis.

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At a meeting of the Red River Valley Medical Society, held in Thief River Falls, November 24, **Dr. Davitt A. Felder** of University of Minnesota, spoke on "Diseases of the Blood Vessels." Following the dinner, wives of the doctors met for a short auxiliary meeting at the home of **Dr. and Mrs. William A. Feigal.**

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Dr. Joseph C. Michael and **Dr. John W. Schumacher** have been associated in the practice of psychiatry and neurology at the Medical Arts building, Minneapolis, since December 1.

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Dr. John G. Freeman, clinical director of the Fergus Falls State Hospital for the past two and a half years, has accepted a similar position with the State Hospital in Jamestown, North Dakota, where he will take over his new duties February, 1954.

Dr. Freeman participated as speaker at the fall meeting of the executive secretaries of the Minnesota Welfare Boards at the University of Minnesota Center for Continuation Study recently. He was also guest speaker at the American Public Welfare Association's session on "Community Planning for the Care of the Senile Patient" held in Chicago, Illinois, December 5.

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Dr. Donald J. McNairy, Phoenix, Arizona, spent the week of December 7 to 11 in Chicago as a member of the teaching staff at the annual meeting of the American Academy of Dermatology and Syphilology, his subject being "Medical Photography." Dr. McNairy received his Master of Science degree in Dermatology and Syphilology at the University of Minnesota in 1944.

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Dr. and Mrs. B. J. Cronwell, Jr., Austin, **Dr. Herman Miller,** Grand Meadow, and **Dr. P. J. Schneider,** Adams, met at the home of **Dr. Thomas M. Seery** in Austin, November 23, to take the first steps in organizing the Mower County Heart Council. Dr. Seery, designated acting chairman of the Minnesota Heart Association, said the object of the council will be to distribute vast amounts of educational material available through the Heart Association. The group plans a larger meeting for January in which civic and farm groups will be represented.

* * *

Dr. Moses Barron, Minneapolis, discussed developments in the Middle East at a general meeting of the Minnesota Emergency Committee on Israel of the Saint Paul Zionist district, in the Jewish Community Center, November 17.

* * *

Dr. Deane W. Benton, formerly of New Ulm, now specializing in psychiatry in Minneapolis, gave an illustrated talk on a "Laymen's View of Psychiatry" at a Rotary Club meeting held November 17 at the Silver Latch Cafe in New Ulm.

* * *

At the organization meeting of the "Catholic Physician's Guild," held in St. Cloud, November 30, with approximately thirty-five doctors in attendance, **Dr. W. T. Wenner,** St. Cloud, was elected president; **Dr. F. J. Schatz,** St. Cloud, vice president; **Dr. W. F. Skaife,** Little Falls, secretary, and **Dr. J. B. Gaida,** St. Cloud, treasurer.

* * *

Dr. Irvine McQuarrie, Professor of Pediatrics at the University of Minnesota Medical School, has been named one of tthe recipients of the 1954 Modern Medicine Award for Distinguished Achievement, Dr. Walter C. Alvarez of Minneapolis, editor-in-chief of the magazine, has announced. His contributions to the understanding of metabolic disturbance in children earned the award for Dr. McQuarrie. The awards are presented annually by *Modern Medicine* in recognition of significant work in the advancement of medical practice.

Dr. C. A. Undine, Minneapolis, attended the midwest regional meeting of the American College of Physicians at Milwaukee, Wisconsin, November 21, 1953.

* * *

Dr. G. S. Boyer, Crookston, radiologist for the Northwestern Clinic at Crookston, addressed Roseau physicians, recently, on "Infertility Studies."

* * *

Dr. John L. Delmore, Jr., Roseau, attended the orthopedics course at the Center for Continuation Study at the University of Minnesota, November 16 to 18, 1953.

* * *

Dr. A. C. Hilding, Duluth, was recently elected to membership in the Kunglia Fysiografiska I. Lund, honorary Swedish scientific organization which limits its membership to approximately 100 Swedish and 100 foreign members.

* * *

Dr. Louis L. Freidman, Saint Paul, participated in a symposium on "Surgery in Sterility" presented by the Western Division of the American Sterility Society in Palm Springs, California, November 6 to 8, 1953. Dr. Friedman also addressed the Lynn County Medical Society at Cedar Rapids, Iowa, November 12, on "Management of the Infertile Couple."

* * *

Dr. Robert S. Abernathy, Fellow in the Department of Medicine at the University Hospitals, Minneapolis, has been awarded a Research Fellowship in Medicine by the American College of Physicians. The appointment is effective July 1, 1954. He will work under the preceptorship of Dr. Wesley W. Spink, Professor of Medicine, on the pathogenesis of brucellosis. Dr. Abernathy is a graduate of Duke University School of Medicine. He interned in the University of Minnesota Hospitals, and following a year as Fellow in Medicine, he served two years in the army in Korea. Dr. Abernathy's wife, Dr. Rosalind Abernathy, is a part-time assistant in the Department of Pediatrics at the University Hospitals.

* * *

Dr. R. L. Parsons, formerly of Monterey, is now located in Glencoe, Minnesota, for the practice of medicine and surgery.

* * *

Dr. George H. Olds, New Richland, has been named a member of the Mental Health Clinic, which has its main office in Albert Lea.

* * *

Dr. E. F. Beyer, Braham, called into the army from his medical practice in May, has been promoted to the rank of captain. Dr. Beyer has been stationed with the Seventh Division in Korea since August.

* * *

Dr. Milton Moore Howell, previously in charge of a fifty-bed medical ward at the United States Public Health Hospital, Norfolk, Virginia, is now associated with **Dr. C. W. Truesdale** in the operation of the Glencoe Clinic. Dr. Howell is a graduate of the University of Rochester School of Medicine, Rochester, New York. Prior to his entering medical school, Dr. Howell was in the army from 1940 until 1945. Starting as a private he left the service a major.

* * *

Dr. George W. Snyder, Saint Paul, trustee of the American Legion's Hospital Association, pointed out at the opening session of the Minnesota fall conference, that dues allotted from membership fees are not sufficient to sustain the hospital program to be carried out by the association. This program, for wives and dependents of veterans who are financially unable to provide hospital care for their families, receives twenty-five cents of each member's dues, and five cents from each auxiliary member. Dr. Snyder urged formation of programs to raise funds for the association, especially during hospital week in February.

* * *

Dr. Raymond L. Page, St. Charles, was presented a merit award at the annual refresher course of the State General Practice Academy held at Hotel Lowry, Saint Paul, November 4. **Dr. Charles C. Cooper,** Saint Paul, is president of the group, which this year had more than 500 physicians in attendance at the sessions.

* * *

Dr. Louis Loes, who recently opened an office in St. Cloud for specialization in obstetrics, spoke at a meeting of the United Cerebral Palsy group of Central Minnesota, held in St. Cloud, December 12. He discussed problems of pre-natal care and delivery from the cerebral palsy viewpoint.

MINNESOTA BLUE CROSS-BLUE SHIELD

Of interest to all participating physicians are the improvements in both the present standard Blue Shield contract and its schedule of payments, which have been approved by the Board of Directors, for services rendered Blue Shield subscribers on and after January 1, 1954. In addition to these changes in the present standard contract, the Board expects to have a second contract with higher fees and higher service benefit annual income levels available for the public soon after March 1, 1954. The changes in the standard contract and fee schedule are the result of the Board's policy of improving the contract as rapidly as possible.

Dual Contract and Fee Schedule

A new standard contract and schedule of payments incorporating these improvements have been prepared. In the new contract will be clauses applying to the present standard contract, which will be known as Plan A, and also clauses pertaining to the new, higher fee, higher service benefit income contract, which will be known as Plan B. Principal differences in the two plans will be the fees paid and the annual incomes which determine eligibility for service benefits.

Although the new Plan B contract is not to be available to the public before March 1, 1954, in order to avoid duplication of printing and other costs, a new fee schedule containing both Plan A and Plan B fees has been prepared for early mailing to all participating physicians. Misunderstandings and other difficulties will be avoided if it is borne in mind that fees listed in the new schedule in the column headed "Fee Schedule B" do not apply to the present, or Plan A, contract.

Benefit Increases in Present Standard Contract

Service benefit annual income levels of the present contract are to be increased on January 1, 1954, from $2,000 to $2,400 for single subscribers and from $3,000 to $3,600 for family subscribers. It is believed that the increases in fees compensate physicians for providing service benefits for subscribers with these higher incomes. Prior to January 1, 1954, these annual income levels for service benefits are $2,000 and $3,000 for single and family subscribers, respectively.

When both husband and wife have a single contract, the annual income level which determines eligibility for service benefits will be the combined annual income of the family, and not the income based on the type of contract. Ambiguity has previously existed on this point. On July 1, 1953, this interpretation was approved by the Board of Directors and the contract is now being changed in this manner to clarify this ambiguity.

While the clause in the contract pertaining to in-hospital medical care limits such care, or combined medical and surgical care for the same condition, to thirty days at present, it is being changed to cover seventy days of such services rendered on and after January 1, 1954. As a result of this change, after January 1, 1954, as much as seventy days of care, instead of the present thirty days, will be provided each twelve months for every unrelated eligible condition or illness.

Waiting periods in the present group contract involving benefits for maternity care, pre-existing conditions, tonsillectomies and adenoidectomies for minor dependents, and for these and herniotomies and hemorrhoidectomies in the present non-group contract are of ten months' duration. These waiting periods are being decreased in the contract to only nine months on and after January 1, 1954. In other words, while claims for these conditions have not been eligible for payment until the subscriber has had a Blue Shield contract for ten months, claims for services rendered for these conditions on and after January 1, 1954, will be valid after the contracts are only nine months old.

Under the present contract, circumcisions are not valid claims unless the child is at least one year old. This will be changed so that circumcisions performed on and after January 1, 1954, will be eligible for payment in accordance with the fee schedule if done after the date of discharge of the mother from the hospital following the obstetrical delivery.

At present, services performed by dentists have not been valid Blue Shield Claims. Services performed by licensed dentists in Minnesota on and after January 1, 1954, in the treatment of jaw fractures, dentigerous cysts, adamantinomas, oral and extra oral drainage of alveolar abscesses will be eligible for Blue Shield allowances in accordance with the contract. Previously, such services covered by the contract were eligible for Blue Shield payment only when performed by a doctor of medicine.

Benefit Increases in the Present Schedule of Payments

In addition to the benefit improvements written into the present standard contract, an extensive revision of its fee schedule has made available more liberal allowances in surgical, medical and maternity care. In fact, of a total of ninety-five changes in the fee schedule, fifty-eight consist of fee increases, new items not listed before, or revisions of the method of payment. Most of the remaining changes represent bringing the description of the items up-to-date with present-day medical or surgical practice.

Especially important are twenty-seven distinct fee increases for different types of medical-surgical-obstetrical care. Of these, seventeen are for surgery and include $50 increases for seven difficult and more costly operations, and 10 increases in fees for recently developed and frequently performed operations. Also, six increases are for in-hospital medical care. Not only are benefits for each of the first two days of both routine and emergency in-hospital medical care being increased, but the total period of in-hospital medical care or combined in-hospital medical and surgical care for the same condition is being increased from thirty to seventy days for each period of twelve consecutive months. Maternity benefits are also being increased from $50 to $60, and three increases in anesthesia benefits are included.

Of the remaining changes, two are of particular importance to participating physicians. For treatment of sprains or strains on or after January 1, 1954, a fee of $5.00 is listed and this will permit allowances for the x-ray necessary for their diagnosis, in accordance with the contract. Also, circumcisions which have been excluded from benefits till the child is twelve months old, when performed after the date of the mother's discharge, and after January 1, 1954, will be eligible for a $10 fee till the child is three months of age, and a $15 fee from three months to six years of age. In addition to these increases, fourteen other new items are being added, and fifteen changes are being made to improve the methods of paying for foreign body removal, lacerations, burns and other surgical procedures.

No Increase of Rate for Present Standard Contract

All of these many improvements in the present contract and the schedule of payments are being made without any increase in the rate or cost of the present contract to the subscriber. These increased benefits mean several hundred thousand dollars more in benefits paid for subscribers' medical, surgical and maternity care each year. Improving the benefits without increasing the rates clearly demonstrates the Board of Directors' desire and intention of providing the greatest benefits at the lowest possible cost. This course of action is possible only with the full support and co-operation of all members of the medical profession.

BOOK REVIEWS

Books listed here become the property of the Ramsey, Hennepin and St. Louis County Medical Libraries when reviewed. Members, however, are urged to write reviews of any or every recent book which may be of interest to physicians.

BOOKS RECEIVED FOR REVIEW

SALT AND THE HEART. Edward T. Yorke, M.D. Attending Cardiologist, Alexian Brothers Hospital, Associate Cardiologist, St. Elizabeth Hospital, Dispensary Physician, Elizabeth General Hospital, Elizabeth, N. J.; Consultant in Medicine, Rahway Hospital, Rahway, N. J. 83 pages. Illus. Price $3.45, cloth. Linden, N. J.: Drapkin Books, 1953.

MENTAL HEALTH IMPLICATIONS IN CIVILIAN EMERGENCIES. Report of Subcommittee on Civil Defense, Community Services Committee, National Advisory Mental Health Council, May, 1953. 25 pages. Price 15c, paper cover. Washington, D. C.: U. S. Government Printing Office, 1953.

REVIEW OF PHYSIOLOGICAL CHEMISTRY. 4th Edition. Harold A. Harper, Ph.D. Professor of Biochemistry, University of San Francisco; Biochemist Consultant to Metabolic Research Facility, U. S. Naval Hospital, Oakland; Biochemist Consultant to St. Mary's Hospital, San Francisco. 328 pages. Illus. Price $4.00, paper cover. Los Altos, Calif.: Lange Medical Publications, University Medical Publishers, 1953.

A PRIMER OF CARDIOLOGY. By Geoge E. Burch, M.D., F.A.C.P., Henderson Professor of Medicine, Tulane University School of Medicine; Physician-in-Chief, Charity Hospital; Consultant in Cardiovascular Diseases, Oschner Clinic; Visiting Physician, Tuoro Infirmary, New Orleans. Second Edition. Thoroughly Revised, with 214 Illustrations, 339 pages. Cloth, $5.50. Philadelphia: Lea & Febiger, 1953.

This book is intended as a guide to the student's early clinical studies of heart disease, according to the author's preface. It is provocative and it emphasizes the use of physiological data plus logic to solve selected clinical problems. I am afraid this is not the publisher's concept of the book since he refers to it as "comprehensive in scope . . . (a) complete presentation . . . (with) emphasis on problems (of) daily practice." A modern book relating to cardiology is hardly complete if it contains not a single electrocardiogram. It is not emphasizing daily problems if it devotes fourteen pages to calculation of congenital shunts and three to angina.

The prospective buyer of the book, having in mind the author's classic monograph on electrocardiography which is similarly titled, is not likely to realize how little clinical wisdom he is purchasing. The electrocardiographic book is a primer and is practical and helpful to the practitioner. The volume being reviewed is none of these.

Even as a guide to the student the book has serious defects. For example, contrasting theories of congestive failure are presented, but the essential points of the theories are controverted in the telling. This surely is a postgraduate presentation of an interesting sort, but must be bewildering to the beginner. So much is made of physiological facts and graphs and so much stress is placed on the imperative need of more reliable data of a similar sort that the reader might not realize very notable clinical experiments have been presented in recent years directly beneficial to the science of cardiology: for example, the precise assessment of virtue in modes of treatment in infarction and the quantitative role of electrolytes in the failing heart. The medical student should be progressing steadily and rapidly to clinical considerations. Books can help, but a shiny display of mathematical formulae related to heart sounds, a forensic presentation of the author's attitudes on congestive failure (page 56), and an individualistic assessment of new techniques such as catheterization (page 219) are not likely to hasten the maturity we all yearn for.

The author's insistence on severe anemia as a sign of heart disease (meaning the latter is always present with former) and on classifying functional heart complaints as heart disease will antagonize many readers.

The pages contain some excellent discussions. The treatment of functional complaints is handled in a manner that suggests the vast clinical experience of the author. The discussion of tracheal tug in aneurism is one of the clearest the reviewer has read.

For the teacher of cardiology, even though a casual and informal one, the book should be useful. It is factual and it is an attempt at the correlation much discussed of basic science and medicine. For the student of undergraduate rank, truly physiological or clinical monographs are more suitable. For the practitioner, the book will be disappointing if not misleading. So much excellent writing has been done on heart disease it would be easy to employ the few idle hours in a busy life to better advantage.

W.B.M.

DISEASES OF WOMEN. By Robert James Crossen, A.B., M.D., F.A.C.S., Assistant Professor of Clinical Gynecology and Obstetrics, Washington University School of Medicine, Section Head of Unit Obstetrics and Gynecology, St. Louis City Hospital; Assistant Gynecologist and Obstetrician to Barnes Hospital. 10th ed. 935 pages. Illus. Price $18:50 (cloth). St. Louis: C. V. Mosby Co., 1953.

It is indicative of passing time that the new tenth edition of Diseases of Women for the first time bears the name of only one Crossen. Dr. Robert J. Crossen, as sole author, has accomplished a difficult task well. To revise a standard text, to bring it up to date and still maintain the same degree of excellence and completeness for which the original manuscript long has been known, is a monumental project. The new author, with his deletions and additions has greatly improved this already well-known book. Removing many of the pages devoted to operative gynecology, allied fields such as gastroenterology, and pathology remote from that of gynecology has not, as one would expect, made the text less complete but rather allows space for the addition of more pertinent and up-to-date data. The addition of Willard Allen's chapter of "Endocrine Control and the Ovarian Cycle," A. N. Arneson's detailed yet clear explanation of radiation therapy in cervical cancer, and the treatise on the psychosomatic aspects of gynecology from a down-to-earth viewpoint has greatly augmented the value of the book to student and practitioner alike.

Diseases of Women continues to be one of the most complete books of its kind. One seldom finds a more complete review of the anatomy, embryology, and physiology of pelvic organs even in texts devoted exclusively to those specific fields. Biochemistry and its relation to pelvic physiology is very detailed, more so than perhaps is necessary in a book of this character. The unusual section devoted to pitfalls in gynecological diagnosis alone should guarantee this edition a respected place on the shelf of the specialist as well as the general practitioner.

Perhaps this very completeness reveals itself as the only criticism the new edition demonstrates. To be used as a reference the countless paragraphs on theory of etiology, related physiology and embryology may prove to be a disquieting barricade. As a text for the student, a complete study reference for the specialist, and a thorough approach to diagnosis and treatment for the practitioner, Diseases of Women equals all and surpasses many comparable gynecological texts.

Richard R. Fliehr, M.D.

MAY'S MANUAL OF THE DISEASES OF THE EYE FOR STUDENTS AND GENERAL PRACTITIONERS. Revised and edited by Charles A. Perera, M.D., Associate Clinical Professor, College of Physicians and Surgeons, Columbia University, New York. 21st ed. 512 pages. Illus. Price $6.00. Baltimore: Williams & Wilkins Co., 1953.

The new twenty-first edition of May's Ophthalmic Manual maintains its high standing of efficiency in describing eye diseases and the latest treatments for them. The various ophthalmic operations are described and illustrated, including the newer ones for cataract, glaucoma and detachment of the retina. The book can be recommended not only for the student and practitioner, but also for the specialist.

E. R. Bray, M.D.

LIVING WITH A DISABILITY.—At Home—At Work—At Play. By Howard A. Rusk., M.D., and Eugene J. Taylor, in collaboration with Muriel Zimmerman, O.T.R., and Julia Judson, M.S. 191 pages. Illus. Price $3.50. New York: The Blakiston Co., Inc., 1953.

Dr. Rusk has had a great deal of experience with disabled people and has followed numerous cases for long periods of time. In this book he has brought out some of the means of help, which are simple in many cases and more complicated in others, but which will aid the person to be more independent. Many of the suggestions that he has, and has well pictured for physical aids, could be made in any home work shop and many of them could be varied slightly to fit the individual case.

The text of the book is interesting and easy reading, and the pictures are all largely self explanatory. It would be advisable for all doctors, and especially those who have patients with disabilities, to carefully review this book and use it as a reference for suggesting different types of aids to the handicapped.

John C. Feuling, M.D.

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Minnesota Medicine

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Volume 37 February, 1954 Number 2

FLOW AND PRESSURE IN THE ARTERIAL SYSTEM, THEIR HEMODYNAMIC RELATIONSHIP, AND THE PRINCIPLES OF THEIR MEASUREMENT

ERIK WETTERER, M.D.
Munich, Bavaria, West Germany

BY THE SYSTOLIC ejection of blood into the arterial system, the heart causes a flow of fluid, the rate and course of which depend in the first rank on the diastolic filling and the momentary muscular conditions of the ventricle, and on the arterial pressure. The ejected volume extends the arteries and raises the pressure above its diastolic value. This sudden event initiates a pulse wave in the elastic arteries which is propagated through the system up to its branches where wave reflections take place. At the end of systole, the pressure is usually increased in all arteries, each of them being augmented in volume and sharing in the systolic storage of blood. The subsequent elastic recoil of the arterial walls maintains the blood flow through the capillaries during diastole.

First, the course of blood flow generated by the contraction of the ventricle, and the pressure course in the arteries near the heart, have to be considered. As an example, a selection of tracings obtained by Wetterer and Deppe [49] is seen in Figures 1 to 4. The flow rate in the unopened ascending aorta has been recorded with an electromagnetic flowmeter, the pressure in the lower carotid artery with Broemser's optical manometer,[5] and the femoral pulse with a sphygmograph. The chest of the animals was opened.

In Figure 1, the tracings of pressure and pulse taken on a rabbit show some well-known facts. In each pulse, the pressure rises suddenly, reaches its maximum near the middle of systole, and then decreases towards the end of systole where the incisura appears. During diastole, the pressure is generally falling, but, due to a superimposed wave, there is an elevation in the first half of diastole, synchronous with a deep depression of the peripheral pulse. In this way, the standing wave in the aorta makes its appearance. The flow rate rises suddenly, too, reaches its maximum before the maximum of pressure, then turns back to zero and becomes negative for a very short time at the end of systole during the closure of the aortic valves. During diastole, the flow remains practically at zero.

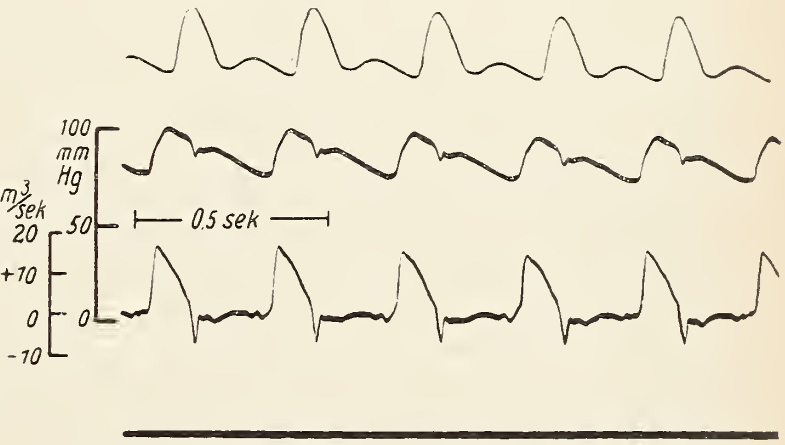

Fig. 1. Records taken on a rabbit. Tracings from bottom to top: flow rate in the ascending aorta, pressure in the carotid, femoral pulse.

A large stroke volume was obtained by adrenaline, as demonstrated in Figure 2. In this special case, the flow course is characterized by a plateau or two maxima, respectively. This is a very

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Presented in the Symposium on Recent Advances in Cardiovascular Physiology and Surgery, University of Minnesota, Minneapolis, September 15, 1953.

unique phenomenon which was observed in no other animal. In consequence of the high peripheral resistance and the increased pulse wave velocity, the course of pressure is remarkably elevated in the last part of systole by a large reflected wave, the onset of which is clearly seen. The relatively small pressure fall after the systolic maximum and the curved descent of pressure during the prolonged diastole are obvious.

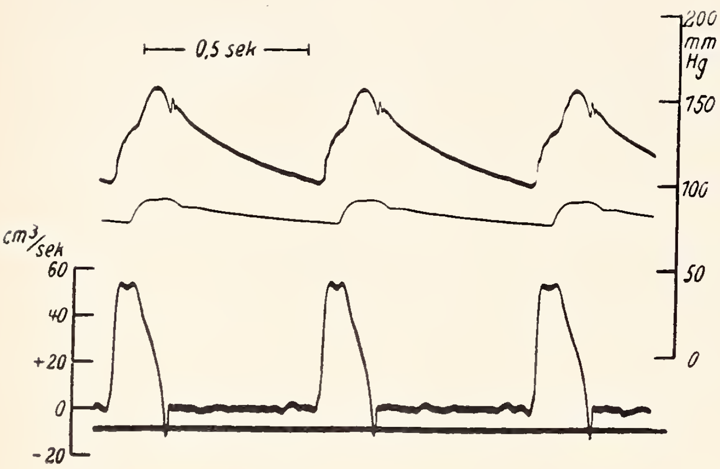

Fig. 2. Records taken on a rabbit after injection of adrenaline. Tracings from bottom to top: flow rate in the ascending aorta, femoral pulse, pressure in the carotid.

An opposite state of circulation is seen in Figure 3 after the injection of pilocarpine. The peripheral resistance and the mean pressure are low, and the pulse wave velocity is slow. At the end of systole, there is only a minute negativity of flow and, therefore no visible incisura in the pressure curve. After the relatively deep descent from the systolic pressure maximum to the end of systole, the diastolic pressure course is almost horizontal. The time interval between the maxima of flow and pressure is very short. In this case of low pressure level, the courses of pressure and flow are nearly congruent, if presented on the same scale.

Figure 4 shows a record obtained on a dog of 15 kgm. The maximum of flow rate approaches 200 cc. per sec. Attention is to be called to the fact that there are no significant differences in shape of the normal flow course between rabbits, cats, dogs, and monkeys. It may be concluded that the contour of flow course in the human ascending aorta agrees in the principle with those which have been recorded on these animals.

Obviously, the integral of the flow rate (that is the area enclosed by the curve of the flow rate and by the base line of flow) represents the total stroke volume minus that minute part flowing through the coronary arteries during systole. The cardiac ejection curve (that is the time course of volume ejected by the ventricle) results from the stepwise integration of the flow curve from the beginning to the end of ejection time. Some ejection curves, obtained in this way, are seen in Figure 5. Above, the original curves of flow rate. The ejection curves are S-shaped. Generally, the steepest point which corresponds to the maximum of flow rate is situated within the first fourth or third of the ejection time. On the right hand of Figure 5, there is an ejection curve calculated by Remington and Hamilton[40] with their pulse contour method. On the whole, the differential quotient of this calculated ejection curve agrees well with the recorded flow curves although the maximum of the recorded flow course is usually reached somewhat earlier than that of the calculated curve.

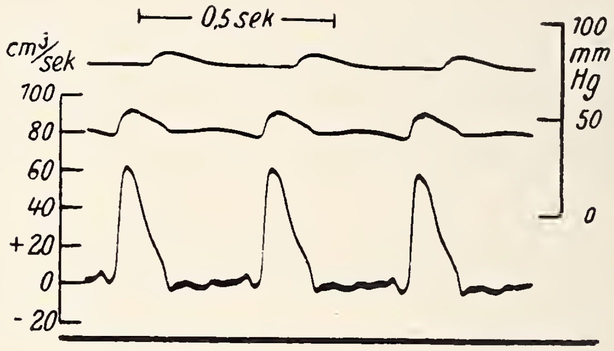

Fig. 3. Records taken on a rabbit after injection of pilocarpine. Tracings as in Figure 1.

Because of the intricate structure of the arterial system, the theoretical treatment of the hemodynamic events is faced with many difficulties. Therefore, several authors tried to formulate the fundamental laws concerning pressure and flow by the study of simplified models. Two different models have proved valuable: the so-called Windkessel and on the other hand, a long elastic tube filled with fluid through which pulsewaves are propagated. The Windkessel model, as shown in Figure 6, consists of a distensible reservoir, representing the distensibility of the total arterial system, an inflow valve representing the aortic valves, and a narrow outlet tube representing the peripheral resistance. Instead of the distensible reservoir, an air-filled rigid box can be used; hence the name air-chamber or Windkessel. If fluid is injected rhythmically by means of a pump, imitating systole and diastole of the heart, the pressure in the Windkessel rises and decreases rhythmically. In 1899, Frank[10] described the relation between rate of inflow i and pressure p by the equation noted in Figure 6 at the right hand. D is the distensibility, that is the ratio of a change in volume to a change in pressure, and R is the resistance. It is seen that the inflow rate is resolved into storage rate i_1 and an outflow rate i_2. i_1 distends the elastic wall and results in an uptake of fluid, while i_2 represents the drainage out of the system.

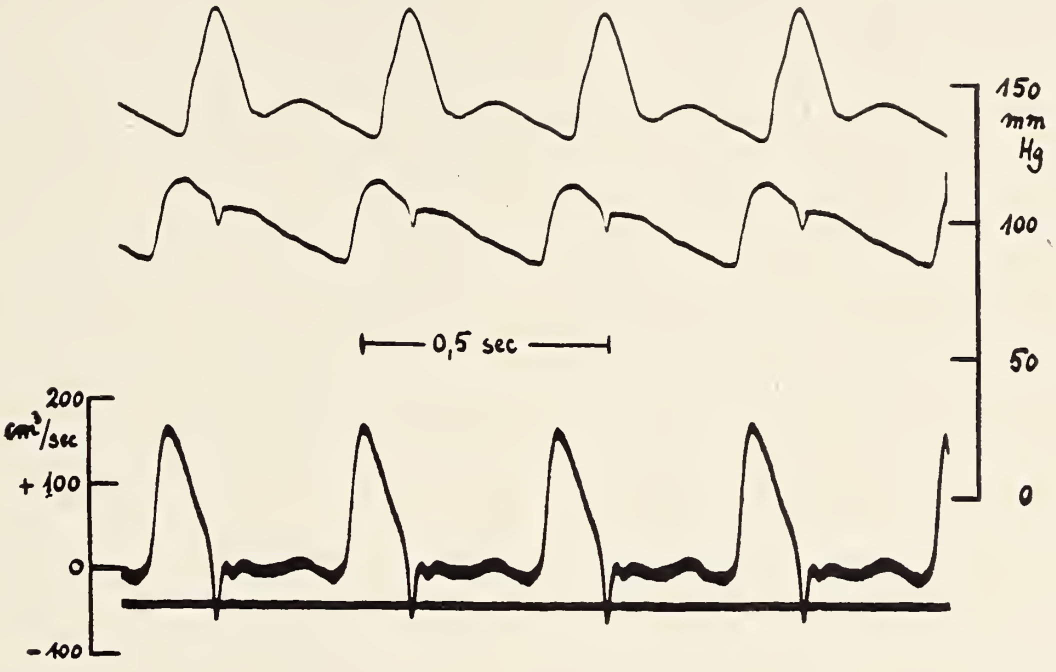

Fig. 4. Records taken on a dog of 15 kgm. Tracings as in Figure 1.

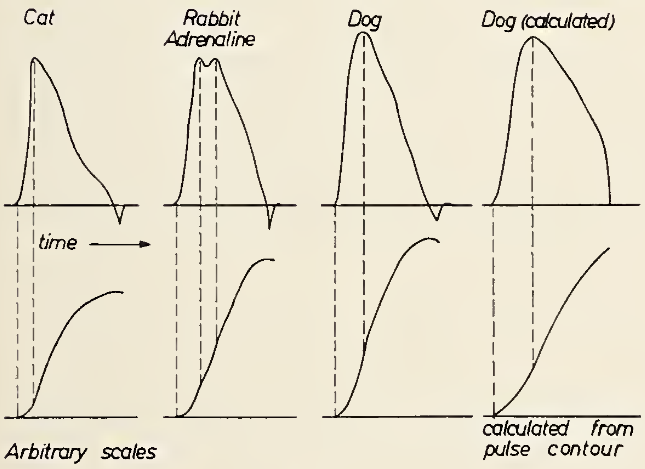

Fig. 5. First, second, and third curve from left to right: Derivation of the cardiac ejection curve (below) from the recorded course of flow rate in the ascending aorta. Last curve at the right hand: Ejection curve (below), calculated by Remington and Hamilton from the pulse contour, and its differential quotient (above).

The pressure course in the Windkessel results from the integration of Frank's differential equation under the assumption of a certain flow course. Thus, a slowly ascending S-shaped pressure course results during systole. The diastolic pressure descent describes, as Frank stated, an exponential curve, if the product resistance times distensibility is constant during diastole. In the limits of a normal diastole, the curvature of the diastolic pressure course is only slight so that it approaches a straight line.

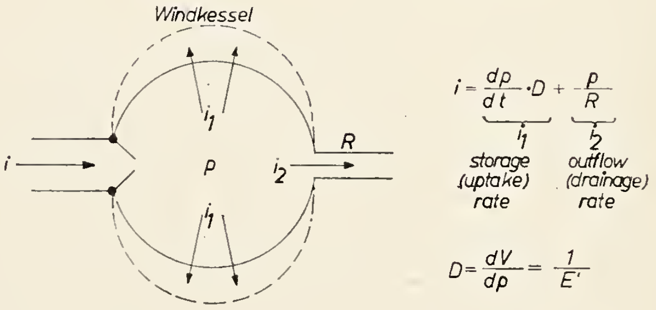
Windkessel
i
i_1
p
i_2
R
$i = \underbrace{\frac{dp}{dt} \cdot D}_{i_1} + \underbrace{\frac{p}{R}}_{i_2}$
storage (uptake) rate
outflow (drainage) rate
$D = \frac{dV}{dp} = \frac{1}{E'}$

Fig. 6. Design of the simple Windkessel model.

Wetterer[50] emphasized that the systolic pressure course in the Windkessel is quite different from that in the normal arterial system. Nevertheless, the following time correlations between flow and pressure during systole are valid for both the Windkessel and the arteries: the maximum of flow precedes the maximum of pressure; the maximum of pressure precedes the maximum of ejected volume. These relations hold true with regard to the total arterial system as well as to its single branches.

The Windkessel model also permits the prediction of the effects of changes in stroke volume or minute volume, in distensibility or resistance on the mean pressure level and on the pulse pressure. In this regard, Wiggers[61] investigated similar physical models in an experimental way in 1938.

It was mentioned above that the Windkessel model is unable to deliver a systolic pressure course analogous to that of the normal arterial system. However, an extremely high peripheral resistance can give rise to an arterial pressure contour similar to that in the Windkessel, for instance, in cases of renal hypertension as recently published by Remington.[41] But usually, the arterial pressure during systole ascends much steeper, reaches its maximum earlier and decreases, after the maximum, to a greater extent than the pressure in the Windkessel does. In 1940, Wetterer[50] tried to explain these differences between the model and the natural circulation system by the following consideration: In all parts of the Windkessel reservoir the pressure is the same at a certain moment so that the total distensibility is effective during the entire pulse time. But in the arterial system, the distensibility is variable and depends on the distance covered by the pulse wave. In the beginning of the systolic inflow, the pulse wave starts from the aortic root. For instance, 10 milliseconds after, the top of the wave will have passed through about four centimeters. Only the distensibility of this short part of the aorta will be available for the uptake of blood injected during the first 10 milliseconds. The longer the distance covered by the pulse wave, the greater will be this part of the arterial system in which uptake occurs. Consequently, the actual distensibility during systole is variable and a function of time. Wetterer calculated this variable distensibility from the pulse wave velocity and the cross sectional area of the aorta under certain simplified assumptions. Furthermore, he supposed the drainage rate obeying two different laws: first, the simple law of Poiseuille, and second, another relation resulting from the consideration that the drainage rate increases with the number of arterioles reached by the pulse wave. In this way, the flow course was calculated from the pressure course and inversely, and in many cases a satisfactory correspondence between the calculated and the recorded curves was obtained. On the other hand, this calculation was failing in cases of a very high or very low peripheral resistance. Therefore, it may be stated that this extension of the simple Windkessel theory improves our classical model to a certain degree, but cannot take into account all variations occurring in circulation.

Independently of these considerations, Hamilton and Remington[21] developed in 1946 and 1947 a new method of calculating the cardiac ejection curve from the pressure course. The distensibility used in their calculations was not derived from the pulse wave velocity, but from volume pressure relations directly determined on dead animals. The arterial system was divided into six main beds, the individual volume uptakes of which were tabulated at different pressure levels. Also the pulse transmission time to and through each bed was listed at various pressures. Using these statements, the authors computed the time course of the volume uptake from the pulse pressure recorded on living animals. Obviously, the contribution of the separate beds to the momentary uptake varies considerably in the course of systole according to the distance covered by the pulse wave, and the variation of the actual distensibility as a function of time, is a striking feature of this method too. Furthermore, Hamilton and Remington described the systolic drainage of blood as a complex function of the distance covered by the pulse wave and of the arterial pressure. The total ejection curve, resulting from uptake plus drainage, was found to be an S-shaped curve as demonstrated in Figure 5 and also confirmed by cardiometer curves of Wiggers and Katz.[59]

The percental contribution of the various arterial beds to the total distensibility, as it may be quoted from the tables of Hamilton and Remington, is very different. The entire aorta comprises at the most 50 per cent of the total distensibility, the arteries of head, neck, and forelegs nearly 30 per cent, the visceral plus hindlimb arteries about 20 per cent. Of course, also the smallest arteries of each bed contribute to the distensibility.

Wetterer and Pieper[54] developed in 1952, a direct method for determining the total arterial distensibility on living anesthetized animals. A piston pump filled with Ringer solution was connected to the aorta by a tube introduced through the carotid. The piston moved forward and backward and effected rhythmic sinusoidal volume changes; each pump cycle lasted three to five seconds. The volume shift of the piston was adjusted to the size of the experimental animal. In dogs of 8 to 30 kgm. volume changes of 10 to 30 cc. were used. The blood pressure in the aortic arch, recorded with a miniature manometer,[52,53] increased and decreased with the rhythm of the pump motion. On these relatively slow fluctuations, the arterial pulses were superimposed, as shown in Figure 7. The pulse transmission time through the whole arterial system is exceedingly short compared to the duration of a pump cycle. Therefore, the arterial system reacts to the pump like the uniform reservoir of a simple Windkessel model. For evaluation, the measurements were carried out on the diastolic parts of the pulses. Within a pump cycle of the aortic pressure course, two pulses were selected, one in the ascending part and the other one in the descending part. The mean pressures during the diastoles of both pulses should not differ from each other by more than 2 per cent so that the mean diastolic drainage rates may be supposed to be equal. The distensibility of the total arterial system was given by the formula:

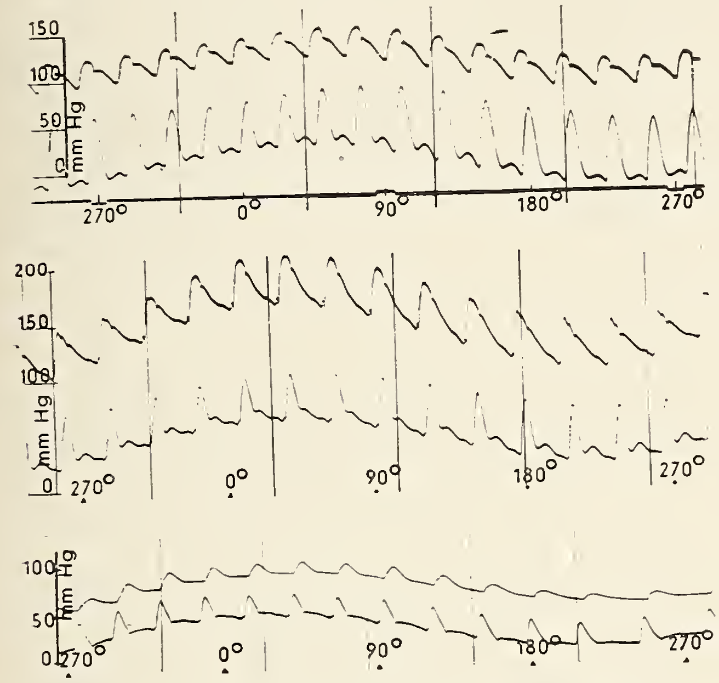

Fig. 7. Tracings obtained on anesthetized dogs during the action of the pump (see text). Above: normal state; aortic pressure and femoral pulse. Middle: high pressure during infusion of adrenaline after atropinisation; aortic pressure and femoral pulse. Below: low pressure due to a ganglionic blocking drug (Pendiomid); femoral pulse and aortic pressure.—Distances between the vertical lines = 1 sec.—Numbers below zero-line indicate the angle-degrees corresponding to the sinusoidal movement of the piston. 270° = lowest position, 90° = highest position of the piston.

$$D = \frac{i_1 - i_2}{S_1 - S_2}.$$

i_1 and i_2 are the flow rates of the pump during the diastoles of the two pulses, calculated by means of a sine table. S_1 and S_2 are the diastolic pressure slopes of the two pulses. The drainage cancels out in this formula because it is the same for both pulses. The evaluation of ten to twenty pairs of pulses in every record containing several pump cycles gave a standard deviation of about 8 per cent and a mean standard error of less than 3 per cent.

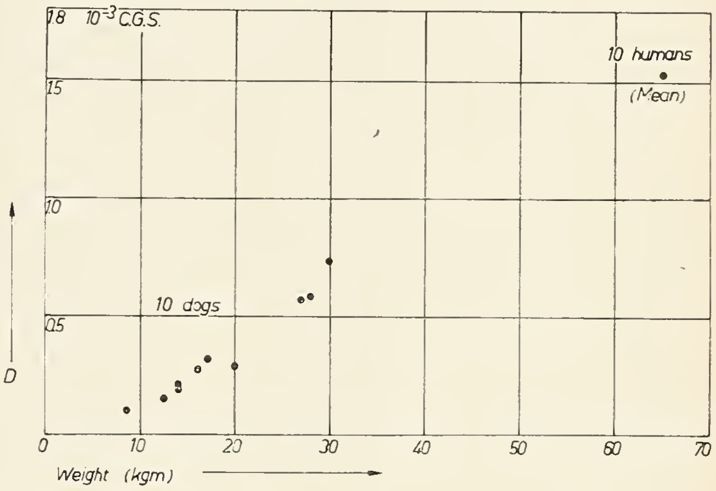

Fig. 8. Total arterial distensibility of ten anesthetized dogs, determined with the pump method. Above at the right hand: Mean of arterial distensibility, determined on ten humans by Warner *et al.*

Figure 8 shows a summarizing diagram of the results obtained on ten dogs of body weights between 8.5 and 30 kgm. in a normal state of circulation. The distensibility, expressed in C.G.S. units, is plotted against the body weight in kgm. The relation between both variables may be regarded as a curve passing through the origin of the co-ordinates and becoming steeper in the range of greater body weights. As far as it can be seen from ten observations, the distensibility is not highly influenced by differences in age and sex of the dogs. The amount of distensibility measured on dogs of 28 to 30 kgm. agrees well with the data calculated from the tables of Hamilton and Remington[21] under the condition of a normal pressure level. Moreover, the results obtained on dogs with the pump method may be substantiated by the investigations of Warner, Swan, Connolly, Tompkins, and Wood[36] who computed on humans, the arterial distensibility from the aortic pressure pulse and from the cardiac output determined by Fick's principle of the dye method. The mean of distensibility determined on ten normal humans and expressed in C.G.S. units is noted in Figure 8 under the assumption of a mean body weight of 65 kgm. Obviously, an extrapolation of the values measured on dogs points almost directly to the mean of human arterial distensibility.

If the distensibility of the entire human arterial system should be simulated by an air-chamber model, an air-cushion of 1.5 to 2 litres would be required.

Now, the arterial pressure course during diastole may be discussed. It was explained above that in diastole the descending pressure in the simple Windkessel follows an exponential course which represents almost a straight line. In the arterial system, the diastolic pressure course resembles a straight line, too, if the diastole is not abnormally long, and if pressure changes induced by wave reflections do not interfere. At a certain pressure level, the slope of this course is reciprocal to the product distensibility times peripheral resistance. An undisturbed diastolic pressure course may be obtained at the node of the main standing wave in the aorta.

If, in an abnormally prolonged diastole, the pressure falls over a wide range, we find a diastolic pressure course which follows a strongly curved line. Extrapolating this curve beyond the end of diastole, one does not reach values which correspond to the capillary or venous pressure. Rather the extrapolated curve seems to be asymptotic at a level which is far above the capillary pressure. Several reasons explain this fact: First, the arterial distensibility is a function of pressure; it increases when the pressure decreases. Second, the peripheral resistance, too, is a function of pressure because the lumen of the arterioles decreases with falling pressure. Both phenomena result in a rise of the product distensibility times resistance. Hence, the pressure course is flattened. Furthermore, in arterioles and capillaries the relation between pressure and flow of blood does not obey Poiseuille's law because blood deviates from the properties of a homogeneous viscous fluid in narrow vessels. As to the diastolic pressure course, see the publications of Frank,[12] Wiggers,[62] Hamilton,[24] Gomez,[17] and Deppe.[8] The complex relationship between pressure and flow through the arterioles and capillaries has been studied chiefly by Hess,[23] Whittacker and Winton,[58] A. Müller,[32,33] Green and co-workers,[18] Pappenheimer and Maes,[34] Lamport,[28] Rashewsky,[38] and recently by Wezler.[56,57]

In the following, the arterial pulse waves shall be discussed. The theory of waves running through elastic tubes has been founded by the brothers Weber,[47] by Moens,[31] Korteweg, and others during the nineteenth century. Several authors contributed to its further improvement in the twentieth century, chiefly Frank[14] since 1920. Also Apéria,[2] in collaboration with a mathematician, Müntz, and recently Lambossy[27] were working on the wave theory. Landes[29] demonstrated that the fundamental hydromechanical wave equations are analogous to those which are valid for the transmission of electric impulses through telegraph-cables. The mathematical wave theory cannot be given here; the explanations may be restricted to some simplified considerations.

If a certain amount of fluid is pumped quickly into an elastic tube containing fluid, the whole column in the tube resists to a sudden dislocation due to its inertia. First of all, the wall of the initial part of the tube is distended by the injected volume. Then the elastic force of the extended wall causes a shift of fluid to the next segment, and so on. Therefore, the extension is propagated along the tube and is accompanied by a pressure rise and a movement of fluid from one segment to the next one. If there is no wave reflection, the courses of wave-pressure and wave-flow will be in phase. This is very important for the understanding of the contour of certain pressure pulses. The ratio of pressure to flow rate of the wave is a sort of impedance and may be called the wave-resistance R_w:

$$R_w = \frac{\delta . c}{A}.$$

δ is the density of the fluid, c the wave velocity, and A the cross sectional area of the tube. Friction and viscosity are not considered in this formula. Another formula developed by Frank[14] in 1920, and by Bramwell and Hill[4] in 1922, shows the direct relation between the wave velocity, the density and the so-called volume elasticity modulus α. The latter represents the reciprocal of the distensibility per unit volume.

$$\alpha = \delta c^2.$$

If the tube wall effects a viscous resistance besides its elasticity, the volume elasticity modulus and the pulse wave velocity depend to a certain extent on the quickness of stretching. Concerning the arteries, Ranke[37] investigated this phenomenon in a theoretical and experimental way. It gives rise to effects of hysteresis, particularly in the first part of systole.

The formula of Frank and of Bramwell and Hill has been used, and also discussed by many investigators, because it renders the possibility to calculate the arterial distensibility from the pulse wave velocity. The application of this formula postulates the measurement not only of the wave velocity, but also that of the total tube volume V or its equivalent:

$$D = \frac{V}{\alpha} = \frac{V}{\delta c^2}$$

The measurement of V is difficult in the case of arteries *in situ* because the arterial diameter is not the same in all sections and depends on the pressure and on the vasomotor tone of the muscular vessels.

In order to obtain an equivalent of volume V, Frank and his associates multiplied the cross sectional area of the ascending aorta by a certain length. Broemser and Ranke[6] supposed that this length was represented by the distance covered by the pulse wave during the ejection time. Frank[15] used the half of the wave length of the main standing wave, Wezler and Böger[55] only one-fourth of this wave length. Bazett and co-workers[3] tried to improve the method by a more detailed consideration of central and peripheral arteries. On the average and under normal circulatory conditions, the values of distensibility, obtained on dogs by Frank's calculation, agree with those determined by the pump method of Wetterer and Pieper[54] which has been described above. The values calculated with the method of Broemser and Ranke are similar or somewhat larger; those of Wezler and Böger are smaller than Frank's data. The calculations of the arterial distensibility from the pulse wave velocity were used for the estimation of the stroke volume in different ways, the validity of which has been examined by many comparison experiments. In numerous cases a satisfactory agreement with the results of the comparison methods was observed; in other cases, the formulas gave remarkable deviations. This is not surprising because, up to now, there is no hemodynamic calculation which respects all the variable properties of the arterial system. Not only the calculation of the arterial distensibility from the wave velocity involves certain difficulties, but also the determination of the ratio of systolic to diastolic drainage may imply some errors, particularly in cases of high pulse pressures and a high arteriolar vasomotor tone.

The pulse contour method of Hamilton and Remington[21] is based on more experimental data than on indirect calculations. Its fundamental conception consists chiefly in the direct and detailed measurement of the arterial distensibility and in the consideration of the pressure distribution in the arteries at the end of systole. About the latest development and the range of applicability of this method see the paper of Dr. Remington in this volume.

Reflections of tube waves occur when the wave resistance, that is the quotient c/A, is changing from one tube segment to another. Generally, this quotient increases from the heart to the periphery so that we conclude that wave reflections take place within the whole arterial system, especially in the arms and legs. Since the increase in wave resistance is highest in the terminal arteries, the major part of all reflections must occur here. It is obvious that the vasomotor state of these vessels determines the amplitudes of the reflected waves.

In his interesting conception, Remington[41] presented the arterial system as to be fused into a single funnel-shaped tube. The length co-ordinate of this figure corresponds to the pulse transmission time, and its local diameter to the distensibility per unit transmission time. It can be demonstrated by a simple calculation that this diameter is inversely proportional to the wave-resistance.

Reflected waves running to the aortic root are reflected anew. The interference of the primary pulse wave with reflected waves, or of reflected waves with one another, gives rise to standing waves which have been described first by Frank.[12,15] The main standing wave, thoroughly investigated by Hamilton and Dow,[20] is generated between the heart and the leg arteries. It is characterized by periodical damped undulations which are superimposed on the fundamental pressure contour. In the arteries near the heart, the phase of these undulations is opposite to that in the lower aorta and its branches. Between both regions of opposite phase we find a node which is situated in the thoracic or upper abdominal aorta. The main standing wave comprises half a wave length,[15] or, in other words, the natural period of the standing wave is twice the pulse transmission time from the heart to the peripheral end of the standing wave. Furthermore, the pulse transmission time from the heart to the node equals that from the node to the peripheral end.[42] In man, the node is usually situated in the upper abdominal aorta[42,24] and can be shifted towards the heart by compression of the leg arteries below or above the knees.[25]

Recently Alexander[1] assumed the standing wave in the aorta to be influenced by a negative wave reflection at the level of the diaphragm where large arteries are branching off from the aorta.

The pressure amplitudes of reflected waves increase if the peripheral resistance is raised, whereas the reflections can be minimized by an extreme vasodilation. The shape of the central pressure pulse is highly influenced, not only by the cardiac ejection course and the drainage, but also by reflected waves, as shown in the Figures 1 to 4. In the state of an increased peripheral resistance and a high pressure level, the pressure will not descend in the second half of systole, but remain at a plateau or even arise to a late maximum because of the superposition of reflected waves. On the other hand, a low peripheral resistance effects a pressure fall from the early systolic peak to the incisura so that the systolic pressure course is similar to the flow course in the ascending aorta. In case of the first extreme, the arterial system approaches the simple Windkessel because the high pulse wave velocity and the multiple and pronounced reflected waves effect an almost uniform filling. The second extreme, however, is characterized by insignificant wave reflections so that the arterial system approaches the simple long tube model in which the course of pressure corresponds to the course of flow. It is seen that the properties of the normal arterial system are intermediate between both extremes and that a combination of the Windkessel and the wave theory is required for the complete understanding of hemodynamics.

The pressure contour in peripheral arteries, for instance in the leg arteries, is generally characterized by a steep and long systolic upstroke, by an increased pulse pressure, and by one or two distinct diastolic undulations, called dicrotic waves, which are related to the standing waves. The shape of upstroke is chiefly caused by the facts that the peak of the pulse wave is propagated more quickly than the foot, and that steady wave reflections raise the pulse pressure. However, the mean pressure is almost the same in all large and middle arteries, provided that the body is in a horizontal position. The main pressure drop takes place in the arterioles.

Within the limits of this brief survey, it is not possible to describe all the details of the pressure course and their changes along the arterial system which have been investigated by many authors, chiefly by Frank and his associates, by Wiggers, by Hamilton, and recently by Laszt and Müller.[30] The so-called water hammer pulse has been analyzed by Alexander[1] and by Peterson.[35] Further particulars concerning pressure and flow may be mentioned in the following methodical notes:

The principles of modern membrane manometers, established by Frank[11] since 1903, are well known. The natural frequency depends on the volume elasticity coefficient of the membrane and on the effective mass of the fluid in the connecting tubes. Well approved optical manometers have been designed by Frank,[13] Broemser,[5] Wiggers,[60] Hamilton.[19] Electrical manometers are advantageous in several respects. Their sensitivity can be adjusted over a wide range only by change in amplification, and the manometers may be operated independently of the distance from the recording camera. The deformations of the membrane can be transduced into electrical magnitudes by changing the electric resistance of a stretched wire, by changing the capacitance of a condensor or the inductance of an electric choke. Also the variable coupling of the two sections of a differential-transformer may be used. Another way of transducing mechanic into electric magnitudes consists in the photo-electric effect. Furthermore, special electronic tubes have been developed in which an internal electrode can be shifted from outside by the manometer membrane.

Special technical problems arose by the cardiac catheterization first performed on man by Forsmann and worked out for clinical use by Cournand.[7] A long and narrow catheter attached to the manometer lowers the natural frequency of the recording system. Furthermore, the catheter becomes distensible if warmed up to body temperature. A suitable damping ratio is required if distortions of the tracings shall be avoided. Hansen[22] published a theoretical treatise about these problems. After Wood,[63] the artefracts caused by impacts on the catheter in the beating heart may be reduced sufficiently by a damping ratio which gives the manometer system a uniform dynamic response up to 10 cycles per second and a sharp cutoff in sensitivity above this range. A manometer adjusted in this way is able to record the practically important components of the human pressure pluse; but, of course, the particulars of higher frequencies will not be reproduced.

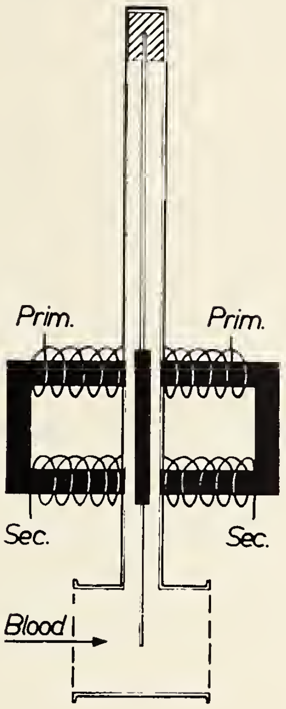

Fig. 9. Sketch of the hydrometric pendulum developed by Pieper and Wetterer. The short horizontal tube is inserted into a blood vessel. The pendulum itself is suspended elastically in the side tube and carries a small slug of iron which, in case of deflection, changes the mutual inductance of the differential-transformer. The natural frequency of the pendulum is more than 200 cps.

A natural frequency of 500 to 1,000 cps, and an artefact-free performance are obtained by miniature manometers attached to the tip of a cardiac catheter. Former devices have been published by Schütz[43] and by Wagner.[45] Wetterer[52] adopted the principle of recording small displacements with the help of differential-transformers for the construction of a miniature manometer with a high natural frequency. This instrument has been improved and adapted for use in humans by Gauer and Gienapp.[16] Ellis, Gauer, and Wood[9] examined its properties in comparison with a strain gauge type.

The measurement of blood flow involves greater difficulties than that of pressure. The mean rate of flow may be measured by instruments called "Stromuhren." Some of them are based on the principle of Ludwig's Stromuhr or on Shipley's[44] rotameter type, and are inserted into an opened vessel while Rein's Thermostromuhr[39] is applicable to unopened vessels. Another sort of flowmeters is able to record the pulsatile flow with all its instantaneous variations. This kind, based on hydromechanic or electric principles, may be called "tachographs." The hydromechanical devices are founded on friction and inertia of the moving blood. For instance, a pressure drop is effected when the blood is led in a Venturi-tube from a wide to a narrow segment. This pressure drop, recorded with a differential-manometer, equals the sum of a term proportional to the blood velocity and another term proportional to the square of velocity. A similar effect is obtained by the Pitot-tubes and their variations. The so-called hydrometric pendulum consists of a minute lever which is suspended elastically in a right angle to the direction of blood flow, and is deflected by the moving blood. In the electromagnetic flowmeter [Kolin 1936;[26] Wetterer 1937[48]], the blood moves through a magnetic field so that potential difference is induced which is proportional to the blood velocity. The application of an alternating magnetic field, introduced by Kolin, has added considerably to the further improvement of this method.

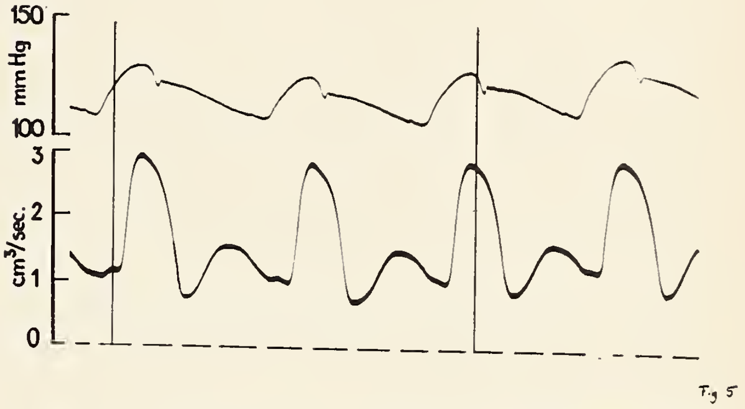

Fig. 10. Course of flow rate in the femoral artery of a dog, recorded with the device of Fig. 9. Above the pressure course in the aortic arch.

Finally, an improved model of the hydrometric pendulum, recently developed by Pieper and Wetterer,[36] may be demonstrated. The deflections of the pendulum are transduced into electrical magnitudes by means of a special differential-transformer as shown in Figure 9.

The course of flow rate in the femoral artery of a dog, recorded with this device, is seen in Figure 10. There is a relatively high diastolic flow rate and a diastolic flow wave which is related to the standing wave.

Figure 11 shows the normal flow course in the carotid. It contains the incisura, coincident with a decline between two maxima. The maximum of carotid flow precedes the maximum of pressure in the aortic arch. The diastolic undulations of flow are probably caused by pressure undulations of the main standing wave. But these undulations of flow are much more pronounced than those of pressure. Towards the end of diastole, the flow rate decreases almost corresponding to the diastolic pressure descent.

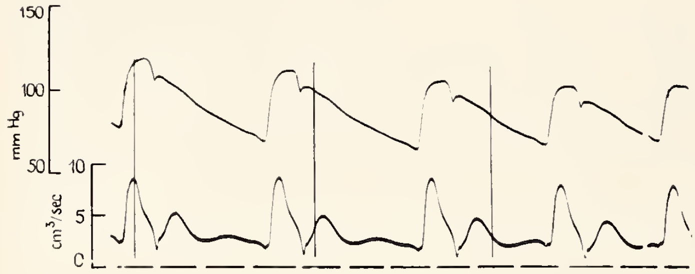

Fig. 11. Normal flow course in the carotid of a dog, recorded with the device of Fig. 9. Above the pressure course in the aortic arch.

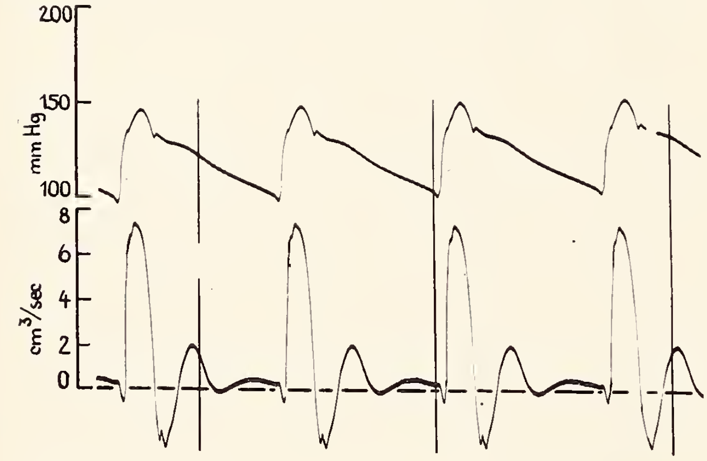

Fig. 12. Caritod flow course after injection of adrenaline. Above the pressure in the aortic arch.

The effect of a moderate dose of adrenaline is seen in Figure 12. In contradistinction to the previous figure, the mean diastolic flow rate is very low. It must be concluded that, under the condition of an increased peripheral resistance, the main portion of drainage takes place during systole; for the high pressure during systole has a much stronger opening effect upon the constricted arterioles than the lower pressure during diastole.

In the present state of hemodynamics, we are not able to explain all the details concerning the relations between pressure and flow in a quantitative way. But we hope that by improvement of flowmeters we will be able to record pressure and flow simultaneously in many parts of the arterial system in order to obtain better understanding of their interrelationship.

References

1. Alexander, R. S.: Federat. Proceed., 11:738, 1952; Cir. Research, 1:145, 1952.
2. Apéria, A.: Skandinav. Arch. f. Physiol., 83:Suppl. 16, 1940.
3. Bazett, H. C.; Cotton, F. S.; Laplace, L. B., and J. C. Scott: Am. J. Physiol., 113:312, 1935.
4. Bramwell, J. C., and Hill, A. V.: Proc. Roy. Soc., 93:298, 1922.
5. Broemser, Ph.: Ztschr. f. Biol., 68:1, 1918; 71:281, 1920.
6. Broemser, Ph., and Ranke, O. F.: Ztschr. f. Biol., 90:467, 1930; Ztschr. f. Kreislaufforsch, 25:11, 1933.
7. Cournand, A., and Ranges, H. A.: Proc. Soc. Exper. Biol. & Med., 46:462, 1941.
8. Deppe, B.: Ztschr. f. Biol., 100:437, 1941.
9. Ellis, E. J.; Gauer, O. H., and Wood, E. H.: Circulation, 3:390, 1951.
10. Frank, O.: Ztschr. f. Biol., 37:483, 1899.
11. Frank, O.: Ztschr. f. Biol., 44:445, 1903; 50:309, 1907.
12. Frank, O.: Ztschr. f. Biol., 46:441, 1905.
13. Frank, O.: Ztschr. f. Biol., 82:49, 1924.
14. Frank, O.: Ztschr. f. Biol., 71:255, 1920; 85:91, 1926; 85:102, 1926; 88:105, 1928.
15. Frank, O.: Ztschr. f. Biol., 90:405, 1930.
16. Gauer, O. H., and Gienapp, E.: Science, 112:404, 1950.
17. Gomez, D.: Les lois physiques de l'hémodynamique. Rech. d'Hémodynam. et Cardiol., 511, Paris, 1937.
18. Green, H. D.: *In* Glasser-Medical Physics. p. 208, 1944.
19. Hamilton, W. F.; Brewer, G., and Brotman, J.: Am. J. Physiol., 107:427, 1934.
20. Hamilton, W. F., and Dow, Ph.: Am. J. Physiol., 125:48, 1939.
21. Hamilton, W. F., and Remington, J. W.: Am. J. Physiol., 148:14, 1947.
22. Hansen, A. T.: Verhandl. d. deutsch. Gesellsch. f. Kreislaufforsch., 15:97, 1949.
23. Hess, W. R.: Pflügers Arch. f. d. ges. Physiol., 162:187, 1915.
24. Kapal, E.; Martini, F., and Wetterer, E.: Ztschr. f. Biol., 104:256, 1951.
25. Kapal, E.; Martini, F.; Reichel, H., and Wetterer, E.: Ztschr. f. Biol., 104:429, 1951.
26. Kolin, A.: Proc. Soc. Exper. Biol. & Med., 35:53, 1936. Rev. Scient. Instruments, 23:235, 1952; 24: 178, 1953.
27. Lambossy, P.: Helvet. Physiol. Acta, 8:209, 1950; 9:145, 1951.
28. Lamport, H.: Howell's Textbook of Physiology, p. 630, 1946.
29. Landes, G.: Ztschr. f. Biol., 101:1, 1942; 101:418, 1943. Arch. f. Kreislaufforsch., 15:1, 1949.
30. Laszt, L., and Müller, A.: Helvet. Physiol. Acta, 9:55, 1951; 9:442, 1951; 10:1, 1952; 10:259, 1952.
31. Moens, A. J.: Die Pulskurve. Leiden: 1878.
32. Müller, A.: Abhandl. z. Mechanik d. Flüssigkeiten, I. and II. I. Freiburg and Leipzig, 1936. II. Arch. f. Kreislaufforsch, 4: Heft 1-6, 1939.
33. Müller, A.: Helvet. Physiol, Acta, 6:C32-C33, 1948.
34. Pappenheimer, J. R., and Maes, J. P.: Am. J. Physiol., 137:187, 1942.
35. Peterson, L. H.: Federat. Proceed., 11:750, 1952.

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MEASUREMENT OF PRESSURES BY CARDIAC CATHETERS IN MAN

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THE RECORDING of pressures from the chambers and vessels of the right side of heart in man must usually be done by means of a cardiac catheter connected to a manometer. The dynamic response of such a system can quite easily be made adequate for sufficiently accurate reproduction for most purposes of the pressure pulses in question.[1,5,8] However, serious pressure artifacts, caused by impacts on and motion of the catheter imparted by the heart beat, practically preclude high fidelity pressure pulse recording by this method if conventional catheter-manometer systems are used.[1,4] This study was carried out to obtain data concerning the optimal dynamic response characteristics of cardiac catheter-manometer systems for recording of pressure pulses by venous catheterization in man.

Methods

Pressure pulses, and other physiologic variables were recorded during routine diagnostic cardiac-catheterization procedures,[7] by a photo-oscillographic assembly described elsewhere.[6] In the majority of the experiments, pressures were recorded via the catheter by means of a specially adapted strain gauge manometer.[7] The over-all dynamic response of this catheter-manometer system could be varied instantaneously by a multipole switch, connected so as to allow recording of the pressure pulses interchangeably by three different galvanometers with natural frequencies of 5, 12, and 25 cycles per second, respectively. In part of the experiments the catheter pressures were recorded interchangeably, by turning a stopcock either by a high-frequency capacitance manometer,* or the strain gauge manometer. The resonant frequency and damping characteristics of the catheter-manometer assemblies used were determined at the conclusion of each catheterization procedure by recording the responses of the systems to square wave and variable frequency sine wave pressure variations generated by an electromagnetic hydraulic pressure oscillator described elsewhere.[3,6] Care was taken that the conditions of fluid filling and hydraulic connections between catheter and manometer were identical to the conditions pertaining during the experiment.

The susceptibility of the catheter-manometer systems to pressure artifacts, caused by motion of the catheter, was also studied at the termination of each procedure soon after withdrawal of the catheter tip from the vein. The apparatus used consists of a motor-driven cam, which imparts either a sine wave or a square wave motion to a shaft, to which the tip or shaft of the catheter can be clamped. The sine wave motions were at a frequency of 2 per second and peak-to-peak amplitude of 2 cm. The frequency of the square wave impacts was 1 per second. The pressure artifacts, generated when the catheter was subjected to these motions along the axis of the tip and perpendicular to the tip or the midshaft of the catheter, were studied.

Results

Studies of this type have been carried out during thirty catheterization procedures using strain gauge manometer systems only, and in seventeen procedures using the strain gauge and capacitance manometers interchangeably. The catheters used were of the Cournand type, either 100 or 120 cm. in length, and varying in diameter from size 4 to 7 French.

In general, the dynamic response characteristics of catheters of a given size and length, connected to the strain gauge manometer, were quite closely reproducible from procedure to procedure. This reproducibility was not obtained with the higher frequency capacitance manometer system, in spite of extreme care in attempting to insure minimal compliance of hydraulic connections, and

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*The Lilly Manometer manufactured by the Technitrol Engineering Company, 2751 North Fourth Street, Philadelphia 33, Pennsylvania.

avoidance of entrapment of minute air bubbles.

Catheter-manometer systems with low dynamic response characteristics uniformly produced pressure pulse recordings with less evident distortion by motion artifact than did the higher frequency systems. This difference was especially evident for pressure recordings taken when the catheter was threaded through the beating heart, as is the case for pulmonary-artery wedge, pulmonary arteries and right ventricle pressures.

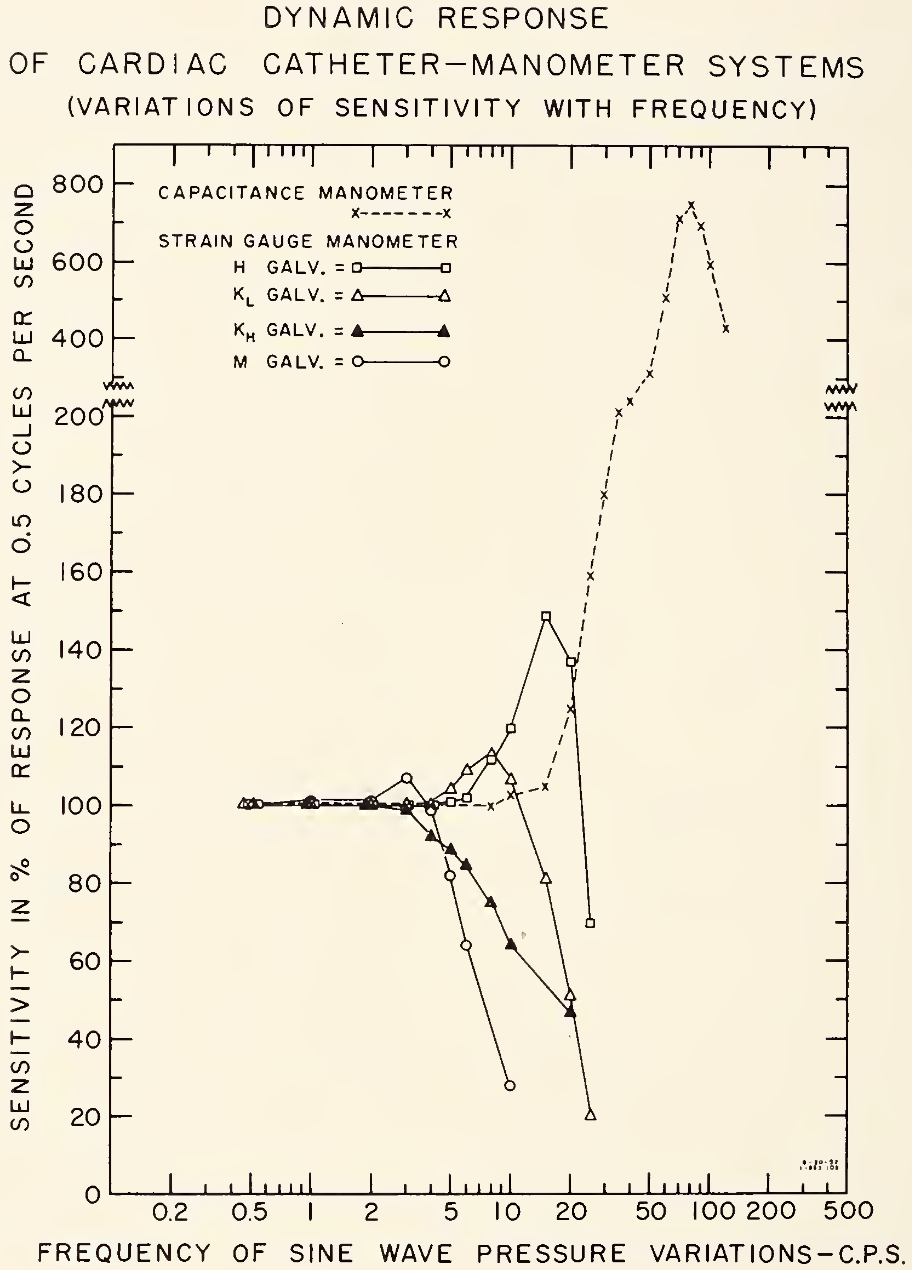

Fig. 1. Variations of sensitivity of cardiac catheter-manometer systems with the frequency of sine wave pressure variations determined immediately after withdrawal of the No. 5 French catheter, 100 cm. long, from the vein (see text for details). The catheter could be connected interchangeably to either a capacitance or a strain gauge manometer. The dynamic response of the strain gauge could be altered interchangeably by connecting it to any one of four different recording galvanometer systems designated as H, K$_L$, K$_H$ and M.

There was an excellent correlation between the degree of distortion of recorded pressure pulses by motion artifacts, and the amount of pressure artifact caused by sine wave, and impact catheter motions produced by the motor-driven cam apparatus. Figures 1, 2 and 3 show typical results illustrating these findings.

These studies were carried out during and after a cardiac catheterization of a six-year-old girl found to have pulmonic stenosis. Pressures were recorded via a 100 cm. No. 5 cardiac catheter from the pulmonary-artery wedge, pulmonary artery, and right ventricle by a strain gauge manometer coupled interchangeably to each of four different galvanometer systems, and by a high-frequency capacitance manometer.

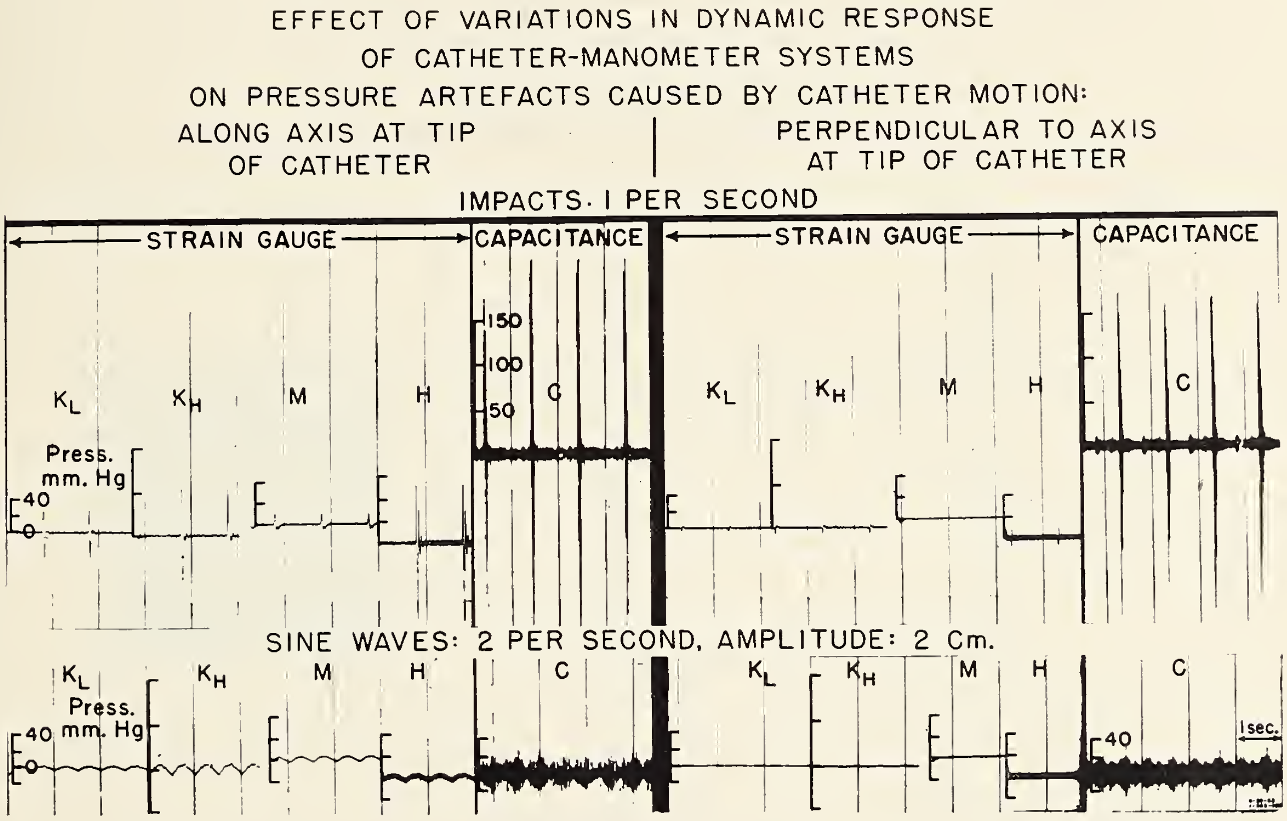

Fig. 2. Effect of variations in dynamic response of cardiac catheter-manometer systems on the pressure artifacts, caused by catheter motion along the axis of the tip of the catheter (left panels), and perpendicular to the axis at the tip of the catheter (right panels). The catheter motions were produced by a motor-driven cam shaft. The dynamic responses of the strain gauge and capacitance manometer systems are shown in Figure 1. Note that the lower frequency strain gauge systems (K_H and M) record less artifact than the 20-cycle system (H), and that the high-frequency undamped capacitance manometer system records continuous high-frequency oscillations probably associated with induced mechanical vibrations in the catheter system. Note also that motions along the axis of the tip of the catheter produce greater pressure oscillations than do identical motions perpendicular to the axis of the catheter tip.

The natural frequency of the capacitance manometer system was 90 cycles per second, and that of the strain gauge-catheter system about 20 cycles per second. By use of lower frequency galvanometers the response could be reduced interchangeably to about 10 or to less than 5 cycles per second (Fig. 1). The susceptibility of these catheter-manometer systems to pressure artifacts caused by motion of the catheter is shown in Figure 2. Since the major portion of the pressure artifacts are caused by the acceleration and deceleration of the fluid column within the catheter, induced by motion of the catheter, it is to be expected that motions along the axis of the catheter generate greater reactive pressures than do motions perpendicular to the axis. Impacts along the axis of the catheter tip caused spikes of pressure varying from 5 mm. of mercury for the overdamped 5-cycle strain gauge system to more than 200 mm. of mercury for the underdamped 90-cycle capacitance manometer system. Sine wave motions at 2 per second of 2-cm. amplitude caused smooth 10 mm. of mercury peak-to-peak pressure variations in the 5-cycle system and continuous noise, 10 to 20 mm. of mercury in amplitude, in the 90-cycle capacitance system. Higher frequency catheter-manometer systems were uniformly more susceptible to motion artifacts than the low-frequency systems.

Pressures recorded by these identical catheter-manometer systems from the pulmonary-artery wedge, pulmonary artery and right ventricle are shown in Figure 3. The most artifact-free tracings were obtained by the 5-cycle system. When the catheter was threaded through the heart into the wedge position, the pressure pulses recorded by the 90-cycle capacitance manometer system were obscured by artifacts.

Tracings obtained by the capacitance manometer can be improved by damping so that the response is limited to 5, 10 or 20 cycles per second. The capacitance and strain gauge systems are then about equally sensitive to motion artifacts. Pressure pulse tracings obtained from the pulmonary artery and right ventricle show the least artifact for the 5-cycle system, most for the 20 and intermediate, and about equal artifact for the 10-cycle capacitance and strain gauge systems.

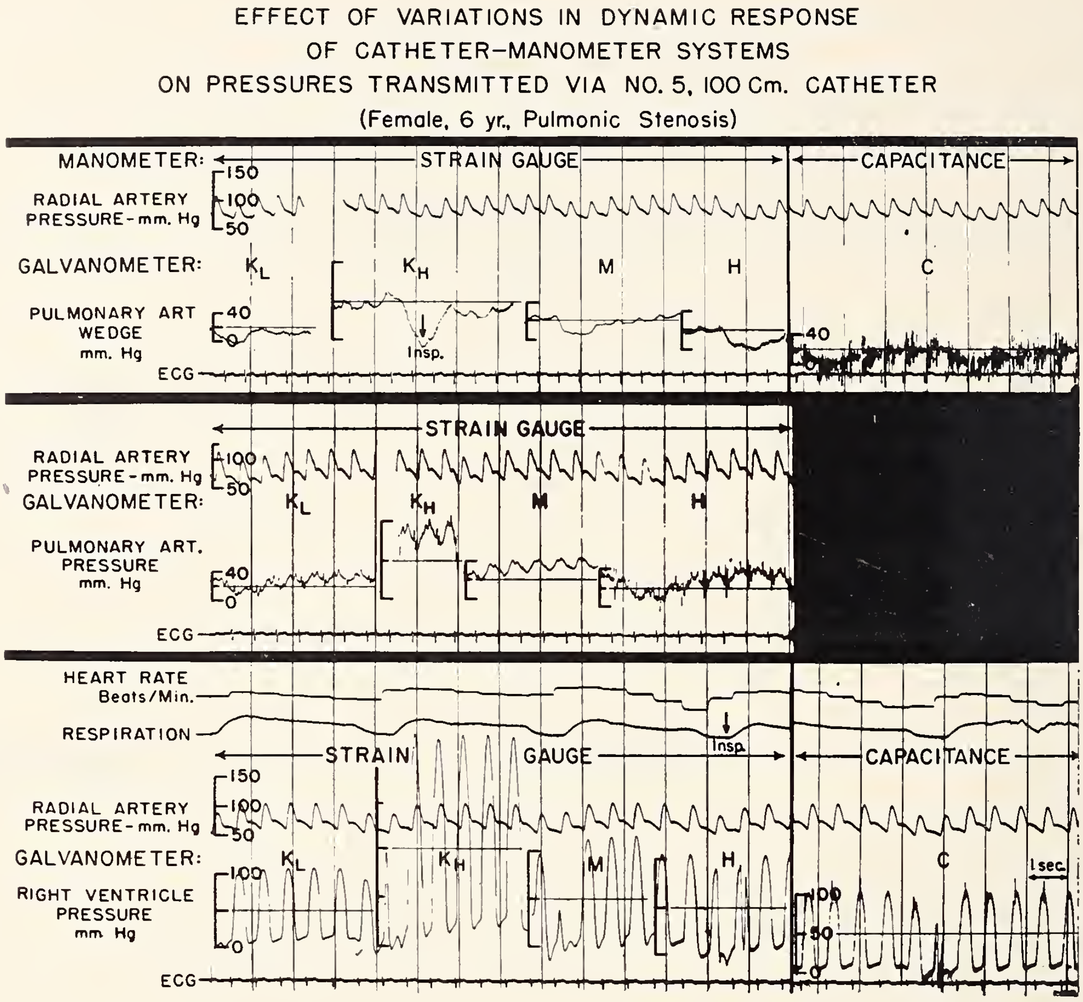

Fig. 3. Effects of variations in dynamic response of catheter-manometer systems on pressure pulses recorded from the pulmonary-artery wedge, pulmonary artery and right ventricle of a six-year-old girl with valvular pulmonic stenosis. The dynamic responses of the strain gauge and capacitance manometer systems are shown in Figure 1. Note that recordings from the lower frequency strain gauge systems (K_H and M), show less evident distortion by artifact than the 20-cycle system (H), and that when the catheter is threaded through the beating heart the pressure pulses in the capacitance manometer recording are almost completely obscured by high-frequency artifact. Note also the correlation between the susceptibility of the various systems to motion artifact shown in Figure 2, and the evident artifact in the pressure pulses recorded by these same manometer systems.

It has been suggested that motion artifacts would be reduced if very small catheters were used or, better still, if pressures were recorded by a small catheter threaded through the standard size cardiac catheter. In our laboratory this has been found not to be the case. The small catheter used was of the Peterson type (145 cm. long, 0.3 mm. I.D., 0.6 mm. O.D.). For pressure recording from the heart it was threaded through a conventional 120 cm. Cournand No. 6 cardiac catheter using a special adapter assembly.

The dynamic response of these systems was determined. The natural frequency of the 0.3 mm. catheter-capacitance system was about 30 cycles per second. For pressure pulse recording the system was damped down to 10 cycles per second, similarly to the standard catheter-strain gauge system. The strain gauge system, recording from the cardiac catheter containing the small catheter, was the most overdamped of the three systems, having a uniform response out to only 2 cycles per second.

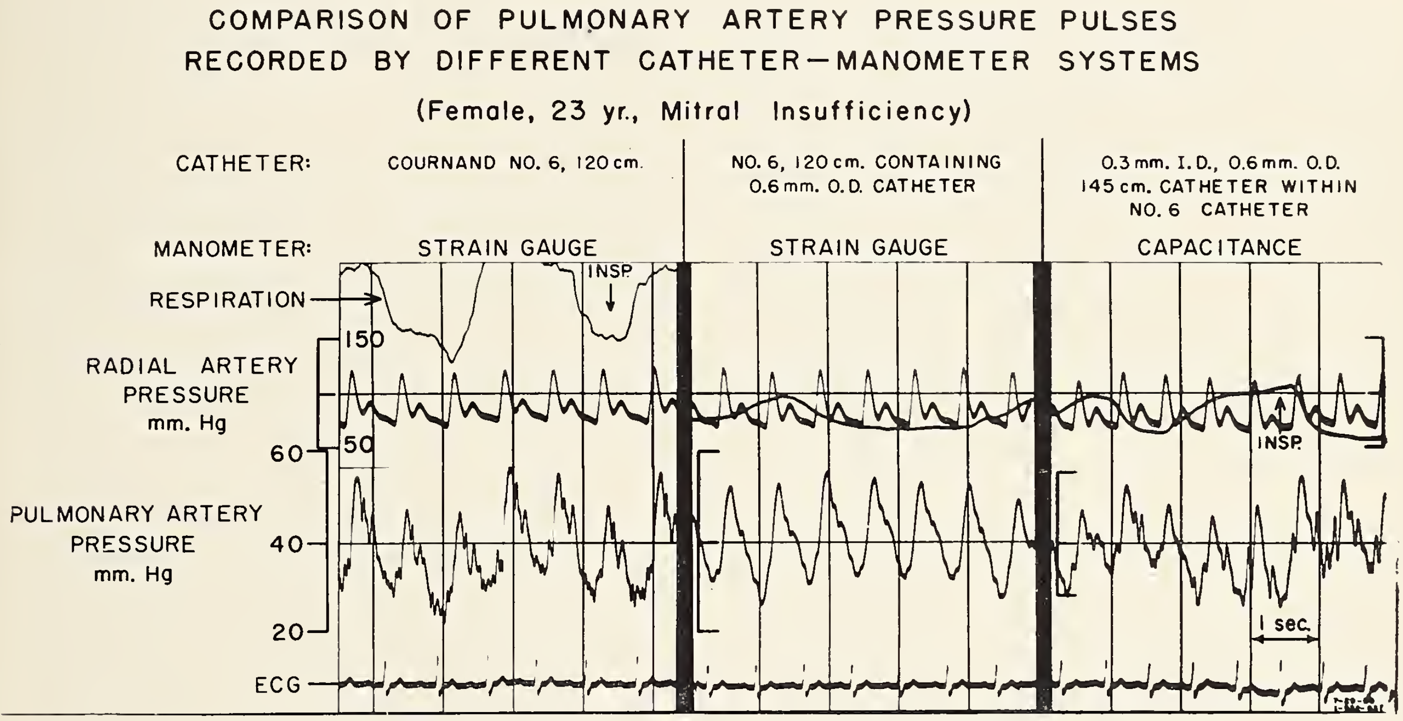

Fig. 4. Comparison of pulmonary-artery pressure pulses recorded by different catheter-manometer systems from a twenty-three-year-old woman with mitral insufficiency. For details concerning the make-up and the dynamic responses and susceptibility to motion artifacts of these three manometer systems, see text. The small plastic catheter was protected from impacts due to the heart action by the No. 6 cardiac catheter through which it was threaded into the pulmonary artery. The pressure pulses recorded by this system (right panel) are nevertheless, still badly distorted by artifact, although somewhat less so than the recordings from the conventional catheter-strain gauge system shown on the left. The frequency response of the overdamped system (strain gauge connected to the No. 6 cardiac catheter containing the small plastic catheter) is probably too low for accurate recording of intracardiac pressure pulses, though the tracings obtained (middle panel) show the least evident distortion.

The susceptibility of these systems to motion artifacts was determined also. The overdamped strain gauge system exhibits the smoothest tracing while the small catheter-capacitance manometer system is intermediate between that and the standard catheter-strain gauge system. Actual pressure pulse tracings from the pulmonary artery of the same patient show similar results (Fig. 4), evident motion artifacts on the small catheter-capacitance tracing being intermediate between those seen on the tracings from the two strain gauge-catheter systems.

Comment

These and similar studies have led us to conclude that it is highly improbable that high-fidelity pressure pulse tracings can be recorded via conventional catheters threaded into the beating heart. The degree of evident distortion can be reduced by using relatively low-frequency recording systems with a sharp cutoff in sensitivity to higher frequencies which thus selectively discriminate against the relatively higher frequency motion artifacts. This requirement is, however, mutually antagonistic to the recording of cardiac pressure pulses, since elimination of evident motion artifact requires such a low frequency system that adequate reproduction of the pressure pulses in question may not be possible. Indeed, motions of the catheter at the frequency of the heart beat, induce pressure artifacts at the frequency of the cardiac cycle which may be relatively smooth in contour and fused with the actual pressure pulse in such a way as to be unrecognizable as artifact.

The only solution to this problem known to us at present, is the use of a miniature manometer attached to the intracardiac tip of the catheter. Such a manometer has been made by Gauer and Geinapp after Wetterer's design.[2] Since its moving element has a mass of only 15 mg., reactive forces to acceleration and deceleration of the catheter tip are very small and hence this catheter-manometer system is practically free of motion artifacts when the catheter is moved outside the body.[1] Pressure pulses recorded from the heart and great vessels are similarly practically devoid of artifact.[1]

Summary and Conclusions

An electromagnetic hydraulic pressure oscillator has been used to study the dynamic response characteristics, and a motor-driven cam apparatus to study the pressure fluctuations caused by reproducible sine wave and square wave motions of cardiac catheters connected to strain gauge or capacitance manometer systems. These studies were carried out immediately after use of these identical systems for recording of intracardiac pressure pulses during forty-seven diagnostic cardiac-catheterization procedures in patients with various types of cardiovascular abnormalities.

1. Sine wave motions, along the axis of the tip of the catheter at a frequency of 2 per second and a peak-to-peak amplitude of 2 cm., produce sine wave variations of pressure at this frequency and with a peak-to-peak amplitude of about 10 mm. of mercury. This motion of the catheter tip resembles that usually seen to some degree at cardiac catheterization, especially when the catheter tip is in the pulmonary artery. Sine wave motion perpendicular to the axis of the tip of the catheter did not produce pressure fluctuations of practical significance.

2. Square wave motions (impacts) produce high-frequency pressure variations of greatest magnitude when the impact is directed along the axis of the tip of the catheter. The amplitude of the recorded pressure variations induced by square wave motion of the catheter (impacts) varied inversely with the frequency response of the catheter-manometer systems used. Impacts of the catheter produced much less pressure fluctuation when in a direction perpendicular to the axis of the catheter than when directed along the axis of the catheter.

3. A close correlation was demonstrated between the susceptibility of cardiac catheter-manometer systems, to pressure artifacts induced by motion of the catheter outside of the body, and the degree of distortion by artifact of pressure pulses recorded by the same catheter system from the beating heart.

4. Catheter-manometer systems, with a uniform dynamic response out to 5 to 10 cycles per second with a sharp cutoff in sensitivity to higher frequencies, were least susceptible to the higher frequency pressure artifacts induced by catheter motion, and likewise produced pressure pulse recordings with the least evident distortion by artifacts.

5. Pressure pulses, recorded by conventional catheters threaded through the beating heart, should be regarded with a high index of suspicion, since the lower frequency artifact induced by motion of the catheter synchronous with the heart beat, may be of such character and be fused with each pressure pulse in such a manner as to be unrecognizable as artifact.

6. Recording of pressures by a small (0.3 mm. I.D.) catheter threaded through a conventional cardiac catheter reduces slightly but not to a sufficient degree the artifact present in pressure pulse recordings from within the heart.

References

1. Ellis, E. J.; Gauer, O. H., and Wood, E. H.: An intracardiac manometer: Its evaluation and application. Circulation, 3:390-398 (Mar.) 1951.

2. Gauer, O. H., and Gienapp, Erich: A miniature pressure-recording device. Science, n.s., 112:404-405 (Oct. 6) 1950.

3. Isaacson, James, and Jones, R. E.: An electromagnetic hydraulic transducer for studying response characteristics of manometer systems in square wave and sine wave pressure variations of variable frequency. (Abstr.) Am. J. Physiol., 163:722-723 (Dec.) 1950.

4. Lambert, E. H., and Jones, R. E.: The characteristics of a resistance-wire manometer for measuring blood pressure in cardiac catheterization studies. Proc. Staff Meet., Mayo Clinic., 23:487-493 (Oct. 27) 1948.

5. Wood, E. H.: Study of minimal dynamic response characteristics of manometer systems required for adequate recording of peripheral arterial pressure pulses in man. (Abstr.) Am. J. Physiol., 163:762 (Dec.) 1950.

6. Wood, E. H.: Special instrumentation problems encountered in physiological research concerning the heart and circulation in man. Science., n.s., 112:707-715 (Dec. 15) 1950.

7. Wood, E. H.: Special technics of value in the cardiac catheterization laboratory. Proc. Staff Meet., Mayo Clin., 28:58-64 (Feb. 11) 1953.

8. Wood, E. H.; Leusen, I. R., and Warner, H. R.: Comparison of aortic pressure pulses recorded by strain-gauge and capacitance manometer-catheter systems in man. (Abstr.) Am. J. Physiol., 171:780 (Dec.) 1952.

MATHEMATICAL CONSIDERATIONS OF INDICATOR DILUTION TECHNIQUES

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THE STUDY of circulatory mixing processes is an important practical approach to the investigation of the circulatory physiology of man in health and disease. Injection of substances into the blood stream and observing the time relations of concentration either at the injection site or at other sites is one of the physiological manipulations which can be sanctioned in human studies. With the advent of a growing multitude of tracer substances, there remains an increasing number of experiments of this type which can be done. Their interpretation will require an improved knowledge of the processes of circulatory transport and mixing.

The measurement of volumes of dilution of injected substances will not be emphasized in this discussion. Instead we will consider the immediate events following the rapid injection of a small portion of labeled material into a large vessel. The extensive pioneer work by Hamilton and his associates[5] on the concentration of dye in blood from the radial artery following injection in the cubital vein is familiar to all. Recent developments include the use of radioactive isotopes[1,8,12] and the development of photoelectric recording methods either with the use of the ear oximeter or with the recording cuvette through which blood from a needle in an artery or vein is allowed to flow.[3,10,11]

Although these technical improvements have been of great value, the interpretation of the data which they furnish requires the use of a few mathematical considerations in achieving a quantitative description of both the dispersing processes which occur as the dye flows through the circulatory labyrinth and the effects of recirculation. The present discussion should be considered as an introduction to a problem which has been explored in a preliminary way only, but steady progress is being made and the results seem to justify the effort, at least, so far. We will confine our attention for the moment to substances such as P^{32}-labeled erythrocytes or T-1824 which do not rapidly disappear from the circulation, since it has been shown recently that even rather rapidly disappearing substances yield results which can be expressed in terms of an equivalent nondisappearing substance.[13]

Circulatory mixing represents one of the powerful processes by which the body resists the action of external disturbances which might interfere with the preservation of a stable internal milieu. When a small quantity of injected substance moves through the circulation, we can imagine it to be resolved into a very large number of small elements, each requiring a different amount of time to move through the circulatory labyrinth. The variation in traversal times causes the substance to be randomized and progressively smeared or dispersed in time during the process of circulatory mixing. It is intriguing, perhaps, to realize that here the body architecture achieves design essentially by lack of it. Because of this apparent lack of design, as we usually think of it, the purely deterministic approach is not likely to be fruitful in describing the dispersion process.

One illustration of our limitations, which was particularly instructive, occurred when an attempt was made to investigate the simple case of dispersion of dye during simple laminar flow along a uniform cylindrical tube. The theory of this process is quite simple. Suppose that water is flowing through the tube at the rate of Q cc. per second and that I milligrams of dye are injected in such a way that the initial distribution in the tube is that of a uniform concentration in a thin layer whose plane is perpendicular to the axis of the tube. If the volume of the tube from the injection point to the outflow is V cc., then the concentration of dye (mg./cc.) in any small uniformly mixed sample taken at time t from the outflow will be

$$C(t)\,dt = 0 \text{ for } t < V/2Q \quad \text{and}$$

$$C(t)\,dt = I\,V\,dt/2Q^2\,t^2 \text{ for } t > V/2Q$$

(Equation 1)

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Figure 1 shows a comparison of the results of one attempted experiment with the theoretical prediction. The results do not agree in anything but a qualitative sense. Further investigation showed that it was extremely difficult to establish the required initial distribution. Not only did small differences in specific gravity of the injected dye compared to the water in the tube cause settling of the dye, but also the concentration was not uniform because of the slow mixing of the dye with the water. Since the arrival time downstream of a small dye element depends critically on its initial radius from the center of the tube, the time relations downstream were greatly affected by the nonuniformity of the initial dye distribution, and due to our inability to control the dye, the results were erratic from one experiment to the next. Although this difficulty may be controlled by special techniques, the difficulty in control of the dye applies in all likelihood to a great many typical physiological situations.

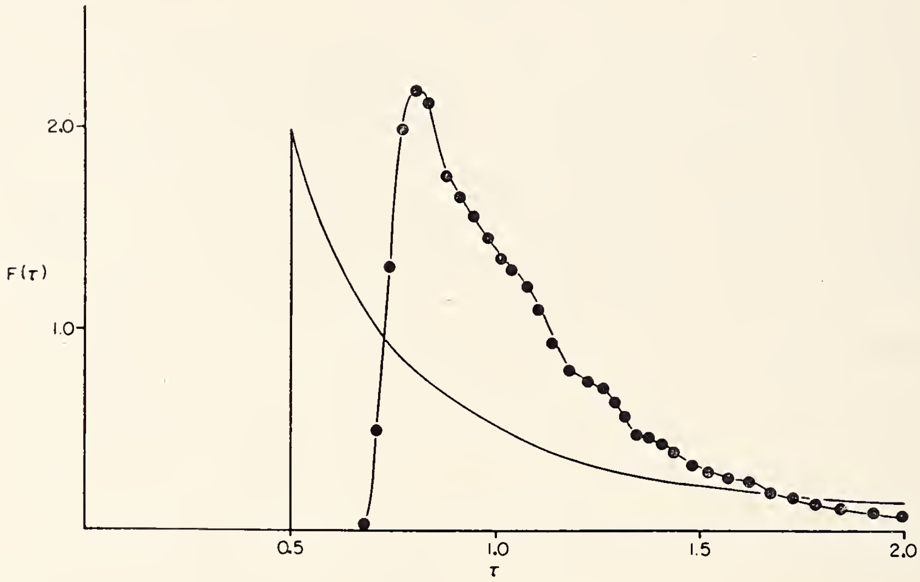

Fig. 1. Theoretical and attempted experimental results on the outflow of a small uniformly injected element of dye from a uniform cylindrical tube in which the transporting fluid moves without turbulence. The experimental results (dotted points) fail to confirm the theory because of uniform inability to control initial distribution of injected dye and to prevent gravitational settling.

Equation 1 can be greatly simplified if we choose a different set of variables. In the first place the concentration in a given sample is not the basic quantity but rather the fraction of the total injected dye contained in it. Secondly, the flow rate, the time, and the bed volume are not individually important variables. Neglecting diffusion and settling out effects, in the ideal situation we can collect the fractions at any rate, or at variable rates, or the experiment can be interrupted and continued at a later time. The factor controlling the situation at the outflow is the volume of fluid collected rather than the time, i.e., the product Qt. If the length of the tube be doubled, the first dye will not appear until twice the volume of water has passed through the system. Thus the basic variable is the fraction of bed volume displaced, i.e., $Qt/V = \tau$. Making these substitutions in equation 1, we obtain the universal relation that the fraction of dye in a given sample for $\tau > 0.5$ is

$$F(\tau)d\tau = C(t)\ Qdt/I = d\tau/2\tau^2$$

(Equation 2)

as before for $\tau < 0.5\ F(\tau) = 0$. This is a universal expression which applies to all uniform bore circular cylindrical tubes irrespective of their dimensions provided the flow is streamline.

It seems scarcely likely that any vestige of organized flow of the type just discussed will remain in the circulation shortly after injection. Even if the settling out effect, and the nonuniformity of the injection process could be controlled, many factors will immediately act to randomize the indicator. A few bifurcations will be sufficient to destroy any regularity which might exist. Turbulence will occur at least in the heart. Most important, however, is the separation of the injected material in the capillaries and the later recombination in a completely undetermined manner. For this reason, we must reject the purely deterministic point of view and turn to the methods of probability in our investigation of the most likely form of F(τ). We thus consider the nonrandom components in a nearly random situation.

The so-called "stochastic" approach based on probability considerations is to consider the type of F(τ) which would occur if the dye elements were being carried through a series of uniformly mixed compartments. There seems to be little anatomical reason to adopt this point of view. Perhaps the outflow from the heart might approximate the situation of two chambers, but nevertheless the advantage of this so-called "washout" model is a theoretical one. From the statistical point of view the dye particles progress through the chain of compartments in a series of jumps. In each compartment a particle makes many trials before it can escape because of the uniform mixing in the compartment. When it finally escapes, this is a rare event. The probability is proportional to the ratio of the flow rate through the compartment divided by its volume. The process of escape from any compartment is not controlled in any way by what happened in previous compartments. In general, when it leaves the n'th compartment, there have been n successes. This problem is a special case of a so-called "Markov chain."[2] It is completely analogous to the problem of radioactive decay of a nonbranching chain of nuclides in equilibrium and its solution is given in standard texts on radioactivity. If all of the dye is initially uniformly mixed in compartment one, then for n compartments

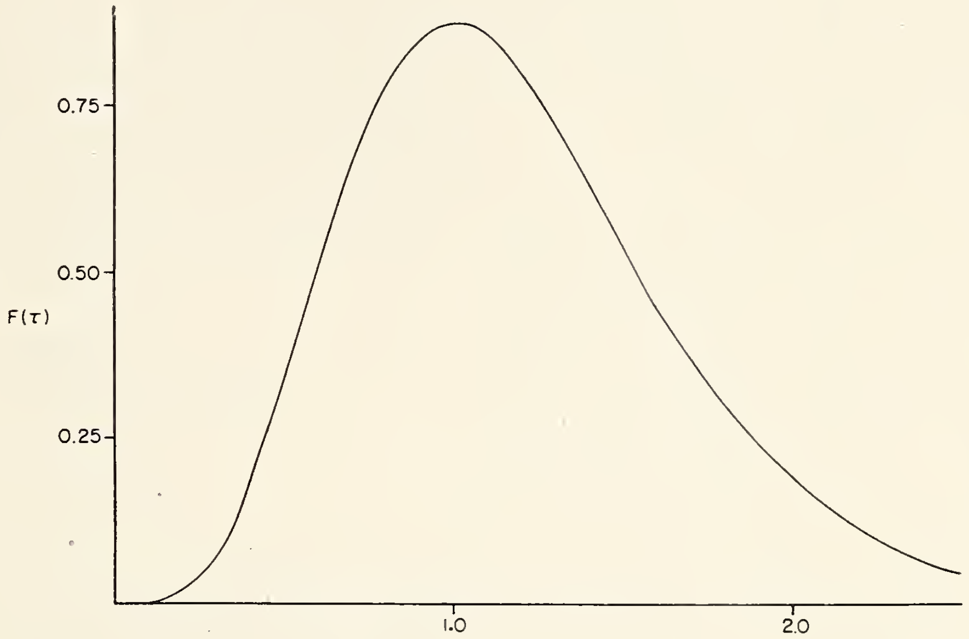

Fig. 2. Theoretical outflow of indicator for a serial system of 5 equal chambers in which uniform mixing is maintained. The abscissa is the fraction of the system volume displaced as fluid passes through the system. The ordinate is the fraction of injected indicator per unit τ interval appearing at a given τ instant.

$$F(\tau)d\tau = \sum_{i=1}^{n} f_n \frac{f_i^{\,n-2}}{f_i \prod\limits_{\substack{j=1 \\ j \neq i}}^{n} (f_i - f_j)} e^{-\tau/f_i} d\tau$$

(Equation 3)

where f_i is the fraction of the system volume in compartment i.* If the compartments are all alike the solution becomes

$$F(\tau)d\tau = \frac{n(n\tau)^{(n-1)}}{(n-1)!} e^{-\tau n} d\tau$$

(Equation 4)

Since for n compartments we are dealing with the probability of n-1 successes in a series of independent equally likely trials of a rare event (i.e., the successive escapes from all but the last), the fact that we have obtained the Poisson law of probability is not surprising. In general, we should consider the Function $F(\tau)$ as a typical statistical frequency distribution function which, when multiplied by $d\tau$, gives the fraction of the total dye elements appearing in a given small class interval $d\tau$ of the independent variable τ.

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*The π in the denominator signifies "the product of a set of factors each corresponding to a given integral value of j ranging from 1 to n but omitting the factor in which j and i are alike."

Figure 2 shows a typical plot of $F(\tau)$ for a system of five equal compartments. It is evident that the curve has some resemblance to typical curves of the outflow of dye from the pulmonary circuit in man and in the dog. This led Newmar and his associates to postulate that often dispersion curves were adequately described by the "washout" theory.[10] It seems, however, that this agreement is the result of a statistical accident. One can scarcely believe that the movement of dye through a capillary bed and extensive system of branching vessels is the result of a small number of successes in a series of trials of a rare event. Instead of a few large jumps and long pauses in the progressive movement of the dye elements, it is eminently probable that a closer representation is given by a theory that considers a very large number of rapidly occurring small jumps. Under these conditions, the probability theory predicts that the particles will execute a "random walk."[2] Random walk problems have received considerable attention in the literature, but most mathematicians consider the situation of a fixed probability distribution for the jumps. This classical version of the random walk will serve as a first approximation to the physiological situation. We will consider it first and discuss the corrections to the theory separately.

A good experimental illustration can be obtained by filling a long uniform tube with 3 mm. glass beads, allowing water to flow through it and injecting a small amount of dye in one end. At the beginning the dye forms a small compact slug at one end of the tube. As the fluid moves through the beads some dye elements begin to move ahead and others to lag behind. The distribution of dye at various distances along the tube appears to be Gaussian, i.e., that of the normal curve of error. As the center of gravity of the distribution proceeds along the tube with essentially the same velocity as the average fluid velocity the dye spreads out and the standard deviation incerases in proportion to the amount of fluid which has passed through the tube. At the end of the tube the beads suddenly come to an end and so the dye can only cross the boundary once, being immediately swept away. A dye element is thus said to execute a random walk terminated by a first traversal. Because of the effect of the end of the bead column and because the standard deviation increases with the volume of fluid which has passed, the distribution of dye among the various fractions collected will not be Gaussian. The relation which can be derived from mathematical considerations[2] is

$$F(\tau)d\tau = \frac{e^{-\frac{(1-\tau)^2}{\kappa^2\tau}}}{\kappa\sqrt{\pi}\,\tau^{3/2}}\,d\tau$$

(Equation 5)

The randomizing constant $\varkappa$ represents $\sqrt{2}$ times the standard deviation relative to the mean of the dye distribution which would be obtained if, after the displacement of one bed volume, the experiment were stopped and if the various fractions could be removed from the tube and their label content determined without disturbing the distribution. $\varkappa$ is expressed in units of τ. In deriving this relation it is assumed that the center of mass of the distribution moves with the same velocity as the liquid, i.e., the probability distribution for the movement of the dye elements relative to the fluid is the same forward as backward.

The classical random walk problem is of general interest. In the theory of games of chance it is often called the Gambler's Ruin problem, since it describes the progress of a game in which a player contends with an infinitely wealthy opponent who has the odds in his favor. It has been used in physics to describe the Brownian movement of a particle. The background mathematics is found in a number of standard texts.[2] The problem is a special case of a Markov chain but is often developed as a limiting case of the random movement of particles between isolated points on a line as the points become infinitely numerous and close together. Particles are permitted to jump to a distant point or to neighboring points. It is required that no jump is in any way affected by previous jumps. Jumps of different amounts are not required to be equally probable but whatever the pattern of probability of the jumps in the classical version of the problem it must be the same for all. The jumps are random in that their magnitudes cannot be predicted but merely the relative likelihood of a given sized jump. Jumps forward can be more frequent or less frequent than jumps backward. The degree of asymmetry will govern the rate at which the centroid of the population will move. The rate of increase of the standard deviation will be governed by the root mean square of the magnitudes of the jumps and the rate at which they occur. This will be determined by the magnitude of the proportionality constant in the denominator of the exponent. This will be the randomizing constant $\varkappa$ in equation 5. In a fine grained bed $\varkappa$ would be larger than in a coarse grained one, and ideally determining $\varkappa$ would yield valuable information.

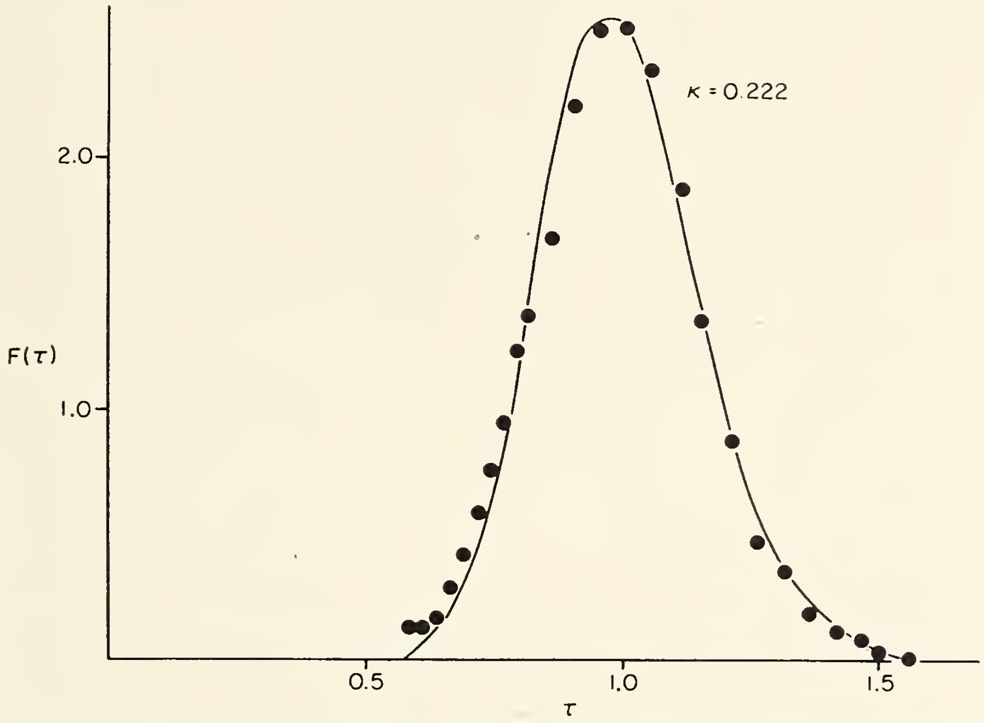

Fig. 3. Comparison of experimental results for outflow from a long cylindrical bed of uniform glass beads (●) with a theoretical one dimensional random walk curve (—). The water volume in the bed was 193 cc. See Figure 2 for ordinates and abscissas.

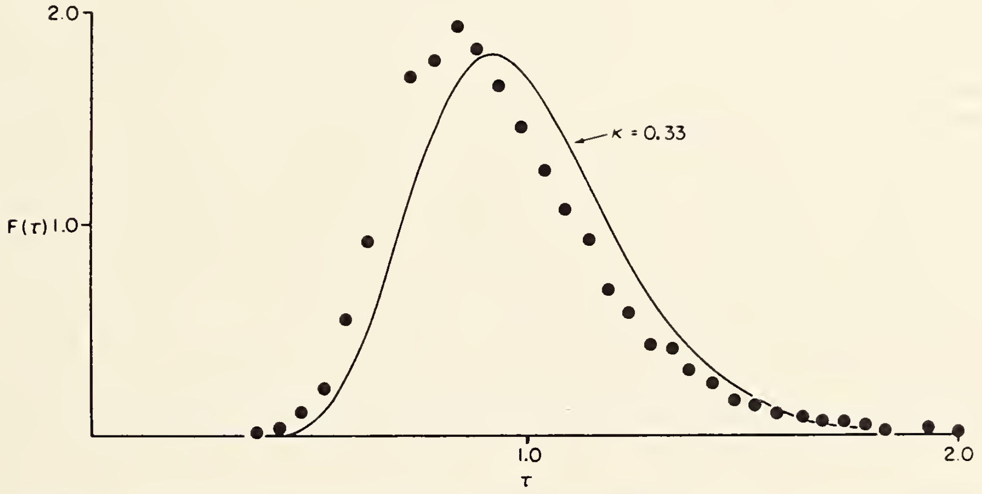

Fig. 4. Similar comparison (see previous figures) for a spherical flask containing 3 mm. beads. The water volume was 193 cc.

Comparison of theory and experiment (Fig. 3) indicates satisfactory agreement in the case of a long thin tube filled with glass beads, but this is quite unrealistic as a description of the flow through a physiological labyrinth such as the pulmonary circuit. In the first place, the shape of the bed plays a role. The outflow of dye from a spherical vessel is shown in Figure 4. The water entered a spherical bulb flowed through the same size beads as in the tube experiment and was collected immediately at the outflow. The result systematically deviates from the random walk hypothesis, but it serves as a first approximation even though in this case the dye elements are not confined into a linear path for their excursions. The value of $\varkappa$ is significantly greater than for the linear tube case although the same size beads were used. Some improvements of fit could probably be achieved by introducing an effective bed volume and starting time, adopting the variable τ' which would be the fraction of the *effective* bed volume which has flowed through the system.

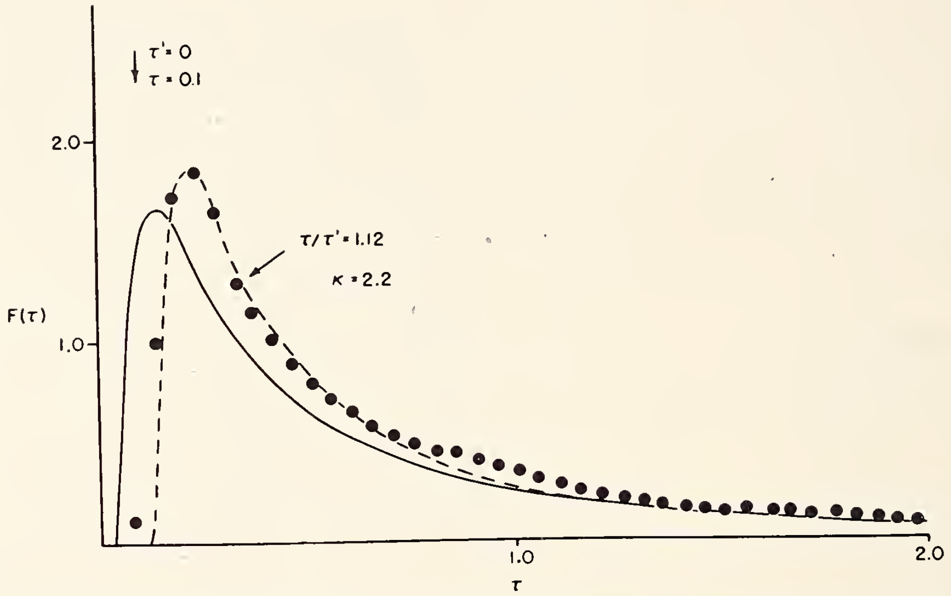

Fig. 5. Spherical flask (Fig. 4) with exit pipe projecting into the center.

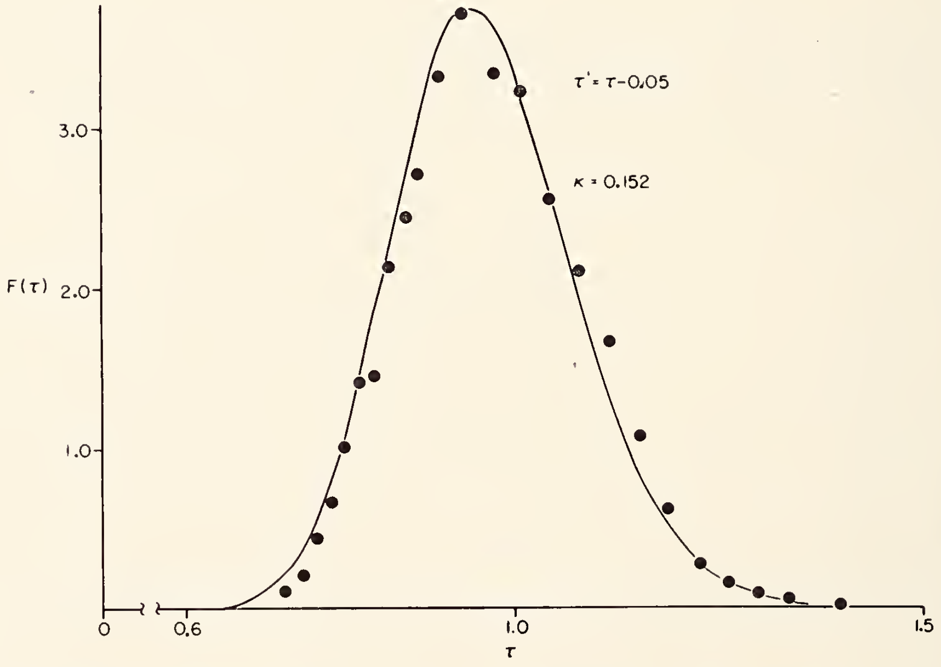

Fig. 6. Same as Figure 3, but dye injected in the rubber supply tube 71 cc. upstream.

When a deliberate attempt was made to produce a severe distortion of flow from that of a straight tube the shape of the curve could be strongly influenced. Instead of allowing the fluid to emerge directly from the bulb, a tube was inserted so that its orifice was at the bulb center. Under these conditions (Fig. 5), there was a large increase in the value of the randomizing constant. Furthermore, to fit the theoretical curve, the volume of the water in the bulb could no longer be used but instead an effective volume 12 per cent greater had to be introduced, together with an effective starting time at $\tau = 0.1$.

The preceding experiments all involved a uniform randomizing structure in the bed. Dye was injected directly at the inflow, traversed a collection of uniform beads, and was collected with as short an outflow tube as possible. This, of course, ignores the fact that in a physiological system the volume of the vessels leading into the system is appreciable. Often, also the material must be collected through a long catheter, whose dispersing effect on the dye must be considered. Since the sum of the randomizing effects of a series of different dispersing systems should be independent of the order in which they occur, a long inflow or a long outflow tube should produce similar effects. To investigate the effect of a long entrance tube, the cylindrical container containing the 3 mm. beads was equipped with a 3-meter rubber tube of approximately 5 mm. 1.D. Dye was injected into the upstream end and the outflow curve determined (Fig. 6). There is also a small 5 per cent correction for effective injection time. The curve is a fairly good fit to a random walk relation using a value of $\varkappa$ derived on the assumption that the equivalent $\varkappa$ for the tube is negligibly small. Since the flow through the tube is not well approximated by a randow-walk relation, a good deal of further research must be done before such an effect can be entirely understood.

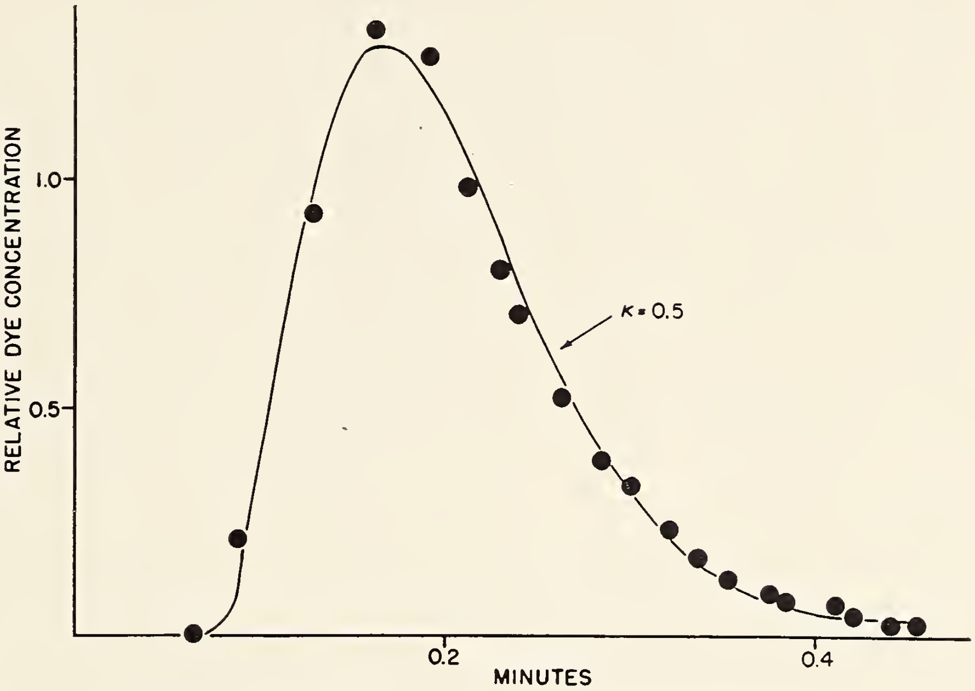

Fig. 7. Data of Hamilton *et al,* for perfused isolated heart-lung preparation in a dog.

It was recently suggested by L. J. Savage and the author[14] that the one-dimensional random walk might serve as a first approximation to the problem of capillary outflow. Applications of the theory to physiological data are limited as yet because most of the curves have been taken from beds of unknown volume, so that at least two constants can be invoked in fitting the data. This gives more latitude than the theorist deserves. Figure 7 shows a comparison between the theory and experiment for data obtained by Hamilton et al.[5] who perfused an isolated heart lung preparation in the dog. A rather satisfactory agreement was obtained using a value of $\varkappa = 0.5$. However, a rigorous test of fit is not possible because the readings were obtained by enlarging the small figure in their paper which produces some transcription error. A similar curve obtained more recently by Howard, Dow, and Hamilton[6] is closely similar if the appropriate scale adjustments are made. Again a fair agreement is obtained. A second experiment which they performed on the same preparation gave essentially the same result. Unfortunately, a comparison between the effective bed volume and the true volume of blood in the pulmonary circuit is lacking since the latter volume was not obtained. Analysis of the curves using the flow rates, which they actually determined, yields only the effective volume.

When we turn to curves obtained on the lungs alone a different situation is observed. Three injections in one preparation and two in another gave similar results if the probable difference in the cardiac output and volume of pulmonary blood in the two dogs and the variation in amount of injected dye are taken into account. A representative curve together with a fit to the random walk expression is shown in Figure 8. It is seen that to obtain a satisfactory agreement a considerably delayed effective injection time must be introduced. This is also true but to a somewhat lesser extent for a curve obtained by Little and Kelly who perfused an isolated canine liver preparation. The data were obtained in the course of other recently published studies on colloidal gold removal by the liver[7] (Fig. 9). Although the volume of blood in the liver was not known, the value of 240 cc. required to fit the data using their lowest flow rate seems high even for the approximately 16 Kg. dogs which they used. Because of these factors the one-dimensional random walk relation is probably only a first approximation to the true curves, and may often require the inclusion of effective

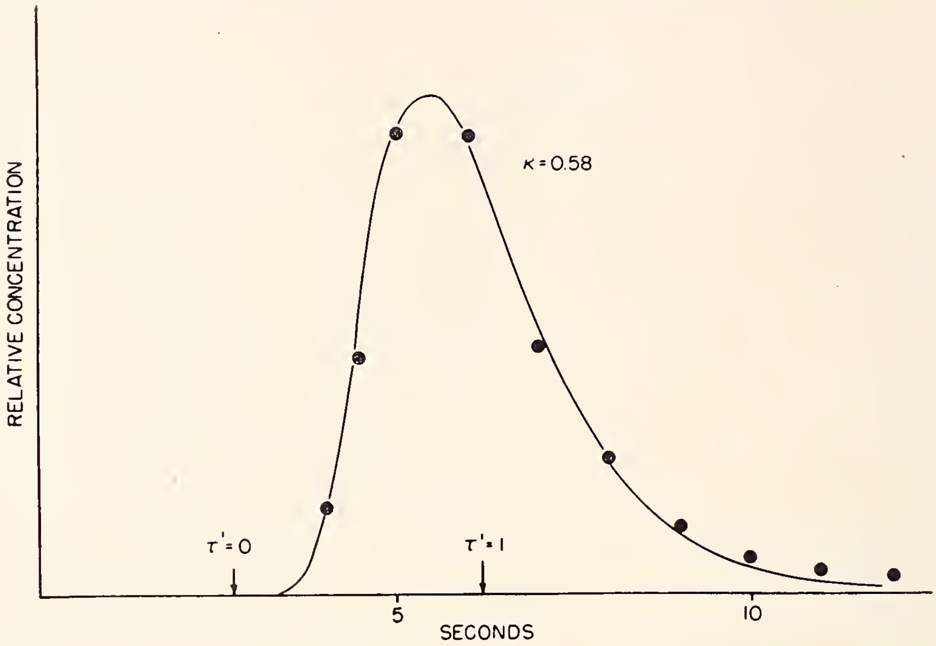

Fig. 8. Data of Howard, Dow, and Hamilton for perfused canine lung without the heart.

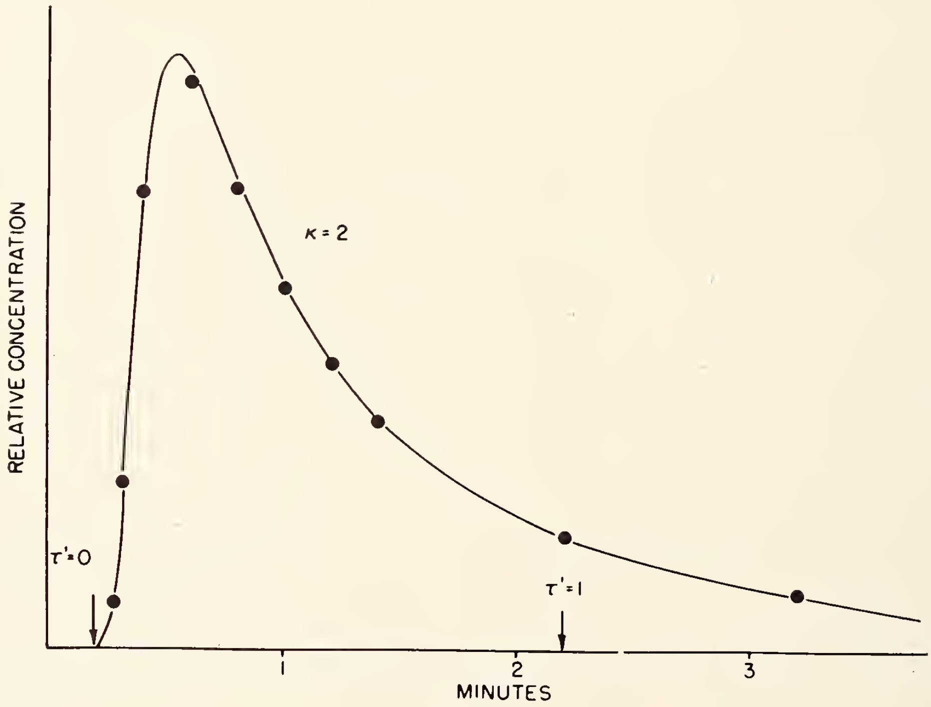

Fig. 9. Data of Little and Kelly for a perfused isolated canine liver.

volumes and injection times in its use. A more complete analysis would consider the movement of dye in terms of a more general Markov problem, but first it would be necessary to get a better idea of the variation in probability distribution of the jumps of the elements of dye as they traverse the circulation.

In addition to the form of the outflow of dye during a single perfusion experiment, we must consider the manner in which recirculation is taken into account before we can discuss the actual process of circulatory mixing. Time will not permit the discussion of recirculation problems in general, but the familiar situation where indicator is injected essentially into the right heart, and samples are taken from the aorta or a close tributary will be sufficiently illustrative. The first step in the analysis is to relate events upstream at some point 1 to events downstream at 2. We will suppose that a small amount of dye is injected suddenly at 1. Let the average concentration of dye be a(0) and the mean duration of the injection Δt. The product of these two factors will be the area under the injection curve. This area, when multiplied by the flow rate Q, will give the amount of indicator injected. We will consider only the case where the injection curve is a very high and very narrow spike. If the dye is injected at zero time, then in the limiting case where the height is infinite and the width infinitesimal (the case of the so-called "delta function"), the time distribution of dye downstream will be the distribution of traversal times, i.e., F(t) and so the concentration at any time t is given by the relation

$$a_2(t) = a(0)\,\Delta t\,F(t)$$

(Equation 6)

This will relate events at 2 to those at 1 provided that there is no dye between 1 and 2 at the moment of injection. If we consider a more general problem, suppose that the time relation upstream is not that of a single spike but a continuous variation given by the relation $a_1(t)$. Then downstream at 2 we will have

$$a_2(t) = \int_0^t a_1(t-T)\,F(T)\,dT \ + \ \text{effect of dye}$$

(Equation 7)

between 1 and 2 at or before $T = 0$. The variable of integration T represents the various time delays of the individual paths between 1 and 2. The second term will be zero if there is no dye in the system before the experiment is begun. The integral is known as a "convolution integral." It is of general utility when it is desired to consider at some point downstream what the effect will be of the smearing of the earlier time relations occurring at some point upstream, when there are the variable traversal times between the two points.

If recirculation is complete, it is postulated that points 1 and 2 coincide in a closed circuit, becoming a single observation point so that

$$a(t) = \int_0^t a(t-T)\,F(T)\,dT \ + \ \text{effect of first circulation.}$$

(Equation 8)

The second term is required because all recirculation effects at the point of observation arise from effects one circulation earlier. The dye already in the loop during the previous circulation will be included only after one initial circulation has occurred. The added term is merely the time relation for one traversal of the labyrinth which for a sudden injection is given by equation 6 so

$$a(t) = \int_0^t a(t-T)\,F(T)\,dT \ + \ a(0)\,\Delta t\,F(t).$$

(Equation 9)

This is an integral equation in which given F(t) and $a(0)\Delta t$ we find a(t). It is so called because the unknown function to be found in solving the equation is related to its own integral which includes the function F(t) called the "kernel."

The solution of the equation involves a principle which is of such potential utility to circulatory physiology that some discussion is warranted. The considerations are based on the so-called "method of Laplace transforms" which is discussed in books on electrical transient analysis.[4] We multiply both sides of equation 9 by e^{-kt} and integrate with respect to t from 0 to ∞. In so doing we convert a (t) to its Laplace transform $\alpha(k)$. Since we have introduced the variable k and removed t by integrating it out, the transform is a function of k. The reason for this maneuver is that the transform of a convolution becomes the product of the transforms of its components which greatly simplifies the solution of the equation. Thus in equation 9 we have simply

$$a(k) = a(k)\,\Phi(k) + a(0)\Delta t\,\Phi(k)$$

(Equation 10)

where $\Phi(k)$ is the transform of $F(t)$. Simple algebra yields the transform of the solution of the equation, namely

$$a(k) = a(0)\Delta t\,\Phi(k) / [1 - \Phi(k)]$$

(Equation 11)

The transform is converted back to $a(t)$ by the process of inversion, which is facilitated by the use of tables of transforms and their inverses which are available.[4,9] The first discussion of the method of Laplace transforms in the study of indicator dilution techniques was given by Stephenson.[15]

In the present case the quantitative discussion of complete circulatory mixing would require a knowledge of the kernel $F(t)$ for the entire circulation from the right heart through the pulmonary and systemic circuits and returning through the vena cava. This is not known but as a first approximation we could assume it to be similar to a random walk curve, taking for k the same value as for the pulmonary circuit. The result could then be reduced to time variables by the use of standard cardiac output and plasma or blood volume figures.

Although the random walk curve is based on a rational set of theoretical assumptions, the curves are not very different for a set of equal uniformly mixed serial compartments, and the transforms for these latter expressions are much simpler and easier to handle. We accept the form of equation 4, but consider n now rather than κ as an empirical parameter and choose τ rather than t for the independent variable, thus we have

$$F(\tau) = n(n\tau)^{(n-1)}\,e^{-n\tau} / (n-1)!$$

$$\Phi(k) = n^n / (k+n)^n$$

(Equation 12)

whose transform by simple algebra is

$$a(k) = a(0)\Delta\tau / [(k/n+1)^n - 1]$$

(Equation 13)

This can be inverted by separation into partial fractions yielding $a(t)$. The case of $n = 4$ will serve as an illustration, thus

$$a(k) = a(0)\Delta\tau / [(k/4+1)^4 - 1]$$

$$= \frac{a(0)\Delta\tau}{2}\left\{1/\left[(k/4+1)^2 - 1\right] - 1/\left[(k/4+1)^2 + 1\right]\right\}$$

(Equation 14)

From tables[4] this yields

$$F(\tau) = \frac{a(\tau)}{a(0)\Delta\tau} = 2e^{-4\tau}[\text{Sinh } 4\tau - \text{Sin } 4\tau]$$

(Equation 15)

Figure 10 shows a plot of this relation. A similar plot is shown for $n = 8$ (Fig. 11).

The theory describes recirculation phenomena at the right atrium but the majority of observations are taken in the aorta, and thus it is necessary to add the further smearing or dispersing effect of the pulmonary circuit. This is simply done by multiplying the transform in equation 14 by the additional transform for the pulmonary circuit. Again we assume the same form for the local portion as for the over-all circulation, but consider for the moment that the volume of the pulmonary portion is some fraction f of the total. The resulting composite transform for observations in the aorta is

$$\frac{a_a(k)}{a(0)\Delta\tau} = \left\{\left[(k/n+1)^n - 1\right]\left[fk/n+1\right]^n\right\}^{-1}$$

(Equation 16)

For $f = \frac{1}{3}$ and for $n = 4$ or $n = 8$, the inverses are shown in Figures 10 and 11. As before, the solutions include only the component after one passage and the first passage contribution must be added. The entire solution is also included in the figures and the curves are not unlike the experimental results so often observed.

Many points remain to be investigated. Time will permit only a brief mention of a few. Little has been said about the transition effects which occur when two randomizing beds with widely different κ's are placed in series. What in general is the effective κ of the combination? The effect of two random walks in parallel is the same as that of a single equivalent one, only if the flow is shared between the two members in proportion to their volume. What kind of linear random walk do we have if $\varkappa$ varies smoothly through the labyrinth according to some prescribed functional relationship? What can we say about the 3-dimensional random walk when the particle can wander sideways into regions of faster or slower flow?

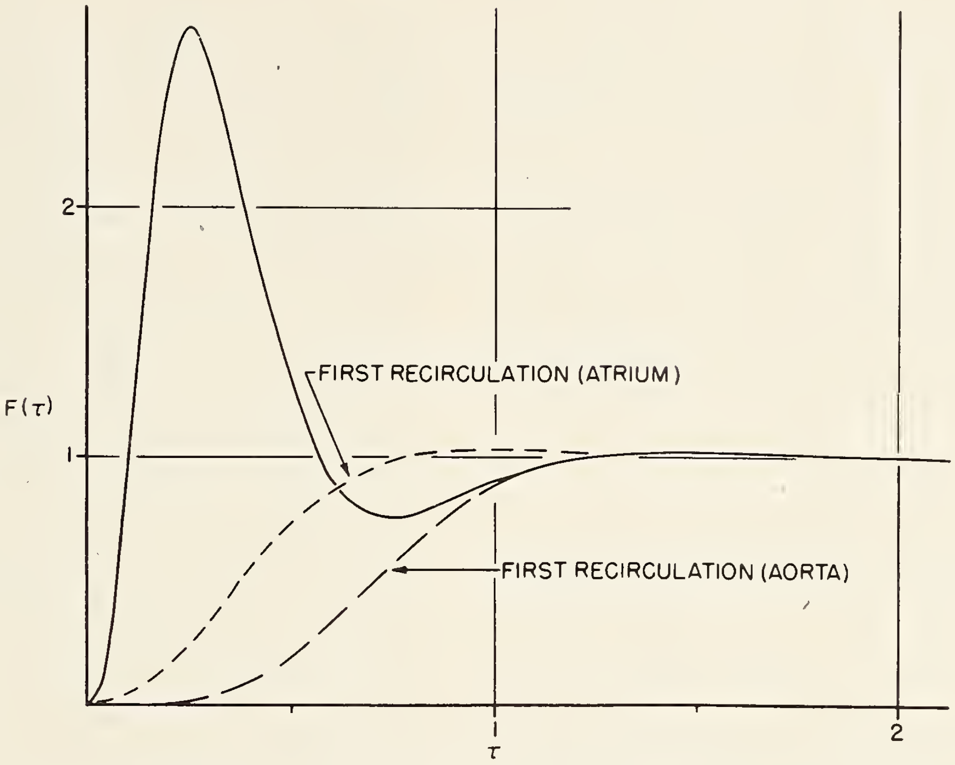

Fig. 10. Solution of the recirculation problem (see text). Solid curve represents theoretical dye curve for injection in the right atrium and observation in the aorta. Dotted curves represent recirculation observed in the atrium and aorta. Distribution in traversal times through the circulation approximated by the relation for the Poisson probability of three successes.

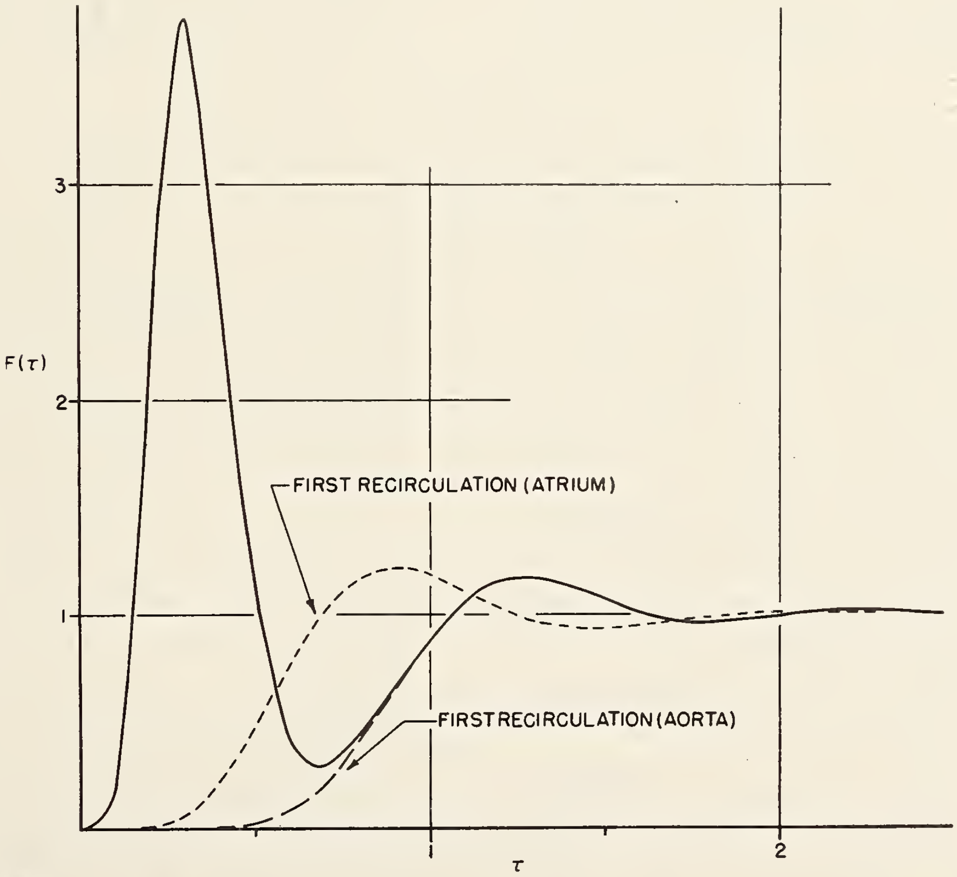

Fig. 11. Same solution as Figure 10 but for seven successes. Note the effect of the increased peaking of the traversal time distribution.

From these considerations alone we see that the present calculations are far from being a complete analysis of circulatory mixing processes. However, they seem to be at least partially successful, and it may be that a rational approach to the study of circulatory transients can be achieved in this way. In any event the results are interesting and the initial successes are sufficient to justify further work of this nature.

In conclusion, I wish to acknowledge the kind assistance of Drs. Howard, Dow, and Hamilton for the canine pulmonary outflow data, and Dr. Robert C. Little for the data on the canine liver perfusion. Some of the laborious numerical calculations were performed by Mr. George Atta of the ORNL Mathematics Panel.

References

1. Dow, P.; Hahn, P. F., and Hamilton, W. F.: The simultaneous transport of T-1824 and radioactive red cells through the heart and lungs. Am. J. Physiol., 147:493-499, 1946.
2. Feller, W.: An Introduction to Probability Theory and Its Applications. New York: John Wiley & Sons, 1950.
3. Friedlich, A.; Heimbecker, R., and Bing, R. J.: Device for continuous recording of concentration of Evans blue dye in whole blood and its application to determination of cardiac output. J. Applied Physiol., 3:12-20, 1950.
4. Gardner, M. F., and Barnes, J. L.: Transients in Linear Systems. New York: John Wiley & Sons, 1942.
5. Hamilton, W. F.; Moore, J. W.; Kinsman, J. M., and Spurling, R. G.: Studies on the circulation. IV. Further analysis of the injection method and of changes in hemodynamics under physiological and pathological conditions. Am. J. Physiol., 99:534-551, 1931.
6. Howard, A. R.; Hamilton, W. F., and Dow, P.: Concentration curves of two dyes injected simultaneously. Limitations of the continuous infusion method for measuring cardiac output by dye dilution. Am. J. Physiol., 175:173, 1953.
7. Little, R. C., and Kelly, H. B.: Removal of radioactive gold colloid by perfused mammalian liver. Am. J. Physiol., 173:265-269, 1953.
8. MacIntyre, W. J.; Pritchard, W. H.; Eckstein, R. W., and Friedell, H. L.: The determination of cardiac output by a continuous recording system utilizing iodinated (I^{131}) human serum albumin. Circulation, 4:552-556, 1951.
9. McLachlan, N. W., and Humbert, P.: Formulaire pour le Calcul symbolique. In: Memorial des Sciences Mathematiques de l'Academie des sciences de Paris. fasc. C. Gauthier-Villars, Paris, 1950.
10. Newman, E. V.; Merrell, M.; Genecin, A.; Monge, C.; Milnor, W. R., and McKeever, W. P.: The dye dilution method for describing the central circulation. An analysis of factors shaping the time-concentration curves. Circulation, 4:735-746, 1951.
11. Nicholson, J. W.; Burchell, H. B., and Wood, E. H.: A method for the continuous recording of Evans blue dye curves in arterial blood and its application to the diagnosis of cardiovascular abnormalities. J. Lab. & Clin. Med., 37:353-364, 1951.
12. Nylin, G. N., and Celander, H.: Determination of blood volume in the heart and lungs and the cardiac output through the injection of radiophosphorus. Circulation, 1:76-83, 1950.
13. Sheppard, C. W.; Overman, R. R.; Wilde, W. S., and Sangren, W. C.: The disappearance of K^{42} from the nonuniformly mixed circulation pool in dogs. Circulation Research, 1:284-297, 1953.
14. Sheppard, C. W., and Savage, L. J.: The random walk problem in relation to the physiology of circulatory mixing. Physical Rev., 83:489-490, 1951.
15. Stephenson, J. L.: Theory of the measurement of blood flow by the dilution of an indicator. Bull. Math. Biophys., 10:117-121, 1948.

FLOW AND PRESSURE IN THE ARTERIAL SYSTEM

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36. Pieper, H., and Wetterer, E.: Verhandl. d. deutsch. Gesellsch. f. Kreislaufforsch., 1953.
37. Ranke, O. F.: Ztschr. f. Biol., 95:179, 1934.
38. Rashevsky, N.: Bull. Math. Biophys., 7:25; 7:35; 1945.
39. Rein, H.: Ztschr. f. Biol., 87:394, 1928; Ergebn. d. Physiol., 45:514, 1944.
40. Remington, J. W., and Hamilton, W. F.: Am. J. Physiol., 148:25, 1947.
41. Remington, J. W.: Federat. Proc., 11:750, 1952.
42. Schmitt, F.: Ztschr. f. Biol., 101:259, 1943.
43. Schütz, E.: Ztschr. f. Biol., 91:515, 1931.
44. Shipley, R. E.: Rotameter. In V. A. Potter's Methods in Medical Research. Vol. 1, p. 327. Chicago: Yearbook Publ., 1948.
45. Wagner, R.: Ztschr. f. Biol., 92:54, 1931.
46. Warner, H. R.; Swan, H. J. C.; Connolly, D. C.; Tompkins, R. G., and Wood, E. H.: J. Appl. Physiol., 5:495, 1953.
47. Weber, E. H.: Ber. Sächs. Ges. d. Wissensch., 1850:166.
48. Wetterer, E.: Ztschr. f. Biol., 98:26, 1937; 99:158, 1938.
49. Wetterer, E., and Deppe, B.: Ztschr. f. Biol., 99:320, 1939; 100:105, 1940.
50. Wetterer, E.: Ztschr. f. Biol., 100:260, 1940.
51. Wetterer, E., and Deppe, B.: Verhandl. d. deutsch. Gesellsch. f. Kreislaufforsch., 15:91, 1949.
52. Wetterer, E.: Ztschr. f. Biol., 101:332, 1943.
53. Wetterer, E., and Pieper, H.: Ztschr. f. Biol., 105: 49, 1952.
54. Wetterer, E., and Pieper, H.: Ztschr. f. Biol., 106: 23, 1953.
55. Wezler, K., and Böger, A.: Ergebn. d. Physiol., 41: 292, 1939.
56. Wezler, K.: Kongr. Deutsch. Physiol. Gesellsch., 1951.
57. Wezler, K., and Sinn, W.: Das Strömungsgesetz des Blutkreislaufs. Editio Cantor KG., Aulendorf: 1953.
58. Whittacker, S. R. F., and Winton, F. R.: J. Physiol., 78:339, 1933.
59. Wiggers, C. J., and Katz, L. N.: Am. J. Physiol., 58:439, 1921.
60. Wiggers, C. J., and Baker, J.: Lab. Clin. Med., 10:54, 1924.
61. Wiggers, C. J.: Am. J. Physiol., 123:644, 1938.
62. Wiggers, C. J.: The pressure Pulses in the Cardiovascular System. London: 1938.
63. Wood, E. H.: Proc. Staff Meet. Mayo Clinic, 28:58, 1953.

THE RELATION BETWEEN THE STROKE VOLUME AND THE PULSE PRESSURE

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IT would be well nigh impossible to trace the origin of the notion that the pulse pressure is directly related to the stroke volume. Every physician who places his fingers over the radial artery, does so with the firm conviction that the strength of the beat he feels is set by the amount of cardiac discharge. Our province in this symposium is to see to what extent this subjective impression can be set into quantitative terms. Near the turn of the century, the first step in such a quantitation came with the technology with which pressure excursions in man could be approximately determined in millimeters of mercury. Shortly after, Erlanger and Hooker[2] noted that when the pulse pressure, so recorded, was multiplied by the heart rate, the product tended to be reasonably constant. In those conditions where the cardiac output was presumed to be increased, the product was elevated. Conversely, when the total flow should have been low, as after hemorrhage, the product was decreased. This implied that the pulse pressure was varying directly with the stroke volume. Since cardiac output of man was not, at that time, measurable, physiological conclusions reached on the basis of the change in product were entirely speculative. The quantitative testing fell to workers using the most common method of assessing stroke volume, the cardiometer. On the basis of their work on open-chest animals, they rather promptly discarded, almost *in toto,* the notion that stroke volume could be predicted with any reasonable accuracy from the pressure change. Explanations for this seemingly illogical failure were not clearly put, which perhaps explains why newer generations of physiologists have not remained convinced that the problem is hopeless.

Upon hindsight, we could construct a rather forceful story for the cardiometer workers, with most of the points used, taken from work already published before their investigations were done. In 1880, for example, Roy[12] reported volume-pressure measurements for segments of aortas which showed that the volume equivalent of a given pressure change was different at different pressure levels. This nonlinearity of the volume pressure curve should have been obvious. Even if the aortic wall followed Hook's Law in its stretch, and showed a linear tension-length relation, conversion of tension to pressure would be through the first power of the radius, and of length to volume through the square of the radius, so that the volume-pressure relation must now be curved. Since the aorta does not follow Hook's Law, the curvature is even more pronounced. The volume gain per unit pressure rises therefore increases as pressure rises from 0 to 30 to 40 mm. Hg, after which it becomes approximately constant. At a pressure level of about 100 mm. Hg, the volume gain becomes progressively less. This second inflection is attributable to the complex architecture of the aortic wall. The physician is certainly well aware of it, for he would not think of ascribing an enormously large cardiac discharge to a hypertensive pressure excursion of, say 270/130. For some curious reason, some experimental workers tend to ignore it, and assume a straight-line relationship between volume and pressure obtained at all working pressure levels.

Unfortunately, these inflection points do not lie at the same pressure levels in all individuals, so that there can be no common prototype curve applicable to all aortas. In addition, intra-individual differences in aortic size are quite large. In a group of nine dogs, for example, at a diastolic pressure of 75 mm. Hg, the total aortic volumes varied from 19 to 39 cc., the larger value representing a departure of 39 per cent from the mean of 28 cc. One factor contributing to this variation must be body size. We cannot tell from our data whether this variation with size would be better related to body weight or to surface area. Since we are accustomed to expressing cardiac output in terms of surface area, all arterial bed volumes were corrected to figures per sq. M. body surface. When the above dog aortic capacities were so

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revised, the largest error in the group was reduced from 39 to 29 per cent. Variability still remaining after such a size correction has been made, can be laid to congenital differences or to the effect of age. Roy had mentioned that an aortic segment from a young individual showed a different distensibility pattern from that of an older person. This effect of age was later emphasized in greater detail by Hallock and Benson,[3] who, by publishing only average curves, gave the impression that the change was a progressive and orderly one. When we surveyed a group of forty-eight human aortas,[11] we found that the capacity change with age was only a trend, and that the degree of overlap between age groups was very large. This variation can perhaps best be stated by the fact that we found a "young" aorta in a man of sixty-eight, and an "old" one in a girl of eighteen. Roy reported something which I do not believe has been repeated since, that after a long wasting disease, the dog aorta showed the stretch curve of an "old" aorta. It is quite possible that the variations with age are reflecting not the actual years of life, but rather the history of the number of periods of disease and circulatory stress. Certainly the enlargement often attendant upon hypertension can be seen in both the young and old individual.

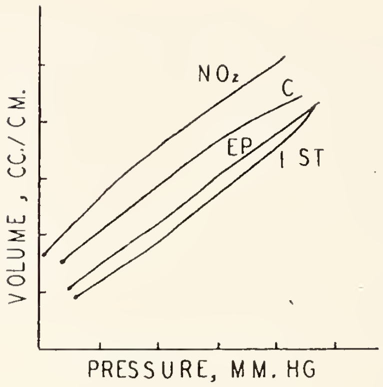

Fig. 1. Volume-pressure relations for a ring of ascending aorta as first removed from the body (1st), after several stretchings (C), after addition of epinephrine (EP), and after addition of $NaNO_2$ (NO_2).

In this "aging" process, as the aorta increases its capacity, the relative volume gain per unit pressure rise, as related to this initial capacity, is diminished. This change, the physicist would clearly term a decreased distensibility. Yet when the actual volume-pressure relations for all forty-eight aortas were plotted on the same figure, the most constant feature between them was the actual volume gain, without any reference to initial size. This relative constancy is fortuitous, for it is this actual volume increase that we wish to know when we would quantitate the pulse pressure in terms of volume. What a relatively stable actual volume increase, in face of the marked differences in relative distensibility, would mean to the physicist can hardly be treated in the time allotted. Individual variation away from the average net uptake figures averaged 25 to 30 per cent for the human series and 15 to 20 per cent for the dogs. Intrinsically, then, if we use these average values to predict actual volume uptakes from the pressure change in living individuals, we can hardly expect an accuracy greater than this. Actually, the error has often turned out to be less, as though the autopsy series contained an undue number of "atypical" aortas.

The cardiometer workers were not protesting the use of the pulse pressure as a measure of stroke volume between individuals, however. Where they had failed was in finding corresponding changes in pulse pressure when the stroke volume was acutely changed in the same dog. This implies that arterial distensibility shows physiological variation. The arteries have muscular walls. Even the aorta has variable amounts of smooth muscle. There is no reason to suppose that this muscle will not be influenced by nervous and humoral stimuli. MacWilliam, in 1902,[6] reported that when the aorta was first removed from the body, it was small and quite firm. After manipulation, it became larger and flabby. Stretch curves made on aortic segments in the two states showed that the firm aorta had a greater relative distensibility. The sort of changes he was observing can be illustrated by some recently recorded volume-pressure relations for a ring of dog ascending aorta, as shown in Figure 1. Upon first removal from the body (1st) the ring was small. After several stretchings, it was considerably larger. After the addition of epinephrine to the bathing medium, the ring again became contracted, while after $NaNO_2$ was added, it was relaxed. Just as was the case of the capacity changes in aging, however, the actual volume-pressure curves show a fairly good parallelism despite these tone changes. This means that an average volume uptake table can discount the effect of tone changes.

When Dr. Hamilton and I started on this problem, it seemed that the really major source of error had not even been considered. So far I have been speaking as though the only variable between pulses was the height of the pressure rise. Actually, in different circulatory conditions, the central pulse contour of the dog may take quite divergent forms, with the peak falling in early, mid or late systole, and the duration of ejection varying from 80 to 230 millisecond. The pulse pressure itself cannot be expected to signify these different contours. The volume uptake of the arterial bed should instead be related to the time-pressure area under the systolic portion of the pulse curve. But the problem is even more complex. The heart is ejecting into one end of a long network of tubes; all of which comprise the arterial receiving chamber. At the very first of systole, only the ascending aorta is accepting blood, and a given volume input must produce an inordinately large pressure rise. As the pulse wave moves out the vascular tree, the size of the reservoir is constantly increasing. Some means must be found to weight the parts of this time-pressure area for the progressive increase in reservoir size.

To evaluate the dimensions of the receiving chamber, we need the net volume increases per unit pressure rise, and the various pulse wave transmission times at different pressures, for the arterial beds. The former were assembled from evidence based on pressure rises following injections of known amounts of blood, or on tension-length curves obtained by stretching rings of vessels.[10] For the latter, transmission times were recorded to various arteries easily entered by manometer needles, and the values for other beds assembled by reasonable extrapolations. Next, all vessels lying the same time-distance from the heart were grouped together, to be treated as though they comprised a single tube. The different values for the volume uptakes of these vessels were also lumped by the same pattern. Hence we can know, for this single tube, the volume gain per unit pressure rise for each millisecond of its length. Now if we assume that a given central pressure pulse will pass through this theoretical tube without any change in form, we can calculate, moment by moment, the volume uptake of all parts of the tube, which can be summated for the total volume uptake.

This total uptake does not represent the stroke volume. As the pulse wave reaches the terminal arterioles, drainage is accelerated because of the increased pressure. The extra blood so lost must come from the reservoir, and rob it of some of its uptake. This drainage is estimated in a rather gross fashion. We assume that the outflow rate will be linearly proportional to pressure, less 20 mm. Hg, which is taken as the level at which flow ceases. This pressure level presumably is altered under different physiological conditions, but there are no convenient handles by which it can be corrected to meet the specific circumstance. Let us also say that all of the above determined uptake drains from the arterial reservoir during the succeeding diastole. Systolic drainage then can be calculated by the ratio of the time pressure areas under the systolic and diastolic parts of the pulse, multiplied by the uptake. Yet not all of systole is available for systolic drainage, for time is required for the pulse wave to reach the terminal arterioles. Transmission times to the various large drainage beds were obtained by extrapolations. Next, the fraction of the total cardiac output that normally flows through each bed was arbitrarily assigned, using values taken from the literature. The products of the flow fraction and the transmission time for all beds were summed, and averaged, to give a flow-weighted time value that we call Tw. This Tw then represents the mean time required for the pulse wave to reach an arteriole that carries an average amount of the total flow. This term Tw has a specific definition. The use of the symbol for times values differently derived can seemingly only lead to confusion.[13]

The effective systolic time for drainage can now be taken as the total systolic duration less Tw. Having calculated the total volume of systolic drainage, a fractionation between beds can be made, and the time course of systolic drainage determined. When the uptake and the drainage curves are summed, we should have the total ejection curve.

When we desire only the stroke volume rather than the ejection curve, the calculation need not be so elaborate. The whole stroke volume must be delivered at the time of the incisura, when the valves close. We should know the uptake values for the different regions of the composite tube at this moment. For simplicity, the tube is treated as composed of four segments only, each segment beginning its uptake when the pulse wave has reached the mid-point.[4] These half-times for each segment were tabulated. The pressure height for each segment, which should be proportional to end-systolic uptake, is obtained by laying back this time from the incisura on an actual pulse. The segment uptake for this pressure height can then be obtained from a table. When this process is repeated for each segment, we will have four uptake values which can be added for the total uptake.

The whole technique rests on the accuracy of transmission times, and the applicability of our set of average time values to different animals, or to different physiological conditions in the same animal. When we studied the arch-iliac transmission time for a number of animals at a given pressure level, we found the individual variations to be about 10 per cent of the mean. We were not able to establish any systematic deviation according to body size, indicating that such a trend was being masked by a larger variation attributable to tone changes and the other physiological factors which influence the wave velocity.[5] We therefore have no data to allow a correction for body size, nor can we safely speculate as to how these times would be affected when both wall thickness and vessel length are increased as the body becomes larger. As to the physiological variations, we have seen that when tone changes occur, they affect the relative distensibility, which conditions the pulse wave velocity much more seriously than the volume uptake values. It would seem safer to apply a standard set of transmission values than to make corrections every time the pulse wave velocity might change in a particular artery.

Alexander[1] did find a trend of arch-femoral transmission times according to body weight. When he calculated stroke volume with time values revised for each animal, the average difference between the values obtained and those given by our single table was some 6 per cent. The difference proved to be random rather than systematic. He concluded that the use of average time values had not introduced a really serious error, a belief we had had from the beginning.

We hear much of a lengthening, with the development of tortuosity, in the vessels in older humans. In our eighty-three aortas, length changes were certainly less clear than were changes in diameter, and individual variations not nearly so large. We should remember, also, that when age and/or hypertension produces such a lengthening, it also produces a thinning of the wall and a decreased relative distensibility. The resultant increased pulse wave velocity might easily compensate for the greater length.

The quantitative justification of the volume calculations, as outlined, requires a check with a recognized measure of cardiac output, done simultaneously. No other procedure, however, measures individual stroke volume, but rather gives a value for mean flow over a relatively long period of time. Since dog pulses may vary quite appreciably from beat to beat, the selection of single pulses which shall typify the whole time span is a problematical procedure. Making as reasonable guesses as possible as to pulse selection, the contour calculation has been compared with the Fick or the dye injection techniques in four different laboratories, and the results in three are fairly similar. The average disparity between the two procedures is from 10 to 15 per cent.[7] The results of some 400 such comparisons, done at Georgia, are shown in Figure 2. These points cover a wide range of experimental conditions, including both hypertension and hypotension, acute and chronic.

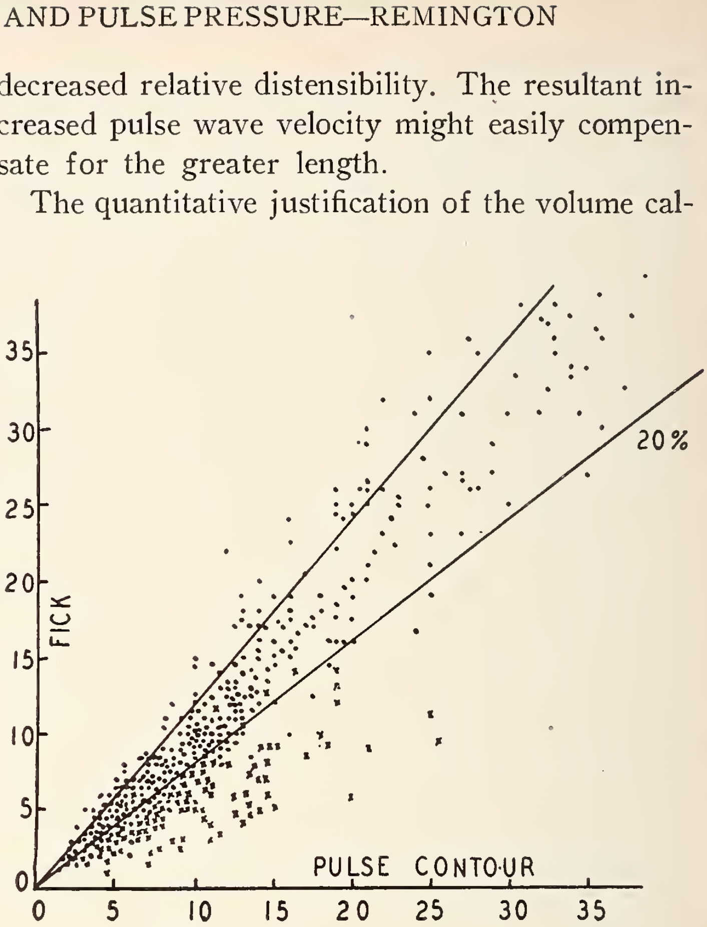

Fig. 2. A comparison of the stroke volumes obtained by the Fick procedure, using oxygen, and the Pulse Contonur calculations for 400 experimental cases in dogs. The lines represent a spread of ±20 per cent from the line of identity. For the difference between points and crosses, see text.

These points are fairly sharply divided into two clusters. The dots group around the line of identity without a systematic deviation. Seventy-three per cent of them lie within the limits of ± 20 per cent. These are from a great many different circulatory states in dogs in relatively good condition. The crosses, on the other hand, show a large number of poor fits with the Fick results, with the disagreement always in favor of an excessive pulse pressure for the corresponding stroke volume. The aberrant pulse contours tend to have a common form, with a very sharp anacrotic pressure rise, poorly sustained pressure in later systole, and a relatively brief duration of ejection.[8] These pulses come, in the main, from animals in what we might term a "normatensive" shock state—animals with but slightly subnormal pressure levels, suffering from only a mild blood volume reduction, but with cardiac output levels that can only lead to death. It is curious that as an open-chest dog deteriorates, it is prone to enter this peculiar state. Why the distensibility behavior of the arterial bed is changed so radically, and by what mechanism, is a most intriguing problem.

The fact that the pulse contour calculation is not applicable to all situations is of course disappointing. But we must not let this disappointment blind us to the fact that if we discard pulses of the aberrant form, and avoid conditions where such aberrancies might appear, the technique can yield a great deal of most profitable information.

In developing a case for the cardiometer workers, we have presented a number of points that could have sufficed to strengthen their case. However, we have also attempted to discredit each of them. The only good reason remaining for the reported lack of correlation was in the assumption that the pulse pressure could typify a pulse contour. It is interesting in this regard that when the method was applied to some records published long ago by Yandell Henderson, the calculated stroke volumes agreed quite well with those he obtained with a cardiometer.[9] Yet even this assumption should have allowed a recognizable trend between stroke volume and pressure change in the earlier work, unless enough of their experimental animals had entered the shock state. In addition, I am often reminded that the favorite device for adjusting the pressure level in their preparations was an occlusion of the aorta, which of course radically altered the size of the receiving chamber. Curiously enough, an increased pressure excursion, with relatively the same stroke volume, produced by such an occlusion seems to have been one of the crowning pieces of evidence that a relation between the two could never be expected.

How much of this methodology is applicable to the human? When we derived the dog technique, central pulse contours in the human were not being recorded. We did collect a series of brachial pulses, recorded directly from the artery at the same time that a Fick determination of cardiac output was being done. A large portion of these were borrowed from the files of Dr. Andre Cournand. With not a great deal of confidence in the results, we empirically constructed a pulse contour method for these pulses. The lack of confidence stemmed from the fact that these pulses often showed only a superficial resemblance to what we might expect of a central pulse, if a human resembled a dog. From the records I have seen in Dr. Wood's laboratory at the Mayo Clinic, this premonition seems to have been well founded. These brachial pulses usually did not show any remnant of the incisura, or even any constant landmark which could be taken as marking the end of systole. The brachial pressure values themselves were heightened to variable degrees by fusion with reflected waves. Curiously enough, despite these handicaps, the contour calculations gave an average fit of ± 20 per cent from the Fick value.[11]

If variability in the basic data were comparable, central human pulse contours would seem better suited to accurate volume predictions than are dog pulses. Changes in contour seem far less extreme, heart rates are much more stable, beat-to-beat variations in pressure much smaller, and the length of systole much longer and more stable than for the dog. The long systole minimizes the effect of the expansion of the arterial reservoir during systole. On the other hand, it increases the systolic drainage fraction of the calculated stroke volume. We have followed with considerable interest the efforts of the Mayo group to construct a contour method for the central pulse of man.[13]

Limited as we were to brachial pulse records, we doubted that the accuracy achieved by application of the contour calculation methods to an unsuitable pulse, was rewarding enough to continue the approach. We therefore returned to the original Erlanger and Hooker notion that the brachial pulse pressure, *per se*, could be quantitatively related to the stroke volume. As we have said, this implies a stereotype contour form.

From the pulse pressures and the known stroke volumes, we derived a set of volume factors

TABLE I. FACTORS FOR THE PREDICTION OF STROKE VOLUME, PER SQ. M. BODY SURFACE, FROM THE PULSE PRESSURE

Pressure	Volume Factor	Pressure	Volume Factor	Pressure	Volume Factor
mm. Hg.	cc.	mm. Hg.	cc.	mm. Hg.	cc.
20	0	100	81	180	140
30	10	110	90	200	148
40	21	120	100	220	155
50	31	130	108	240	161
60	42	140	115	260	167
70	52	150	122	280	173
80	62	160	128	300	179
90	71	170	134		

(Table 1), corrected for body size and for the inflections in the aortic distensibility curve. If, for example, the brachial pulse pressure was 120/80, then the predicted stroke index, from Table I, would be 100-62, or 38 cc. As a rule of thumb, over the usual pressure range, 1 mm. Hg, pressure rise is about equivalent to 1 cc. stroke volume/sq. M. body surface. The average error of these predictions was about 25 per cent. How good a fit this is depends upon one's viewpoint. If workers with the ballistocardiogram got such an agreement, they would be quite content. For the experimental investigator it leaves much to be desired.

For the diagnostician, a prediction with this error should still furnish a great deal of useful information as to the direction and general magnitude of cardiac output changes. After all, the flow changes with which he is most concerned tend to be larger than the intrinsic error. In case of critical, or small, changes in flow, corroborative support from a direct cardiac output determination would, of course, be necessary.

References

1. Alexander, R. S.: (Unpublished Observations).
2. Erlanger, J., and Hooker, D. R.: Johns Hopkins Hosp. Rep., 15:179, 1904.
3. Hallock, P., and Benson, I. C.: J. Clin. Investigation, 16:595, 1937.
4. Hamilton, W. F., and Remington, J. W.: Am. J. Physiol., 148:14, 1947.
5. Hamilton, W. F.; Remington, J. W., and Dow, P.: Am. J. Physiol., 144:521, 1945.
6. MacWilliam, J. A.: Proc. Roy. Soc. London s. B., 70:156, 1902.
7. Opdyke, D. F.: Feder. Proc., 11:733, 1952.
8. Remington, J. W.: Feder. Proc., 11:750, 1952.
9. Remington, J. W.: Am. J. Physiol., 162:273, 1950.
10. Remington, J. W., and Hamilton, W. F.: Am. J. Physiol., 148:25, 1947.
11. Remington, J. W.; Noback, C. R.; Hamilton, W. F., and Gold, J. J.: Am. J. Physiol., 153:298, 1948.
12. Roy, C. S.: J. Physiol., 3:125, 1880.
13. Warner, H. R.; Swan, H. J. C.; Connolly, D. C.; Tompkins, R. G., and Wood, F. H.: J. Appl. Physiol., 5:495, 1953.

DIAGNOSTIC PROCEDURES IN DETERMINING CONGENITAL CARDIAC DEFECTS

Aside from a thorough history and physical examination, the most important single diagnostic procedure which can be done in the physician's office is careful fluoroscopic examination. A good deal will be missed if the fluoroscopy does not include the right and left anterior oblique views in addition to the usual posterior-anterior view. Fluoroscopy will demonstrate enlargement of the individual cardiac chambers and will give information as to the contour and activity of the pulmonary vessels.

The electrocardiogram, especially with precordial leads, is useful in demonstrating ventricular hypertrophy. Right axis deviation is found in the tetralogy of Fallot, left axis deviation in tricuspid atresia.

Catheterization of the heart may be helpful in a difficult case. A special catheter is inserted into an arm or leg vein and gently advanced under fluoroscopic visualization to the right auricle. The catheter is then passed through the valve into the right ventricle and, usually, into the pulmonary artery.

In the case of an over-riding aorta, the catheter may be passed from the right ventricle directly into the aorta. It is sometimes possible to pass a catheter through an auricular or ventricular septal defect. Blood samples are taken from the different chambers and analyzed for oxygen content. Information may thus be obtained concerning left-to-right shunts, such as patent ductus, by analysis of the oxygen content of blood samples drawn from the right ventricle and pulmonary artery. (The latter sample will have a greater oxygen content because of the flow of oxygenated blood from the aorta.) The presence of auricular and ventricular septal defects will similarly be demonstrated. By a manometer attachment to the catheter a recording of pressures during systole and diastole is made. This is helpful in showing the presence of pulmonary stenosis when a recording of the pressures in the right ventricle is high and in the pulmonary artery is low.

Catheterization must be carried out by a well-trained team in a hospital, and usually general anesthesia is necessary.

Angiocardiography is a procedure in which a radio-opaque dye is injected into a vein and with a special camera x-ray films of the heart are taken in rapid sequence. These films visualize the great vessels and individual chambers of the heart as they are filled by the dye.

Angiocardiography is particularly useful in the diagnosis of defects such as coarctation of the aorta. It may be of some help in the diagnosis of transposition, truncus arteriosus, tricuspid atresia and anomalous venous drainage. In an older, co-operative child, it may be carried out without anesthesia.

—From Diagnosis of Congenital Cardiac Defects in General Practice, American Heart Association

QUANTITATION OF STROKE VOLUME CHANGES IN MAN FROM THE CENTRAL PRESSURE PULSE

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IN the preceding paper Dr. John Remington has given us an historical background to the problem of estimation of left ventricular stroke volume from an arterial pressure pulse. He has also presented some of his own work in this field, and has pointed out some of the difficulties he encountered in trying to apply this method to the study of man. I should like to present briefly a somewhat different approach to the problem. The work I shall describe was carried out in conjunction with Dr. Earl Wood, and others in the physiology laboratory of the Mayo Clinic.[4]

The pressure wave generated in the aorta and large arteries by the periodic ejection of blood into this system by the left ventricle is a complex phenomenon. Necessarily, in any attempt at the present time to quantitate volume changes, from a recording of this pressure pulse, must be considered only a first approximation, and the attempt is justified only if the results obtained possess a useful degree of accuracy, and if the attempt suggests new and more precise means of approaching the problem.

Inasmuch as the pressure wave traverses the arterial system with a finite velocity, pressure in this system is a function of at least two variables, namely, time in the cardiac cycle (T), and distance down the arterial bed (X). Pressure can be recorded as a function of time at a fixed point in the system. If a plot of pressure as a function of distance at certain specified time, could be obtained, namely the onset of systole and the end of systole, the difference between the mean pressures over the length of the system at these two instants would be the pressure change corresponding to the volume change which Dr. Remington has named the systolic uptake—that is, the volume of the arterial bed at the end of systole minus its volume at the onset of systole.

In order to calculate (P) as a function of (X) from a record of (P) as a function of (T), one must know (X) as a function of (T). The first derivative of this relationship $\frac{(dx)}{(dt)}$ is the pulse wave velocity, and this has been found to be an increasing non-linear function of (X), and varies considerably from one individual to the next. However, it was found that changes in mean pulse wave velocity between two points in the arterial bed (for instance between the brachial and the radial artery) are proportional to changes in mean velocity between any other two sites (aortic arch to femoral artery for instance). Thus changes in pulse wave velocity between two recording sites in the arterial bed can serve as an index to the time of transmission of the pulse wave down the whole system (Dr. Remington's Tw).[2]

The length of that portion of the arterial bed that acts primarily as a passive elastic chamber is arbitrarily assigned a value of 60 cm. Then on the basis of an average pulse wave velocity in the resting supine subject of 6 meters/sec. in this segment, transmission time over the length of the system (Tw) would be 100 milliseconds at rest. A proportionality factor, between this and the measured transmission time at rest, between the two recording sites, is then calculated and used to estimate subsequent values for Tw in that individual. The accuracy of the resting, assumed value for Tw is not at all critical to value for stroke volume obtained by this method, since only changes in stroke volume are to be calculated.

In Figure 1 is shown a pressure-time record recorded through a small catheter, the tip of which lay in the aorta, and another, whose tip lay in the femoral artery. The integrated mean pressure between the limits of the onset of systole on the central pulse and a point Tw back from this point, serves as an index to the mean pressure down the aorta and large arteries at this instant. The difference between the mean pressures down the system at the onset, and the end of

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systole, is the pressure increment which we have called the mean distending pressure, and which corresponds to the volume change—systolic uptake.

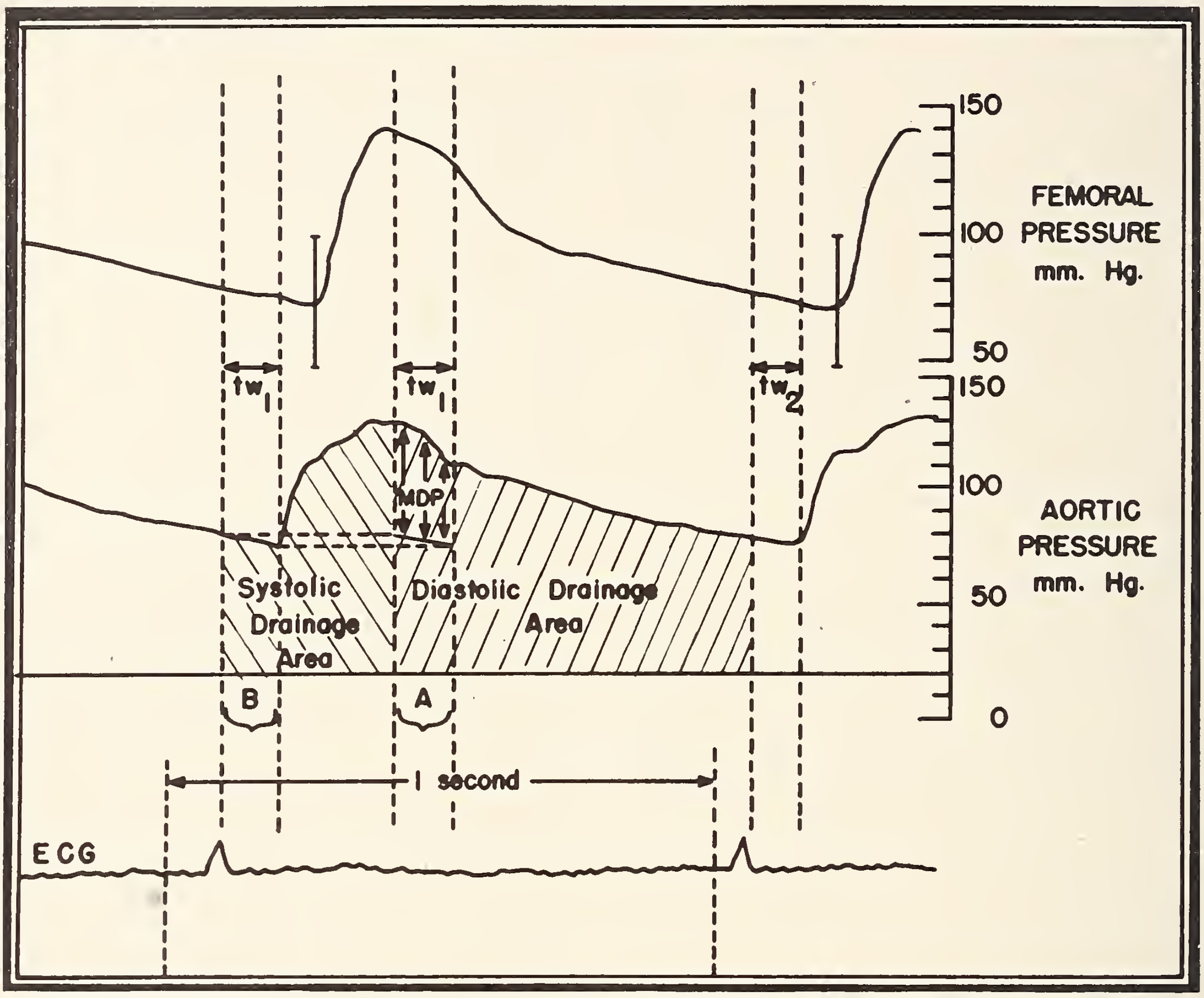

Fig. 1.

of this, a linear relationship between volume and pressure is used as an approximation in the method being described.

The other measurement to be made from the pressure record is also shown in this figure, namely, the ratio of the systolic to the diastolic time-pressure integral above 20 mm. Hg, corrected for the delay in transmission of the pressure wave to the periphery.

Dr. Remington has shown a plot of the volume-pressure relationships which they obtained by stretching rings of aorta from recently dead humans.[3] They found a marked individual variation in size and distensibility within any given age group. Although not evident in their plot of average values, examination of the individual values shows considerable variation in slope from one individual to the next. As a matter of fact, the data would indicate that the variation from linearity, when it occurs, is relatively small in comparison to the individual variation. Because

In order to arrive at this proportionality factor relating change in pressure to change in volume for a given individual, cardiac output is measured with the subject at rest by an independent method (either the Fick or the dye method) while the pressure pulse is being recorded. Systolic and diastolic time-pressure integrals and mean distending pressure are measured, from each pulse during the independent cardiac output measurement, and averaged.

A sample calculation, that illustrates the application of these equations to the measurement of changes in stroke volume produced by exercise, will clarify the preceding paragraphs.

Person at rest:
Pmd = 39 mm. Hg (measured value)
Sa/Da = 0.48 (measured value)
SV (dye) = 127 cc. (measured value)

Equation 8:

$$SV = U + \frac{Sa}{Da}U$$

$$127 = U + 0.48U$$

$$U = \frac{127}{1.48} = 86 \text{ cc.}$$

Equation 4:

$$k = \frac{U}{Pmd} = \frac{86}{39} = 2.21 \text{ (to be used in subsequent determinations).}$$

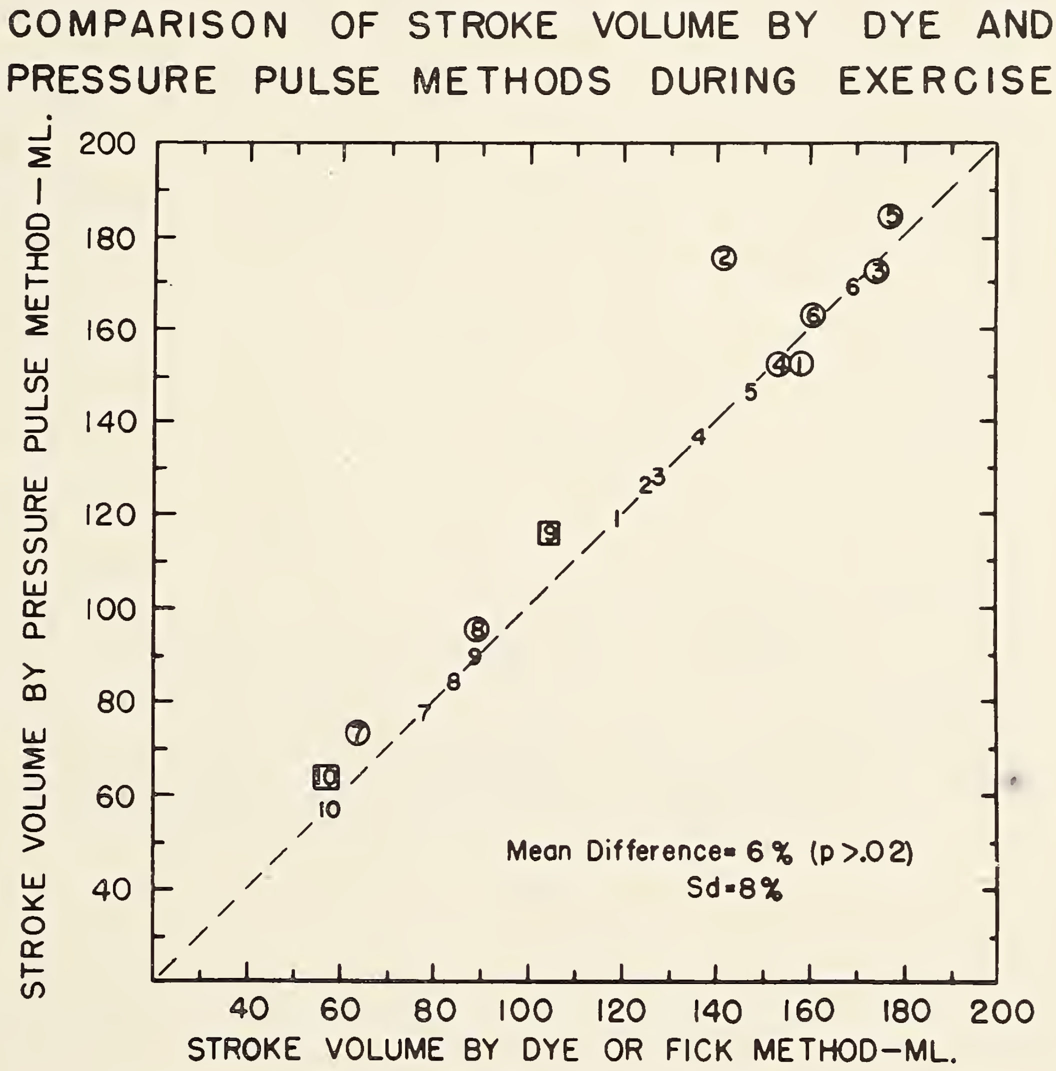

Fig. 2.

Person exercising:

$Pmd = 41$ mm. Hg (measured value)

$Sa/Da = 0.93$ (measured value)

Equation 9:

$$SV = kPmd\left[1 + \frac{Sa}{Da}\right]$$

$$= 2.21 \cdot 41\ (1 + 0.93)$$

$$= 174 \text{ cc.}$$

SV (dye) = 179 cc. (measured value).

Before showing the results of comparisons of this method with the Fick and dye method, I should like to describe briefly the techniques employed and some of the precautions taken to insure a faithful recording of pressure. Central arterial pressure-time records were obtained through a small plastic catheter[1] 0.5 min. interval diameter advanced either into a subclavian artery, through a thin-walled 18-gauge needle in a brachial artery, or into the aortic arch by way of a needle in a femoral artery. The proximal end of the catheter was attached to a three-way stopcock which allowed alternate recording with a strain gauge and a capacitance (Lilly type) manometer. The output of the manometers was fed into d'Arsenval type galvanometers and recorded photographically using a two meter light arm. This alternate recording arrangement was used to test the minimum response characteristics necessary to record a central arterial pressure pulse.[5]

After withdrawing the catheter at the conclusion of each experiment, the tip was placed in a low frequency oscillator chamber and the response of the system to square wave and sine wave pressure variations of varying frequency was tested. The strain gauge system was almost optimally damped with its peak response at 15 cps while the capacitance manometer was underdamped, overshooting 700 per cent at its peak frequency of 90 cps.

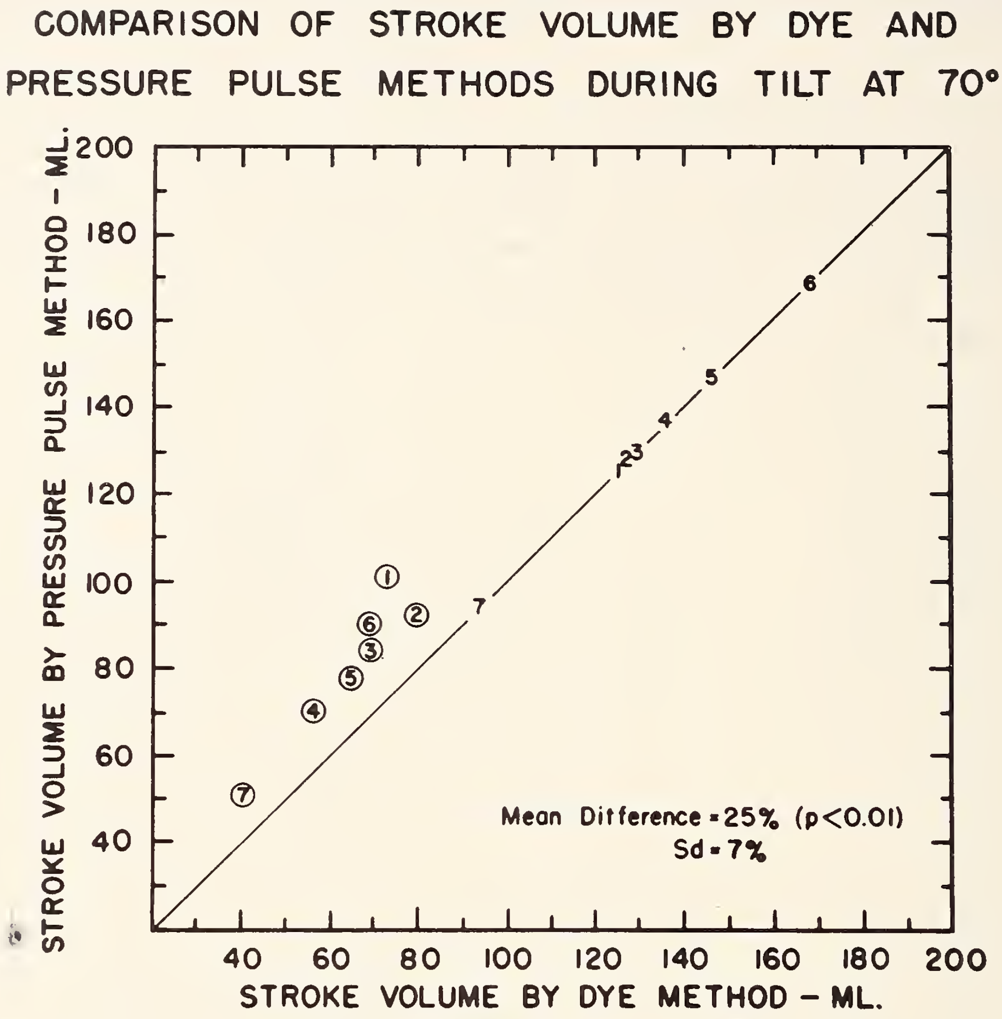

Fig. 3.

A comparison was made of aortic pressure pulses recorded by the strain gauge and the capacitance manometer-catheter systems. It is of interest that the vibrations observed, just preceding the onset of systole, and at the anacrotic and dicrotic notches, were of the resonant frequency of the catheter-manometer system, and thus do not represent actual pressure variations within the arterial system. No measurable difference in the pulse recorded by the two systems was found except in the time delay in onset as measured from the R wave of the electrocardiogram. The calculations made in the present study were carried out on recordings made with the strain gauge manometer. Because of the stability of this instrument, it was considered the manometer of choice for this type of study. Femoral artery pressure was recorded through a short catheter and right brachial and right radial artery pressure through indwelling needles. Dye concentration in arterial blood was recorded continuously with a curvette oximeter and an ear piece oximeter. Curves of dye concentration were recorded at a camera speed of 5 mm/sec. while the pressure pulses were recorded simultaneously with another camera at a speed of 80 mm/sec. In this method I have just described a record of a central and only one peripheral pulse is required.

Figure 2 shows a comparison of stroke volume by the dye or Fick and the pressure pulse methods during exercise and includes data from six normal subjects and four patients with mitral stenosis. The uncircled numbers on the line of identity are the resting values and are shown only to demonstrate the magnitude of the change in stroke volume that occurred during exercise. The circles are comparisons made during exercise with the dye method and the squares with the Fick method. In this series the mean stroke volume increased only 11 per cent, but the average exercise cardiac output was 182 per cent of the resting value and heart rate increased thirty-five beats per minute. The mean difference between the values obtained by the pressure pulse, and the control method was 6 per cent and the standard deviation of the differences was 8 per cent.

Figure 3 shows a comparison of stroke volume by the pressure pulse and the dye method carried out four to five minutes after tilting the subject to head up position, 70° from the horizontal. Under these circumstances a significant systemic difference was observed, but the standard deviation about this difference was small, being only 7 per cent. In the tilt position, stroke volume averaged 61 per cent of the resting value by the pressure pulse method, and 53 per cent by the dye method. The reason for the systematic difference in the tilted position is not clear as yet.

In Figure 4 are shown twelve comparisons of values for stroke volume obtained by the Fick and pressure pulse methods under a variety of physiologic conditions, including tilt to various positions and inflation of a new type antiblackout suit which applies 75 mm. Hg pressure to the entire body below the xyphoid. Under these circumstances no systematic difference was obtained and the standard deviation of the differences was 10 per cent. In this series of observations, mean arterial pressure varied from 81 to 149 mm. Hg, and heart rate from 84 to 125 beats/mm., providing a rather rigid test of the ability of the method to follow stroke volume changes under conditions producing marked alterations in the status of the cardiovascular system.

In conclusion, a method for estimation of stroke volume in man from a recording of a central and a peripheral pressure pulse has been presented. This requires the obtaining of a factor relating change in arterial pressure to change in volume, by carrying out a determination of stroke volume during a steady state period by an independent method. Inasmuch as the usefulness of a pressure pulse method lies primarily in its ability to follow rapid changes in stroke volume, this would not appear to be a serious disadvantage. Basically this approach differs from

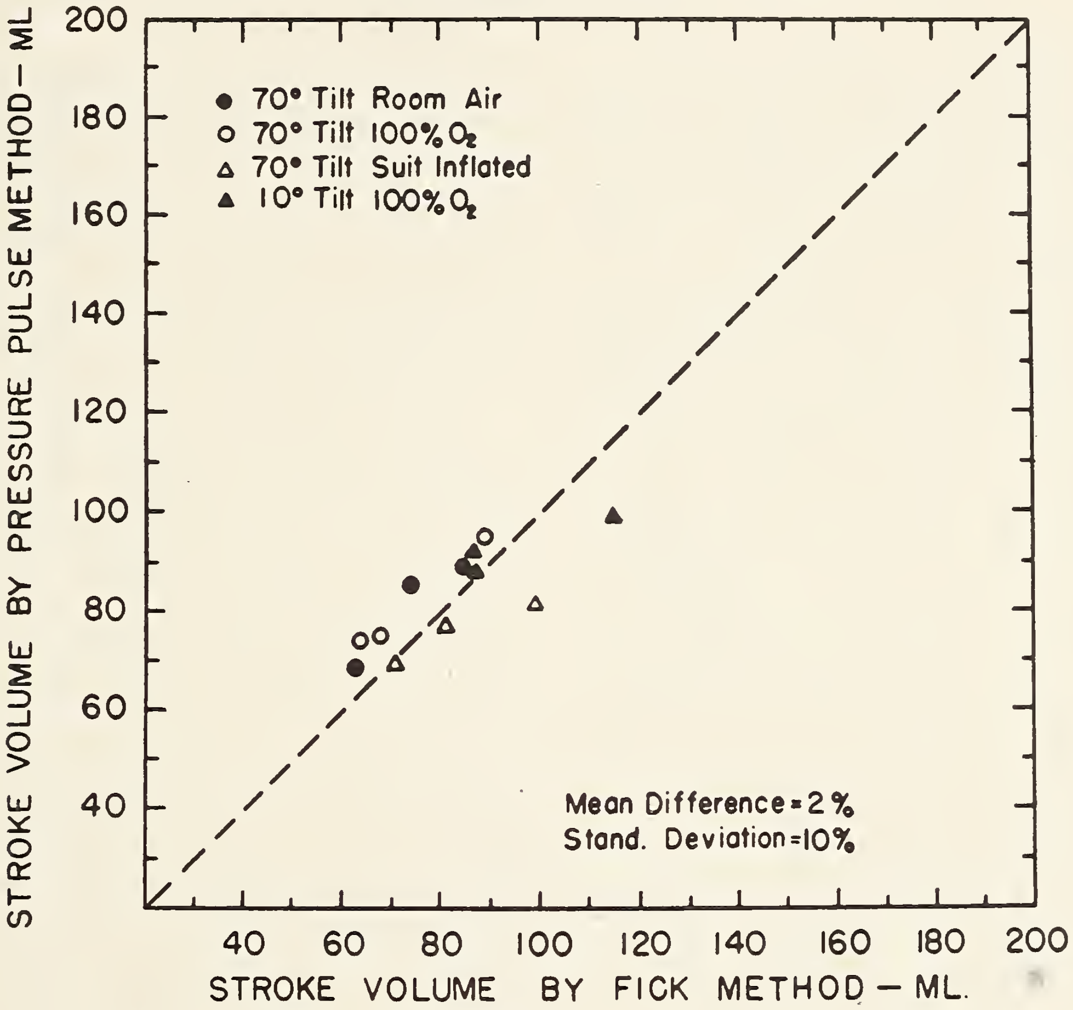

Fig. 4.

(Continued on Page 130)

CARDIAC CATHETERIZATION IN DIAGNOSIS OF CONGENITAL HEART DISEASE

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NOTABLE IMPROVEMENT in the diagnosis of congenital heart disease has been achieved as a result of the introduction of cardiac catheterization. The purpose of this communication is to point out information that can be obtained by this procedure as well as to indicate its shortcomings. Catheterization may provide useful data by the following means: (1) the demonstration fluoroscopically of the *introduction of the catheter* into an *abnormal pathway;* (2) the demonstration of an abnormal pressure gradient across the pulmonic or tricuspid valve, indicative of stenosis of the valve; (3) the demonstration of the identity of the systolic pressures in the right ventricle and brachial artery, indicative of an overriding aorta or a functionally similar condition; (4) the demonstration of the presence of arterialized blood in one of the chambers of the right side of the heart or in the pulmonary artery, indicative of a left-to-right shunt; and (5) the demonstration of arterial oxygen unsaturation not corrected by the administration of 100 per cent oxygen of a right-to-left shunt. The latter procedure does not involve catheterization, but is often carried out in conjunction with it.

In addition, the determination of flows, pressures, and resistances at rest and during various physiological stresses, permit a better appreciation of the abnormal physiological processes and a deeper insight into the clinical course and natural history of the disease.

1. *Introduction of catheter into or through abnormal pathways.*—This is one of the most reliable ways of diagnosing a congenital heart lesion if the abnormal pathway can be definitely identified. Catheters have been passed through or into the patent ductus arteriosus, aortic septal defects, atrial septal defects, anomalous pulmonary veins, ventricular septal defects, overriding aortas, double superior vena cavas and others. It is usually not difficult to determine the catheter's exact position with the aid of oblique and lateral as well as anterioposterior fluoroscopy. In my opinion it is, however, extremely difficult, if not impossible, to differentiate an anomalous pulmonary vein from an atrial defect, as the course pursued by the catheter may be very similar.

2. *Abnormal pressure gradient across a valve.*—There is normally no more than 1 to 2 mm. Hg difference in pressures between the right auricle and right ventricle during the first fraction of a second of diastole, or between the right ventricle and pulmonary artery, during systole. The most accurate and sensitive indication of stenosis of the tricuspid or pulmonic valve is the demonstration of a significant pressure gradient across the valve. Until 1946, only about seventy cases of pure pulmonic stenosis were described pathologically in the medical literature. Since the advent of physiological measurements, enabling pressures to be recorded on both sides of the valve, many hundreds of cases have been recognized. Pulmonic stenosis may now be considered to be one of the most common of congenital defects. I hope Dr. Bing will describe the manner in which the valvular type stenosis can be differentiated from the infundibular type by means of a cardiac catheter.

3. *Identical systolic pressure in right ventricle and brachial artery.*—With dextroposition or overriding of the aorta, the aorta and right and left ventricles are in direct communication during systole. With minimal overriding, there may be a difference in systolic pressures of the right ventricle and aorta. When free communication is present, however, the pressures are essentially the same *under all circumstances.* Brachial arterial pressure tracings in the adult have been found to be more representative of aortic pressure than femoral or radial artery recordings. In all but one of our cases with established overriding, identical brachial arterial and right ventricular systolic pressures have been demonstrated. The

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one exception had a brachial arterial pressure 20 mm. Hg lower than the right ventricular pressure; this was perhaps due to an anomaly of the arteries supplying the upper extremity.

Identical systolic pressures in the right ventricle and the aorta are also present in large intraventricular septal defects, cor triocular biatrium, and common truncus arteriosus. In these conditions, there is no anatomical overriding of the aorta, but there is free communication between the right and left ventricles and the aorta during systole. Thus identity of systolic pressures in the right ventricle and brachial artery is found in lesions with a wide anatomic variation but with a common physiological behavior.

In primary pulmonary vascular disease, or when pulmonary vascular complications have developed in mitral stenosis, ventricular or atrial septal defects, or patent ductus arteriosus, the right ventricular systolic pressure may be elevated to systemic levels and be identical with that of the brachial artery at rest. Our experience indicates the identity of such pressures to be more than mere coincidence. This group of lesions can readily be differentiated from those with free communication between right ventricle and aorta by altering the pressures with exercise or by the administration of hypertensive or hypotensive agents. In those with a free communication between right ventricle and aorta, pressures in both areas rise and fall together, whereas in the second group, the systolic pressure in the right ventricle and/or pulmonary artery rises on exercise much higher than that in the brachial artery.

4. *Left-to-right shunts.*—Left-to-right shunts can be recognized by noting the appearance of arterialized blood, that is, blood high in oxygen content, in the pulmonary artery, right ventricle, or right auricle. The interpretation of this may not be simple. For example, the presence of arterialized blood in the pulmonary artery may signify any of the following: (a) patent ductus arteriosus, (b) aortic septal defect, (c) blood aspirated from the pulmonary capillaries and veins. The latter occurs only when the catheter is wedged into a terminal branch of the pulmonary artery, and thus should not lead to the erroneous interpretation that a shunt is present. Differentiation between the first two, however, is impossible by determining the oxygen contents of pulmonary arterial blood samples.

The appearance of arterialized blood in the right ventricle indicates either: (a) ventricular septal defect, or (b) patent ductus arteriosus with pulmonic insufficiency. Usually there is no difficulty distinguishing the two by clinical means unless both defects co-exist.

Arterialized blood in the right auricle may be due to: (a) atrial septal defect, (b) anomalous pulmonary vein entering the right auricle, (c) ventricular septal defect with tricuspid insufficiency, (d) common auriculoventricular valve with shunt from left ventricle to right auricle, and (e) ? coronary auriculoventricular aneurysm. With the exception of ventricular septal defect with triscuspid insufficiency these lesions cannot be accurately differentiated from one another by cardiac catheterization. I am not sure that a coronary auriculoventricular aneurysm provides a sufficient amount of oxygenated blood to raise the oxygen content in the right auricle sufficiently to be recognized. An elevated oxygen saturation of blood withdrawn from the coronary sinus should be diagnostic. Atrial septal defect, is by far the most common of the lesions listed, and it is for this reason that this diagnosis is often made so dogmatically. I am a sinner in this regard. Actually, the above differential always exists.

Thus, abnormal physiology can be translated to abnormal anatomy only as an approximation. The precise location of the defect in the atrial, or ventricular septum, cannot be determined. The size of only the smaller defects, wherein there is a pressure gradient across them, can be calculated. The larger ones can easily be recognized, but it cannot be ascertained if it is a defect of 3 cm.^{2}, or one in which there is no septum, or only an irregular peripheral rim. These points are of extreme importance to the surgeon, but cannot be answered by cardiac catheterization.

The magnitude of the shunt can be calculated using the Fick principle. I agree with Dr. Stow and Dr. Visscher that the Fick output, under the most ideal conditions, lacks precision, and that when shunt flows in congenital heart diseases are calculated, the errors may be great. It was with considerable hesitation that we originally made such calculations, and at best they may afford only crude approximations. The figures are at times so grossly in error as to be ridiculous. We have studied two patients with presumable atrial septal defect in whom the oxygen content of the pulmonary arterial blood was the same as that taken from the brachial artery. The pulmonary

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blood flow, as calculated by the Fick principle, was infinite. Our most recent case had a calculated pulmonary blood flow of 70 liters per minute. Obviously the error in calculation was so great as to be meaningless, except that it indicated a large shunt.

As discussed previously, the main errors stem from the presence of extremely narrow arteriovenous oxygen differences, and the difficulty of obtaining representative mixed venous blood samples. Depending on the reliability of these two factors, the calculation of flows, shunts, resistances, and cardiac work may or may not be justified.

Not infrequently young patients are seen with a congenital murmur, but with no disability except for cardiac anxiety of iatrogenic origin. Cardiac catheterization, satisfactorily performed, fails to detect abnormalities of pressure, oxygen content of blood samples, or calculated flow. Two possibilities exist to explain the murmur—either that there is a lesion in the left side of the heart, for example, subaortic stenosis, or that there is one in the right side of the heart which is too small to be detected by this technique. If, on consideration of all information available, the latter possibility appears to be the answer, one can then reassure the patient that the lesion, no matter what it may be, is of no clinical significance, whatsoever, and that he may consider himself to have in essence a normal heart. There is one reservation that must always be added, that is the potential vulnerability to subacute bacterial endocarditis. Of all lesions under these circumstances, the one most easily escaping detection is a small ventricular septal defect. Although small atrial septal defects are undoubtedly undetected more frequently than any other lesion, by the time they produce a murmur, their detection is usually straightforward.

5. *Right-to-left shunts.*—In general, angiocardiography is a much more valuable method for identification and delineation of right-to-left shunts than cardiac catheterization. Right-to-left shunts are recognized by the failure of arterial blood to become fully saturated upon the administration of 100 per cent oxygen. However, the location of the right-to-left shunt cannot be determined by this method, although this can often, but not always, be accurately deduced by noting that a given chamber of the right side of the heart has a higher pressure than the corresponding chamber of the left side. Actually such studies are poor in the precise information they give, and have been greatly improved by the ether test of Soulié, and especially by the dye injection technique of Wood, which will be discussed by Dr. Swan in the next communication.

6. *Understanding of abnormal physiology.*—Perhaps the most important contribution of cardiac catheterization to congenital heart disease, is the knowledge that is being acquired with regard to the pathological physiology of the circulation in the different types of lesions. In the past, congenital heart disease has been studied mainly from the anatomical and embryological points of view. The physiology and clinical aspects are currently under intensive study. As corrective surgery becomes more and more applicable to the various lesions, such studies will increase. There are now considerable gaps between the anatomical, physiological, and clinical interrelationships of the various congenital lesions. These must, and will be, filled in in the years to come. What, for example, is the clinical course of patients with atrial septal defect? I do not know the answer clinically or physiologically. Cardiac catheterization, with all its handicaps and limitations, serves as the single most important physiological tool of approximate diagnosis and investigation. Other physiologic tools must be added to bridge some of the many gaps that exist in our knowledge of these diseases.

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Discussion

Dr. R. J. Bing, Baltimore, Md.: I would like to comment briefly on two problems. One, the relative diagnostic value of special tests as compared to clinical procedures, and secondly, some of the problems of pulmonic stenosis with particular reference to its surgical correction.

There has been a great deal of discussion on the relative value of clinical methods, versus special tests in congenital heart disease. Most of us agree, that in a great majority of patients with congenital malformations of the heart, a diagnosis can be made by clinical means. I use the term "clinical" very loosely, because it comprises not only the history and physical examination, but also electrocardiography and fluoroscopy.

Special tests are catheterization of the heart and angiocardiography. I would like to mention here some of the specially helpful points in the clinical diagnosis of congenital heart disease. As to history: repeated respiratory infections suggest the presence of increased pulmonary blood flow and/or pulmonary hypertension; anoxic convulsions are in line with a marked diminution in pulmonary flow, and hemoptyses suggest pulmonary hypertension. If a non-cyanotic child is reported to be dyspneic on slight exertion, the possibility of isolated valvular pulmonary stenosis must be entertained.

The most important point, as regards to physical examination, is absence or presence of cyanosis. If cyanosis is present, then a right-to-left shunt exists. But since a right-to-left shunt can exist in patients who have diminished pulmonary artery pressure, and in those in whom it is elevated, other physical signs of diminished or increased pulmonary pressure and flow must be looked for. Thus, a marked accentuation of the second pulmonic sound points toward pulmonary hypertension. The location and character of the murmur, although not as important as in acquired heart disease, may give valuable information. If a systolic murmur is present in a non-cyanotic child, high in the second intercostal space to the left of the sternum, a valvular pulmonic stenosis may be the underlying malformation. Absence of a murmur in a severely cyanosed child suggests pulmonary atresia.

It may be seen from these isolated examples, that a good deal of information may be gained from conventional methods. Add to this the information gained from electrocardiography, and particularly from fluoroscopy, and it is little wonder that one is often able to arrive at a correct diagnosis by conventional methods alone.

Why, we must ask ourselves, do we then in many cases resort to the difficult procedures of catheterization and angiocardiography? Is it that in complicated malformations, clinical examination is not sufficient? This we believe not to be correct. It has many times been our experience that the diagnosis of complicated malformations has been made more difficult by catheterization and angiocardiography. Exact location of the catheter tip in the heart, is often debatable in these patients, or the contrast material may have entered a portion of the heart which cannot be accurately defined. On the other hand, special tests are often essential in diagnosing the more common malformations of the heart, particularly if there is overlapping of signs and symptoms. This is particularly the case in the non-cyanotic group. Frequently the differential diagnosis between auricular, ventricular septal defect, or patent ductus arteriosus is difficult without catheterization, particularly in the absence of typical murmurs. In the cyanotic group it may be difficult to differentiate a tetralogy of Fallot with large collateral circulation from an Eisenmenger's Complex or true truncus.

I would like to say a few words about the problem of pulmonic stenosis. Anatomically, it is not an isolated malformation, rather the type of associated malformation that influences the physiologic pattern, and therefore the surgical correction of the stenosis.

When pulmonary stenosis is associated with a ventricular septal defect, the stenosis is usually of the infundibular type. In the presence of an auricular defect, the stenosis is usually at the pulmonic valve. If there stenosis is usually at the pulmonic valve too. When is neither auricular nor ventricular septal defect, the there is overriding of the aorta, such as in the tetralogy of Fallot, the infundibular type is frequently found in the tetralogy of Fallot.

In infundibular stenosis, the subpulmonary tract is bounded by the right ventricular muscle, by the ventricular septum and by a muscle which separates the tract from the ventricular septal defect. This latter muscle is the crista supraventricularis. This subpulmonary tract may at times be very narrow and it is believed by some, including Blalock, that this subpulmonary tract is the primary source of obstruction in the infundibular type. Brock, on the other hand, while admitting that the infundibular tract is often hypoplastic, maintains that as a rule an effective lumen is adequate, and that the actual stenosis occurs at one level and is linear and diaphragmatic.

This difference in interpretation is of considerable surgical treatment. If the subpulmonary tract has an effective lumen and if the stenosis is linear, infundibulotomy may be practical; if the stenosis is a long narrow channel, this procedure is hardly adequate, and even infundibular resection may be difficult.

We examined this question by means of drawings and wax casts, which were prepared by pouring parafin into the cardiac chambers and thus obtaining a mold of the cardiac cavities. By means of these casts and drawings, we could show the reverted funnel-shaped entrance into the subpulmonary tract of the right ventricle, and we were also able to show that in addition to the narrowing of this tract resulting from hypertrophy of the muscle of its posterior wall producing "muscular stenosis;" there is also a secondary stenosis or "fibrous stenosis" which is linear and diaphragmatic and which is composed of fibrous rather than muscular tissue. It is our opinion that the fibrous stenosis results in very little additional narrowing of the internal diameter of the subpulmonary tract. Therefore, infundibular resection is the only direct surgical approach; whether this is able to overcome the obstacles, by removing a mass of muscular tissue of the subpulmonary tract, is still a very controversial question.

In valvular pulmonic stenosis, the morbid anatomy is very different, as has been shown by Edwards et al. Although there is often muscular hypertrophy, this does not result in narrowing of the right ventricular outflow tract. It is not surprising, therefore, that valvolotomy is the operation of choice in this malformation.

The physiopathology of these malformations have been described in great detail, and Dr. Dexter has mentioned most of the essential features. I want to mention only the physiologic and surgical importance of the degree of overriding of the aorta, in the surgical correction of pulmonic stenosis. It has been shown that most patients in whom the aorta overrides more than 50 per cent, died, during or shortly after construction of an artificial ductus, from acute pulmonary edema or severe pulmonary congestion. On the other hand, if there is no overriding, the construction of an artificial ductus results in right sided failure. The reason for this is that when there is marked overriding, the increased volume of blood reaching the left ventricle from the lungs, often this construction of the ductus has no escape. In the presence of pulmonic stenosis and an auricular defect, where there is no overriding, the construction of an artificial ductus leads to right side failure because increased return of blood into the left auricle will raise the left auricular pressure, and by tending to equalize the gradients between right and left auricle, diminish the right-to-left shunt. The subsequent increase in right auricular pressure leads to failure. Special tests have contributed very little to the recognition of the degree of overriding of the aorta. However, in Sweden, the use of regional angiocardiography has shown the way.

Physiologic tests have been particularly useful in the interpretation of operative procedures for pulmonic stenosis. In the tetralogy of Fallot, the construction of an artificial ductus produces a postoperative rise in the oxygen content of right ventricular blood. This may be due to increased inter-cardiac left to right shunting. This increased left to right shunt may also be responsible for the elevation of the right ventricular pressure which was observed in many patients.

In the treatment of isolated pulmonic stenosis with auricular defect, a condition in which valvolotomy is the method of choice, there is a fall in right ventricular pressure, which, however, never returns to normal; the pulmonary blood flow increases and the peripheral oxygen saturation rises.

Physiologic data obtained before and after infundibular resection in the tetralogy of Fallot are still scarce. However, Bailey and Glover used the infundibular resection in forty-two patients with the tetralogy of Fallot; of these twenty-two had infundibular stenosis. In patients who were catheterized postoperatively, the gradient between the systolic pressure in the right ventricle and the pulmonary artery was still very marked; the clinical results on the other hand, were far more satisfactory than one might expect from these physiologic data. It is hoped that future physiologic studies will justify some of the optimism evoked by these clinical observations.

Dr. H. B. Burchell, Rochester, Minn.: In respect to the general principles outlined by Dr. Dexter pertaining to cardiac catheterization and its diagnostic application, I am in agreement, although there are a number of small points about which I have somewhat different opinions. I can re-emphasize the difficulty in the differentiation of atrial septal defect and anomalous pulmonary venous connection (drainage) into the right atrium. Actually, no true differentiation can be made when the posterior segment of the atrial septum is missing.

I should like to reiterate the diagnostic value of exercise, not only for patients who have ventricular septal defect, but also for persons who have pulmonary stenosis. The obstructive nature of pulmonary stenosis, with an intact ventricular septum is often dramatically portrayed after exercise, when the right ventricular pressure may be increased much further while the cardiac output is insignificantly increased. Like Dr. Dexter, I consider that similarity of right ventricular systolic and systemic systolic pressures, particularly when this pressure relationship persists with exercise, is a strong indication of a large ventricular septal defect. It is important to remember, however, that the actual values need not be identical; systolic pressures in the radial artery that are 10 to 25 mm. of mercury in excess of the right ventricular systolic pressure can be regarded as "physiologically" identical. While in general it is considered that the right ventricular systolic pressure is correlated with the size of the ventricular septal defect, exact correlations in this regard are not available.

Dr. Dexter mentioned that breathing 100 per cent oxygen would differentiate cyanosis due to an intracardiac shunt from that due to pulmonary disease. It is important to recognize, that exceptions to this conclusion occur in that a right-to-left shunt, that is present when the patient is breathing air, may disappear when the patient breathes 100 per cent oxygen. This is thought to be related to a decrease in pulmonary resistance. A number of such instances have been encountered in which right-to-left shunts are demonstrable by dye techniques when the patient breathes air but which disappear when 100 per cent oxygen is used. In such cases the arterial oxygen saturation may be 100 per cent and the blood may carry its normal complement of dissolved oxygen. Previous papers in this symposium have pointed out the potential errors in the determination of pulmonary flow using the Fick principle; Dr. Dexter has placed proper emphasis on this problem. This is something that must be remembered when we calculate pulmonary resistance with high pulmonary flows, and we must avoid drawing too definite conclusions regarding the change in resistance following the cure of large left-to-right shunts.

Any physician, intimately concerned with the diagnostic problems presented by patients who have congenital heart disease, probably will admit two things: (1) his clinical diagnostic ability has been increased many fold in the past ten years as the result of correlation of clinical findings and data derived from cardiac catheterization, and (2) in an occasional baffling case, the results of cardiac catheterization may be diagnostically inconclusive or even misleading.

One principle needs constant repetition and reemphasis. In the diagnostic approach all the facts in the history and examination should be carefully sifted and weighed; any single laboratory finding that superficially appears to be definitely diagnostic should be studied with care if it does not appear to fit with the other features of the case. As an example, the finding of arterialization of blood in the left atrium might be due to many causes, including: (1) sampling error, including difficulties related to differences in the saturation of the three main veins emptying into the right atrium, namely, the superior and inferior vena cava and the coronary sinus; (2) anomalous pulmonary venous connections; (3) ventricular septal defect, with the right-sided opening above or through the tricuspid valve, this shold be suspected after the occurrence of subacute bacterial endocarditis in patients who have ventricular septal defect; (4) rupture of an aneurysm of the sinus of Valsalva into the right atrium, and (5) persistent atrioventricular canal with a "low" atrial septal defect as well as the atrial septal defect itself.

Two procedures that have been introduced into the technique of cardiac catheterization and brought to a state approaching technical perfection by Dr. Wood and his associates, have added a great deal to the diagnostic accuracy of cardiac catheterization.

The first is the use of the oximetric curvette to give nearly instantaneous readings of oxygen saturation of the blood at the tip of the catheter. This allows many determinations to be made; in addition, continuous sampling during withdrawal of the catheter from one part to another permits small differences in the oxygen saturation between such parts to be validated.

The second procedure is injection of dye into various parts of the central cardiovascular structures. This has had the rewarding results of fairly reliable localization and quantitation of venous arterial shunts.

Certain concepts presently held concerning four of the most common congenital malformations may be of some interest:

A. *Atrial septal defect.*
   1. Preferential drainage of the right lung to the right atrium exists in the majority of cases.
   2. Slight modifications of bilateral shunting are demonstrable by postural and respiratory maneuvers.
   3. Preferential drainage of the inferior vena cava toward the left atrium often is demonstrable.
   4. Functional openings of the foramen ovale with right-to-left shunting may occur after pulmonary embolism or pulmonary disease.
   5. Easy catheterization of the left ventricle through an atrial septal defect may be suggestive of the partial form of a common atrioventricular canal (auriculoventricular commune).
   6. In anomalous pulmonary venous connections, one should also suspect the presence of an atrial septal defect.

B. *Ventricular septal defect.*
   1. Many "transitional" conditions exist between uncomplicated ventricular septal defect and cyanotic Eisenmenger's complex.
   2. Severe pulmonary hypertension may coexist with a left-to-right shunt (pulmonary blood flow greater than the systemic) without hypoxemia.
   3. Cardiac failure in early infancy is not infrequent and may be associated with clinical evidence of left ventricular enlargement.
   4. Diastolic shunting of blood may be an important avenue of investigation.

C. *Patent ductus arteriosus.*
   1. The time of appearance of the continuous murmur is variable.
   2. Evidence of associated defects, particularly coarctation of the aorta and ventricular septal defect, is to be searched for.
   3. A patent ductus arteriosus is to be considered as the possible cause of cardiac failure appearing in early infancy.
   4. The disease in adult patients who do not have continuous murmurs and "reversed" shunts may be of two types: (a). An unchanged pattern since infancy, with no evidence of previous left-to-right shunt and no cardiac enlargement. (b). Acquired pulmonary hypertension and true reversal of shunt, which often may be exaggerated by breathing mixtures that are low in oxygen.

D. *Pulmonary stenosis.*
   1. Many difficulties in the evaluation of the subpulmonic tract remain, even though the problem has been approached through cardiac catheterization, blind surgical exploration and anatomic dissection.
   2. Extremely large hearts, as manifested by their roentgenologic silhouettes, may be present in infants who have isolated pulmonary stenosis.
   3. Surgical exploration sometimes deserves precedence over cardiac catheterization.
   4. Left-to-right shunts (pulmonary blood flow greater than the systemic) at either atrial or ventricular levels may coexist with severe pulmonary stenosis.
   5. Mild pulmonary stenosis is a definite entity, although its anatomic basis is obscure.

Time does not allow discussion of the difficult diagnostic problems often presented by the newborn or young cyanotic infant or the difficulties in the interpretation of murmurs, electrocardiographic records or roentgenologic findings. This often is best done by presentation of cases. This symposium has, I think, been properly oriented toward the discussion of broad concepts and of accurate measurment of hemodynamic variables.

In closing, it is proper to pay tribute to Dr. Cournand, who demonstrated the relative safety and value of cardiac catheterization in man, but to emphasize that this procedure does present some risk to the patient, the mortality rate probably approximating one death in 500.

Dr. Paul Adams, Minneapolis, Minn.: At the University of Minnesota, we have been particularly interested in what we consider the most challenging and difficult problem in the differential diagnosis of congenital heart disease, that is, infants under two years of age.

Challenging, because next to prematurity, it has been the largest single cause of infant mortality in Minnesota. Review of our autopsy material reveals that a certain number of these patients had defects which could have been corrected or improved by present surgical techniques. An ever-increasing variety of malformations may soon be similarly considered.

We consider it difficult because, in our experience, symptoms and findings in the infant differ markedly from the classical descriptions when malformation is severe. We are gradually becoming aware of findings, which are fairly typical of each individual malformation in the infant. We have often been asked, and indeed occasionally ask ourselves, just how much is accomplished by these special procedures in the infant, compared to the added risks involved? With that question in mind, our recent experience with heart catheterization in infants will be briefly outlined.

Right heart catheterization has been performed in fifty-three infants under two years of age—about 40 per cent of the total number of pediatric catheterizations during 1952-1953. Angiocardiography and retrograde aortography were done on seventy-four infants during the same period. These special procedures were undertaken only when the infants were considered to be severely handicapped, when the risk of death during infancy appeared to be greater than the risk of special procedures, and when the specific type of malformation could not be diagnosed by routine methods. The smallest patient weighed only 3 kilograms at four months of age. Of the total of 127 special procedures (heart catheterization, angiocardiography and retrograde aortography, one death occurred during heart catheterization. This death was probably related to the medications used rather than the procedure, *per se*.

The final diagnoses of the fifty-three patients who had heart catheterization studies are listed as follows:†

1. Patent ductus arteriosus	6
2. Coarctation of the aorta	3
3. Interatrial septal defect	3
4. Interatrial plus interventricular or A-V canal	4
5. Anomalous pulmonary venous drainage into the right atrium	5
6. Interventricular septal defect or Eisenmenger's	13
7. Tetralogy of Fallot	7
8. Transposition of the great vessels	5
9. No left-to-right shunt	
(a) aortic stenosis	4
(b) endocardial fibrosis	3

Patent ductus arteriosus is the malformation in which the greatest salvage has been obtained. All patients had been severely ill with respiratory infection, and/or growth retardation, and their survival had been considered doubtful. None of these infants had the typical continuous murmur. The location and character of the systolic murmur only occasionally was suggestive of the patent ductus ateriosus. Surgical closure of a very large ductus was successfully accomplished in every case with marked improvement. One diagnostic error occurred in which the oxygen saturations of the pulmonary artery were increased over the remaining venous samples. However, at surgery and at post mortem a high interventricular defect alone was found. This occurred before we were using retrograde aortography. Since that time we have used that method successfully in three additional cases.

Three out of a total of ten infants, who were operated on for coarctation of the aorta, were considered to need heart catheterization studies prior to surgery because of the suspicion of serious intracardiac defects. Although surgery was not denied any of these patients, a more guarded prognosis could be given.

The "flush" method for determination of blood pressure has been most helpful in infants. Occasionally, when the infant is crying or struggling during the examination, an unusually high blood pressure has been found in the arms in normal infants but normal pressures in the leg when done subsequently. To avoid this possibility of error in young infants we have simply joined two infants' cuffs together with a Y tube connector and taken pressures of the arm and leg simultaneously by the "flush" method. In the normal infant without coarctation, the leg flushes slightly before or simultaneously with the arm. In coarctation the flushing of the leg is delayed.

Transposition of the great vessels and tetralogy of Fallot can best be diagnosed by angiocradiography.

Although seven patients had no demonstrable left to right shunts, we feel that they were valuable in localizing the lesion to the left side of the heart. Two patients thought to have endocardial fibrosis clinically before catheterization, were shown to have patent ductus arteriosus as a result of catheterization.

The results of our diagnostic efforts have in many instances been encouraging, especially in those infants where excellent surgical correction has been accomplished. At times we have been disappointed, but overall, we feel that these sick infants deserve every effort of diagnosis that we can make.

We have felt that valvular pulmonary stenosis could be easily distinguished from infundibular stenosis by the characteristic sharp change in pressure at the valve in the valvular type, and the slower transitional change in the infundibular type. On two occasions, however, this sharp transition occurred and at thoracotomy they were found to have an infuidibular type. Both of these patients showed a small left to right shunt in the right ventricle in addition.

I should like to ask (a) whether this experience has occurred elsewhere? (b) if so, what explanation can be offered as to why it occurs, and (c) is there a way that this error may be avoided in the future?

Dr. Dexter mentioned the use of certain drugs and exercise as a differential aid in cases of suspected pulmonary hypertention, as opposed to the aorta arising from the right ventricle.

We have had one death in a patient with pulmonary hypertension following fluorescein circulation time, and recently the *New England Journal of Medicine* has reported such a case with mortality following a decholin circulation time. I would like to ask Dr. Dexter if he feels that the use of drugs or exercise in such a situation might be considered to add more risk to the procedure of heart catheterization than they care to undertake.

Dr. Dexter stated, if I understood him correctly, that in the absence of a significant shunt as shown by oxygen saturations during a satisfactory heart catheterization, any defect that existed could be safely considered as clinically insignificant.

An exception to this, I believe, might occur in a high interventricular septal defect with pulmonary hypertension, at a certain point where the right ventricular hypertension was balanced with the left so that no left to right or right to left shunt occurred, and yet the defect obviously would be clinically significant.

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†It should be stressed, as Dr. Dexter stated, that heart catheterization was only one factor in the diagnosis. The patient's history, physical examination, x-ray, angiocardiogram and retrogradeaortogram were occasionally necessary. In some instances, the correct diagnosis was learned only during postmortem examination.

DIAGNOSTIC APPLICATIONS OF INDICATOR DILUTION CURVES IN HEART DISEASE

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AN INDICATOR dilution curve shows the changing concentration of an indicator at a specific point in the vascular system at various times following its injection at some different point in the vascular system. Usually dilution curves of the indicator are obtained from the arterial system following rapid injection of the indicator into the venous system proximal to the pulmonary capillaries. An indicator substance should possess the following qualities: (1) The substance must be nontoxic and sterilizable in regard to pathogenic organisms. (2) The concentration of indicator should remain relatively constant for at least a short time and preferably should allow for storage over several days. Otherwise a supply of fresh indicator must be readily available. (3) Injection of the substance must be possible in a brief time and this injection must not cause cardiovascular responses. Thus the volume in which the indicator is contained must be small. (4) The indicator must not pass out of the vascular system in appreciable amounts for several minutes. (5) It must be possible to measure the concentration of indicator in blood or plasma with considerable precision.

The indicator in most common use is Evans blue dye (T-1824),[2,4,8] but saline solution was used by Stewart, and brilliant vital red and other dye substances by Hamilton and associates,[3] in their fundamental studies on the problem of dilution of an indicator by the circulation. Hamilton and associates[3,4] used a discontinuous method of sampling in which multiple small samples of blood were collected, the concentration of T-1824 in plasma was determined photometrically, and a concentration-time curve was constructed. From such curves it is possible to measure circulation times and to estimate cardiac output and central blood volume. But when the information to be derived from a dilution curve depends to a major extent on the contour of that curve then use of a continuous recording instrument would appear to be essential. The oximeter[13] and densitometer[6] are instruments which permit the continuous recording of the concentration of T-1824 in whole blood. However, fluctuations in the oxygen saturation of arterial blood which frequently occur in patients with cyanotic congenital heart disease will distort dilution curves of T-1824 recorded by these instruments. This serious disadvantage which is inherent in the oximetric technics renders certain radioactive isotopes attractive as potential indicators when problems pertaining to dose and recording devices are satisfactorily solved.

The observations to be discussed in this communication are based entirely on dilution curves of T-1824 recorded by means of the oximeter.

The pattern of dilution in a normal subject following injection of T-1824 into a peripheral vein is shown in Figure 1. The oximeter responds to decreasing transmission of light and hence to increasing concentration of dye by a downward deflection of the recording beam. A short interval, the appearance time, elapses between the injection of the dye and its first arrival at the recording instrument. Then there is a rapid increase in concentration to a peak, followed by a less rapid decline in concentration which is incomplete and merges into a second peak of concentration which is caused by return to the sampling site of dye which has passed the systemic capillaries.

After injection into a peripheral vein the following average normal values in seconds have been obtained: appearance time 14 (10 to 20); maximal concentration time 25 (16 to 35); buildup time 10 (7 to 15); disappearance time 16 (9 to 26) and recirculation time 21 (16 to 28).*

The most important physiologic factors modifying the dilution pattern are: the rate of flow through the system, the effective mixing volume of blood in the central vascular pool, the variation in volume and rates of blood flow in the blood vessels in the lungs, and the characteristics of flow in the aorta and blood vessels leading to the sampling site. As a rough generalization, an in-

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*Approximated values from Nicholson, Burchell and Wood.

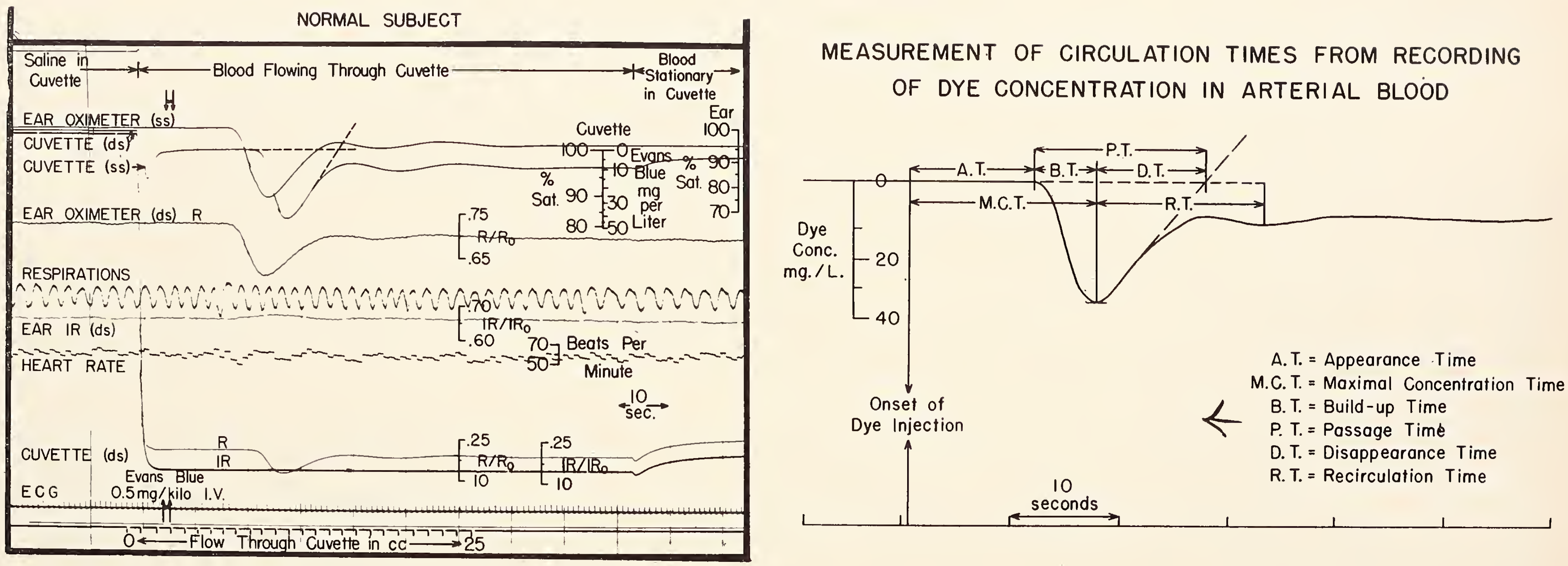

Fig. 1. Arterial dilution curves of Evans blue dye (T-1824) in a normal subject.

Left panel. A section of the photographic record, which is untouched apart from addition of captions. Curves were obtained from an oximeter on a heat-flushed ear, and from a cuvette oximeter through which blood from the radial artery was flowing. This panel shows recordings of the respiration rate, the heart rate, electrocardiogram, and rate of blood flow from the radial artery through the cuvette oximeter; also the change in transmission of red (R) and infrared (IR) light recorded by the oximeter (ds).

Right panel. The time components of the dilution curve are indicated.

crease in the volume of blood between the site of injection and the site of sampling causes a reduction in the concentration of dye, whereas a decrease in the rate of flow through the system results in prolongation of the time components. Therefore, in acquired heart disease, the changes in the dilution pattern found are those associated with reduced cardiac output and perhaps an increased volume of blood in the heart and great vessels.

In those forms of congenital heart disease characterized by an alteration in the circulatory pathway, the contour of the dilution curve may permit interpretations which have frequently been found to be of diagnostic value.

Conditions characterized by right-to-left or left-to-right shunts usually give rise to typical dilution patterns.[7] A dilution curve, following injection of dye into a peripheral vein of a patient with tetralogy of Fallot, is shown (Fig. 2, *left panel*). A shortened appearance time and an abnormal (first) hump on the buildup slope of the main curve are apparent. This pattern of dilution may be explained by the passage of a portion of the dye across the ventricular septal defect directly into the systemic circulation. The appearance time is reduced because this shunted dye reaches the recording instrument before the dye which traverses the longer, normal circulatory path through the lungs. As each portion of dye is subjected to dilution in its passage through the heart and the blood vessels, its arrival in the peripheral circulation takes the form of two curves which partially overlap. In many patients with right-to-left shunts the cardiac output may be normal, but because some dye passes through the defect, the maximal concentration is reduced from that in the blood of the normal subjects. Early in our experiences with dilution curves obtained from patients with intracardiac shunts, it became apparent that the contour of the dye curve is determined largely by the volume of shunt passing through the defect. Thus, in patients with severe cyanosis, the initial (abnormal) deflection was found to be as large or larger than the normal component of the curve, while a very small initial break was found in patients with very small right-to-left shunts producing only a minimal depression of arterial oxygen saturation. If certain assumptions are made, the magnitude of the shunt can be calculated from such curves,[12] and satisfactory agreement has been shown between shunt values calculated from the dilution curve and data obtained from cardiac catheterization. There was a standard deviation between the methods of ± 7 per cent of systemic blood flow in shunts ranging from 51 to 8 per cent of the systemic flow.

In left-to-right shunts also the dilution of indicator is altered in a specific manner[7] (Fig. 2, *right panel*). The appearance time and the buildup time are normal. However, the magnitude of the deflection is reduced, and the slope of declining concentration of indicator is much prolonged. Also, no peak due to systemic recirculation can be identified. This curve, which is typical of left-to-right shunt of considerable magnitude, is explained on the basis of recirculation of dye in the heart and vessels of the lungs with a slow clearance of a constant proportion of the recirculating dye into the systemic arterial system. This slow elimination of dye results in the prolonged disappearance time which is the most notable feature of the curves. Since, in normal subjects, sharpness of individual dilution curves varies considerably, yet the curve remains consistent with regard to internal time relations, the ratio of disappearance time to buildup time of abnormal curves provides a sensitive index of disproportionate prolongation of the disappearance time. Broadbent recorded dilution curves following peripheral injections of dye into sixteen patients with left-to-right shunts and compared the ratio of disappearance time (DT) to buildup time (BT) with the magnitude of shunt calculated on the basis of values of oxygen saturation obtained at cardiac catheterization. An approximate relation based on these data indicated that the arteriovenous shunt expressed as per cent of the pulmonary artery flow equals $10 + 10$ (DT/BT).

These dilution patterns then are typical of right-to-left and left-to-right shunts, respectively. They are dependent not only on the existence of a defect but more fundamentally on direction and volume of blood flow across it. Abnormal dilution patterns will not be seen when the pressures on either side of a defect equal one another at all times, because in this instance no shunting of blood will occur.

Since the altered dilution pattern in right-to-left and left-to-right shunts depends on the presence of an abnormal circulatory path, it appeared

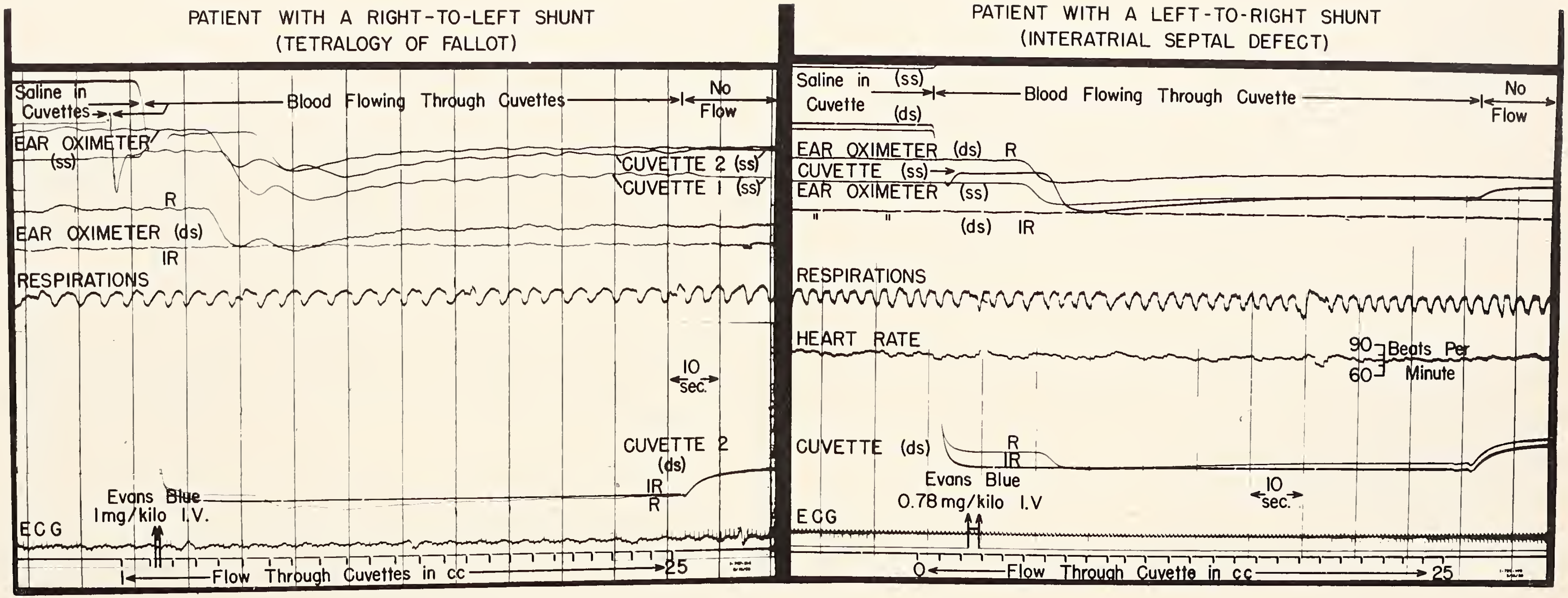

Fig. 2. Recording of dye dilution curves obtained after peripheral injection of T-1824 into a patient with tetralogy of Fallot *(left panel)* and in a patient with an atrial septal defect *(right panel).*

Left panel. The dilution curves were recorded by an oximeter on the heat-flushed ear (ear oximeter ss) and by two cuvette oximeters in tandem (cuvette 1 ss and cuvette 2 ss) through which blood from the radial artery was flowing. The short appearance time (8.5 seconds for the ear oximeter) and the double-hump pattern seen on all curves should be noted. The normal peak due to recirculated indicator is absent. The changes in both cuvette curves were delayed due to the time necessary for dye to pass from the radial artery to the photosensitive portion of the cuvette oximeters. Small fluctuations may be noted in the recording line due to variations in oxygen saturation coincident with respiration. This form of dilution curve is typical of right-to-left shunt.

Right panel. The dilution curves from a patient with an atrial septal defect recorded by means of an earpiece oximeter (ear oximeter ss) and by a cuvette oximeter (cuvette ss) through which blood from the radial artery was flowing. The normal appearance time (11 seconds for the earpiece), the reduced magnitude of the initial deflection and the marked prolongation of the disappearance slope of the dilution curve should be noted. This type of curve is typical of left-to-right shunts.

possible to obtain additional information from dilution curves recorded after injection of dye at several different sites in the heart and great vessels of the same individual.[11] In normal subjects, and in patients without intracardiac or extracardiac shunts, the dilution pattern following injection of dye into the pulmonary trunk differed somewhat from that following injection at a more peripheral site. After peripheral injection the concentration components were reduced, and the time components were prolonged. This phenomenon was explained on the basis of greater longitudinal mixing and dilution after injection into the more peripheral vessels.[5] The curves were, however, similar to one another in general contour. In cases with right-to-left shunts it became possible to localize the site of a defect with relative certainty (Fig. 3). If an injection of dye is made distal to the site of a defect then a relatively normal pattern of dilution is obtained, because dye traverses a fundamentally normal circulatory pathway. But if the injection is made at or proximal to the defect then some of the dye will be shunted through the defect while the remainder traverses the pulmonary vascular bed, thus producing the abnormal "double-hump" pattern. By this technique right-to-left shunts occurring through a patent ductus arteriosus, ventricular and atrial septal defects have been localized and, in many instances, identification would not have been made otherwise. It has been possible to recognize small right-to-left shunts, particularly those occurring at the atrial level, which caused an insignificant desaturation of the systemic arterial blood.

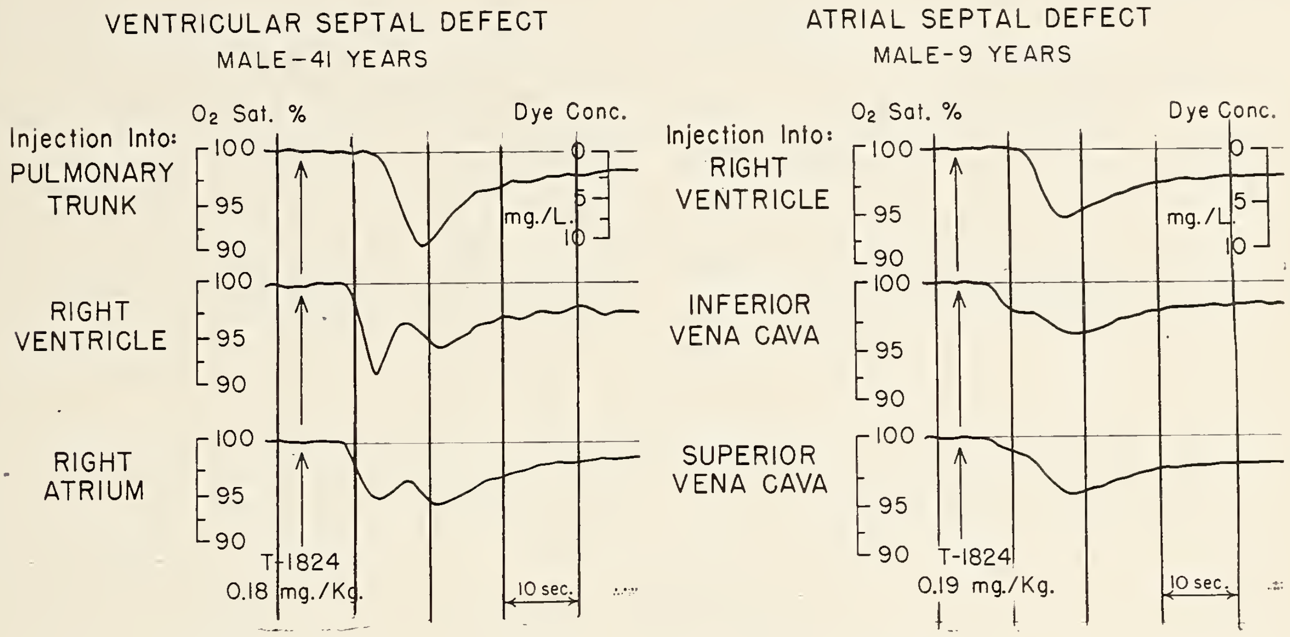

Fig. 3. Localization of the site of a right-to-left shunt in a patient with a ventricular septal defect, and in one with an atrial septal defect. In each case the curves have been superimposed in regard to the time of dye injection. The oxygen saturation scales serve to indicate the relative sensitivity of the oximeters.

Left panel. Dilution curves from a patient with Eisenmenger's complex recorded after injection of T-1824 into the pulmonary trunk, right ventricle and right atrium. Fluctuations in oxygen saturation may be noted in all curves. The appearance time after injection of dye into the pulmonary trunk is normal for this site (9.0 seconds) and the remainder of the curve is not grossly distorted. The curves after injection of dye into the right ventricle or the right atrium differ from the curve after injection in the pulmonary trunk in that the appearance time is shorter (5.5 seconds) and there is a well-defined double-hump pattern. This series of curves localizes the site of the right-to-left shunt and, hence, of the intracardiac defect to the ventricle.

Right panel. Curves obtained after injection of dye into the right ventricle, inferior vena cava and superior vena cava in a patient with atrial septal defect. The curve after injection of dye into the right ventricle has a normal appearance time (8 seconds) and build-up slope, but the disappearance time is prolonged. This prolongation of disappearance time indicates the presence of a left-to-right shunt. In the curves obtained after injection of dye into both inferior and superior venae cavae, the appearance time is reduced (4 seconds) and a double-hump pattern is apparent. In this instance, the defect can be localized to the atrial septum. The right-to-left shunt from the inferior vena cava is indicated by the curve being of greater relative magnitude than that from the superior vena cava.

In cases with left-to-right shunt recirculation of dye occurs after the dye has passed the pulmonary capillaries, hence specific differences between injections into the pulmonary trunk and more peripheral sites would not be expected. In a relatively limited experience with the central injection technic in patent ductus arteriosus and ventricular septal defect no remarkable distinguishing features have been apparent. However, in both atrial septal defect and anomalous pulmonary venous connection, striking differences in the dilution pattern have been obtained after injections into right and left pulmonary arteries, and after injections into either right or left pulmonary artery and pulmonary trunk.[10] Findings typical of atrial septal defect are shown in Figure 4. The curve from injection into the right pulmonary artery differs from that following injection into the left pulmonary artery in that the magnitude of the deflection is smaller and there is a well-marked second hump about six seconds after the first. These differences in deflections are due to a smaller amount of dye passing from the right lung into the left ventricle and systemic circulation. The remainder of the dye is shunted into the right atrium and passes again through the pulmonary circulation. The second peak is produced when a portion of this shunted dye passes out (mostly by way of the left pulmonary artery) to the systemic arterial circulation. The interval between the two peaks of concentration represents the average time which the dye takes to circulate once through the lungs. The finding of this difference between dilution curves from the right and left pulmonary artery demonstrates that in many cases of atrial septal defect a greater proportion of blood draining from the right lung is shunted in a left-to-right direction than is that from the left. This phenomenon probably is due to the proximity of the openings of the right pulmonary veins to the defect in the atrial septum.

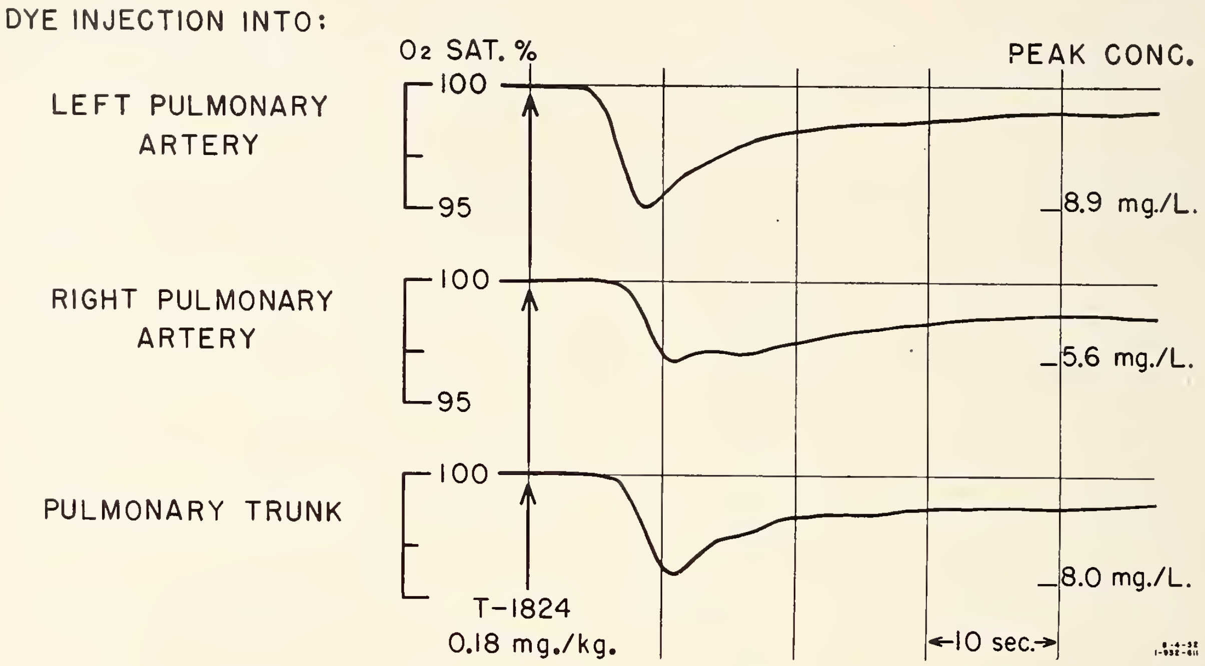

Fig. 4. Dye dilution curves obtained after injection of T-1824 into the left pulmonary artery, right pulmonary artery and pulmonary trunk of a sixteen-year-old girl with atrial septal defect. Injection of dye into the left pulmonary artery resulted in a dilution curve with a normal appearance time and build-up slope, but with a prolonged disappearance time, indicative of a moderate degree of pulmonary recirculation. After injection of dye into the right pulmonary artery, however, the magnitude of the initial deflection is reduced, and there is a well-developed second hump on the disappearance slope. The curve following injection into the pulmonary trunk is a composite of both right and left pulmonary arteries (for discussion see text).

In partial anomalous venous connection, the veins from one lung communicate directly with either the right atrium or one of its tributaries. This situation is associated with an anomalous drainage of the blood from that lung; hence, injections of dye into the right and left pulmonary arteries will serve to identify this condition in many instances. In the case of the anomalously draining right lung (Fig. 5) the appearance time is considerably prolonged, and the magnitude of the deflection is reduced in comparison with the curves from the normally draining left lung. If equal volumes and equal rates of blood flow through each lung are assumed, then the prolonged appearance time following injection into the right pulmonary artery is due to the greater distance between the sites of injection and of sampling. This distance includes the pathway through the pulmonary vascular bed and right side of the heart which the dye has to traverse to reach the normally draining left lung which is the only route through which dye passes to the systemic circulation. Division of this dye in the pulmonary trunk into portions traversing the right and left lung reduces the total amount of dye passing to the systemic circulation and hence diminishes the magnitude of the resulting deflection. Recirculation of the remaining dye through the anomalous pathway causes a prolonged clearance of dye from the central circulation and, therefore, an increased disappearance time. When an anomalous pulmonary venous connection into the right atrium coexists with a defect of the atrial septum, it may not be possible to demonstrate the presence of an anomalous connection with certainty.*

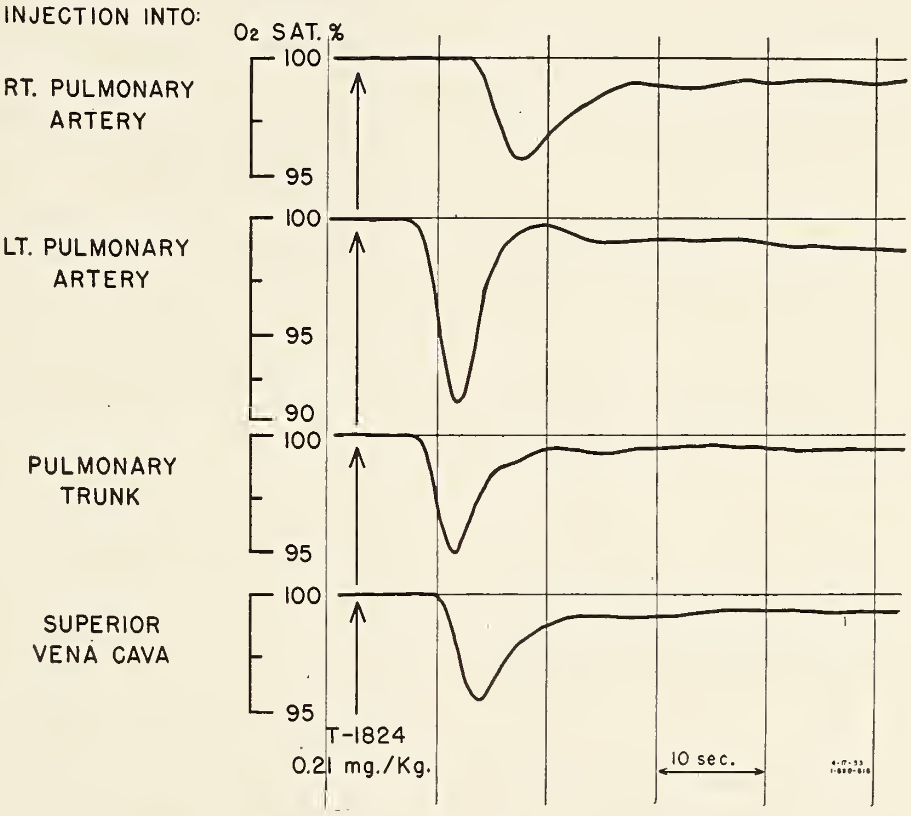

Fig. 5. Arterial dilution curves recorded following injection of T-1824 into right, left and main pulmonary arteries and superior vena cava of a forty-three-year-old woman with anomalous pulmonary venous connection and agenesis of the upper lobe of the right lung. The curve obtained after injection into the left pulmonary artery is normal (appearance time, 5 seconds). In contrast, curve obtained on injection of dye into the right pulmonary artery shows the appearance time is much prolonged (10.3 seconds), the magnitude of the deflection is reduced, and the disappearance slope is prolonged (for discussion see text).

Summary

Analysis of indicator-dilution curves based on differences in contour and time relationships offers a new tool in the study of heart disease. In particular the method permits a more detailed study of the dynamics in congenital heart disease and hence permits better appraisal of the anatomic defect present. In this presentation certain of the concepts basic to the technic have been outlined. In the individual case the physician at catheterization makes use of the methods as he proceeds with the study since careful selection of sites of injection on the basis of data being obtained during the procedure, will allow a more precise diagnosis to be made. In the cardiac catheterization laboratory of the Mayo Clinic this technic is an established integral part of the routine investigation of the majority of cardiac patients studied. In these days of the increasing possibility of successful surgical therapy for congenital heart disease the added accuracy of diagnosis provided by the technic has proved to be of practical value.

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*This problem was discussed in some detail in a recent publication.[10]

References

1. Broadbent, J. C.: A Study of Acyanotic Congenital Heart Disease Using Dye Injection and Cardiac Catheterization Technics. Thesis, Graduate School, University of Minnesota, 1953.
2. Friedlich, A.; Heimbecker, R., and Bing, R. J.: A device for continuous recording of concentration of Evans blue dye in whole blood and its application to determination of cardiac output. J. Applied Physiol., 3:12-20 (July) 1950.
3. Hamilton, W. F.; Moore, J. W.; Kinsman, J. M., and Spurling, R. G.: Studies on the circulation; IV. Further analysis of the injection method, and of changes in hemodynamics under physiological and pathological conditions. Am. J. Physiol., 99:534-551 (Feb.) 1932.
4. Hamilton, W. F.; Riley, R. L.; Attyah, A. M.; Cournand, Andre; Fowell, D. M.; Himmelstein, A.; Noble, R. P.; Remington, J. W.; Richards, D. W., Jr.; Wheeler, N. C., and Witham, A. C.: Comparison of the Fick and dye injection methods of measuring the cardiac output in man. Am. J. Physiol., 153:309-321 (May) 1948.
5. Hetzel, P. S., and Swan, H. J. C.: Comparison of arterial dilution curves following central and peripheral injections of dye. Federation Proc., 12:66 (Mar.) 1953.
6. Milnor, W. R.; Talbot, S. A.; McKeever, W. P.; Marye, R. B., and Newman, E. V.: A photoelectric ear densitometer for continuously recording the arterial concentration of T-1824 in the dye-dilution method. Circulation Research., 1:117-121 (Mar.) 1953.
7. Nicholson, J. W., III; Burchell, H. B., and Wood, E. H.: A method for the continuous recording of Evans blue dye curves in arterial blood, and its application to the diagnosis of cardiovascular abnormalities. J. Lab. & Clin. Med., 37:353-364 (Mar.) 1951.
8. Nicholson, J. W., III, and Wood, E. H.: Estimation of cardiac output and Evans blue space in man, using an oximeter. J. Lab. & Clin. Med., 38:588-603 (Oct.) 1951.
9. Stewart, G. N.: The output of the heart in dogs. Am. J. Physiol., 57:27-50 (Aug.) 1921.
10. Swan, H. J. C.; Burchell, H. B., and Wood, E. H.: Differential diagnosis at cardiac catheterization of anomalous pulmonary venous drainage related to atrial septal defects or abnormal venous connections. Proc. Staff Meet., Mayo Clin., 28:452-462 (Aug. 26) 1953.
11. Swan, H. J. C., and Wood, E. H.: Localization of cardiac defects by dye-dilution curves recorded after injection of T-1824 at multiple sites in the heart and great vessels during cardiac catheterization. Proc. Staff Meet., Mayo Clin., 28:95-100 (Feb. 25) 1953.
12. Swan, H. J. C.; Zapata-Diaz, J., and Wood, E. H.: Dye dilution curves in cyanotic congenital heart disease. Circulation, 8:70-81 (July) 1953.
13. Wood, E. H.: Oximetry. In Glasser, Otto: Medical Physics. Vol. 2, pp. 664-680. Chicago: The Year Book Publishers, Inc., 1950.

STROKE VOLUME CHANGES

(Continued from Page 115)

that of Dr. Remington in the assumption of a linear volume-pressure relationship, the slope of which is measured in each individual, while Dr. Remington accepts this individual variation by utilizing an average curve.

Since many assumptions and approximations are of necessity made in the derivation of the equations used, care must be taken to carefully control studies made with this technique by simultaneous determinations using independent methods. This is particularly true when used in conditions other than those already tested.

References

1. Peterson, L. H.; Dripps, R. D., and Risman, G. C.: Am. Heart J., 37: 771, 1949.
2. Remington, J. W.; Hamilton, W. F., and Dow, P.: Am. J. Physiol., 144:536, 1945.
3. Remington, J. W.; Noback, C. R.; Hamilton, W. F., and Gold, J. J.: Am. J. Physiol., 153:298, 1948.
4. Warner, H. R.; Swan, H. J. C.; Connolly, D. C.; Tompkins, R. G., and Wood, E. H.: J. Appl. Physiol., 5:495, 1953.
5. Wood, E. H.; Leuser, I. R., and Warner, H. R.: Am. J. Physiol., 171:780, 1952.
6. Wood, E. H.: Otto Glasser's Medical Physics, Vol. 2, pp. 664-680. Chicago: Year Book Publishers, Inc., 1950.

MINNESOTA MATERNAL MORTALITY STUDY

The Maternal Mortality Committee of the Committee on Maternal Health of the Minnesota State Medical Association

THIS REPORT is the third concerned with the statewide maternal mortality study being carried out in Minnesota. The present report deals principally with the results of the current study, which includes the period April 1, 1951, through March 31, 1952. Certain comparisons are made between these results and those of the preceding year and with the results of a similar study carried out in Minnesota from July 1, 1941 to June 30, 1942. (The three surveys will hereafter be referred to as the "1951 survey," the "1950 survey," and the "1941 survey," respectively.)

The study of maternal deaths is sponsored by the Minnesota State Medical Association and the Minnesota Department of Health with the latter organization providing the necessary funds. Each death is thoroughly studied in a personal visit by a trained obstetrician, and the data so obtained is then considered in a completely impartial manner by the Maternal Mortality Committee of the Maternal Health Committee of the State Medical Association. The exact method by which the study is carried out has been detailed in a previous report.[1]

It is gratifying to note that a further reduction in the maternal mortality rate occurred during the period covered by the present report (Table I). For the purposes of this study, all deaths associated with pregnancy and those occurring during a period of three months postpartum are included, regardless of cause of death. In many instances it has been clear that the pregnancy or the postpartum state had no direct relationship to the cause of death, but these deaths are nevertheless included in this study for completeness. This group of cases constitutes the so-called non-obstetric deaths. The remaining deaths (excluding those due to chorionepithelioma which are also included in the present report) are those from which state and national maternal mortality rates are ordinarily calculated. Deaths due to chorionepithelioma are not included as maternal deaths in state and national rates, but are customarily included in reports on cancer. In Table I are shown the all-inclusive maternal death rates and the "maternal death" or corrected rates for each of the three years referred to. It can be seen that the reduction in the maternal death rate in Minnesota has been progressive and that the rate is consistently well below that of the United States generally.

TABLE I. MATERNAL DEATHS IN MINNESOTA

Studies for 1941, 1950, and 1951, Respectively

	Number Deaths	Number Births	Gross Mortality Rate Per 1000 Live Births†	Minn. Maternal Mortality Rate Per 1000 Live Births‡	U. S. Maternal Mortality Rate Per 1000 Live Births
1941	112	55,293	2.03	1.68	2.8*
1950	68	76,074	0.89	0.59	0.83**
1951	57	80,099	0.71	0.45	0.71**

*Average for 1941 and 1942.
**Unofficial.
†Including all maternal deaths from whatever cause.
‡Excluding deaths considered as non-obstetrical on the basis of the definitions set down by the United States Bureau of the Census. This is the ordinarily reported maternal mortality rate.

The comparison would be even more favorable to Minnesota were it not for the fact that in the three years of the study, thirty-nine cases have been added to the totals for this state by special efforts at case finding. These cases have included eight "obstetric deaths" and have been discovered principally by cross-matching birth and death certificates and by reports of various individuals interested in the study. In all thirty-nine instances, there was no mention of pregnancy or the postpartum state on the death certificate. Since the number of maternal deaths is customarily arrived at from information appearing on death certificates, none of the thirty-nine would have been included in the determination of maternal death rates had it not been for the special efforts undertaken to discover them. It is the opinion of the Committee that very few if any maternal deaths in Minnesota escape recognition under present circumstances.

Many factors are probably involved in the continuing reduction of maternal mortality. These include increased use of whole blood, the availability of various chemotherapeutic and antibiotic agents, and extensive use of hospitals for obstetrical deliveries. In the final analysis, the chief

TABLE II. PREVENTABILITY OF MATERNAL DEATHS IN MINNESOTA
Studies for 1941, 1950, and 1951, Respectively

	1941		1950		1951	
	No.	Per-cent	No.	Per-cent	No.	Per-cent
Preventable	82	73.2	22	32.4	24	42.1
Not preventable	27	24.1	45	66.1	32	56.1
Not determinable	3	2.7	1	1.5	1	1.8

TABLE III. CONSULTATIONS IN RELATION TO MATERNAL MORTALITY IN MINNESOTA
Studies for 1941, 1950, and 1951, Respectively

	1941		1950		1951	
	No.	Per-cent	No.	Per-cent	No.	Per-cent
None	63	56.2	29	42.6	28	49.1
Adequate	10	9.0	22	32.4	17	29.8
Inadequate	39	34.8	17	25.0	12	21.1

TABLE IV. AUTOPSIES IN RELATION TO MATERNAL MORTALITY IN MINNESOTA
Studies for 1941, 1950, and 1951, Respectively

	1941		1950		1951	
	No.	Per-cent	No.	Per-cent	No.	Per-cent
Obtained, adequate	9	8.0	24	35.3	23	40.4
Obtained, inadequate	27	24.1	2	3.0	3	5.2
Not requested	55	49.1	29	42.6	20	35.1
Permission refused	21	18.8	13	19.1	11	19.3

TABLE V. OBSTETRIC AND NON-OBSTETRIC DEATHS AMONG MATERNAL MORTALITY CASES IN MINNESOTA
Studies for 1941, 1950, and 1951, Respectively

	1941		1950		1951	
	No.	Per-cent	No.	Per-cent	No.	Per-cent
Obstetric deaths	93	83.0	46	67.6	39	68.4
Non-obstetric deaths	19	17.0	22	32.4	18	31.6

credit is obviously due to the practitioners in Minnesota who are engaged in practicing obstetrics and who are ultimately responsible for careful obstetric work and for applying to actual practice the advances being made. The achievement of a mortality rate that was considered to be the irreducible minimum at the time of the original state survey in 1941 must mean that the great majority of Minnesota physicians are administering extremely efficient obstetric care. The material collected in the three years of the survey shows real evidence of improvement in this care.

Perhaps the most noteworthy evidence of improvement concerns preventability of death, as shown in Table II. In 1941 almost three-fourths of the deaths were considered preventable, while in 1950 and 1951 only about one-third and two-fifths, respectively, were preventable. In each year the physician involved was charged with the responsibility for the great majority of the preventable deaths.

That some improvement has taken place with regard to consultations is indicated in Table III. It will be noted that adequate consultation was obtained in less than 10 per cent of cases in 1941, but that this percentage was more than tripled in 1950 and again in 1951. There is room for further improvement here. Consultation was thought to be indicated in an additional 25 per cent of cases in 1951.

The data in Table IV indicate a continuing increase in the relative numbers of autopsies being done. Since trained personnel are frequently not available for the performance of an autopsy, the figure of 40 per cent for adequate postmortem studies in 1951 is considered good.

Another indication of improvement in obstetric care is the increase in the percentage of deaths associated with pregnancy that are due to non-obstetric causes, as shown in Table V. In the last two years of the survey almost one-third of all the deaths were due to non-obstetric causes as compared with less than one-fifth of the 1941 deaths.

While the evidence of improvement in care is most encouraging, much more improvement is still possible as indicated by the material obtained in the studies. It is not assumed that the material covered by the study is at all representative of the practice of obstetrics in the state as a whole. Since all the patients died, it is much more likely that the material so gathered contains a considerable concentration of evidence of bad obstetrics. At the same time it does indicate where improvements can be made.

Adequate care as defined by the Committee in its list of minimum requirements is being given to a pitifully small percentage of the patients who die (Table VI).[1] That these deficiencies are not mere technicalities is indicated by the frequency with which various deficiencies in care contributed to the patient's death. The encouraging evidences of improvement cannot be allowed to obscure the fact that approximately

TABLE VI. MEDICAL CARE GIVEN IN MATERNAL MORTALITY CASES IN MINNESOTA
1951 Study

	Prenatal Care		Care in Labor and/or Delivery		Postpartum Care	
	No.	Per-cent	No.	Per-cent	No.	Per-cent
Adequate	6	13.6	11	25.5	14	42.4
Faulty, contributory	10	22.7	23	53.5	15	45.5
Faulty, not contributory	28	63.6	9	21.0	4	12.1
None	13		14		24	

TABLE VII. PELVIC MEASUREMENTS IN MATERNAL MORTALITY CASES IN MINNESOTA
Studies for 1941, 1950, and 1951, Respectively

	1941		1950		1951	
	No.	Per-cent of Regis-tered Pa-tients	No.	Per-cent of Regis-tered Pa-tients	No.	Per-cent of Regis-tered Pa-tients
Unregistered	18		6		13	
Not measured or incompletely measured	77	81.9	40	64.5	32	72.7
Measured	17	18.1	22	35.5	12	27.3

TABLE VIII. SEROLOGIC TESTS FOR SYPHILIS IN MATERNAL MORTALITY CASES IN MINNESOTA
Studies for 1941, 1950, and 1951, Respectively

	1941		1950		1951	
	No.	Per-cent of Regis-tered Pa-tients	No.	Per-cent of Regis-tered Pa-tients	No.	Per-cent of Regis-tered Pa-tients
Unregistered	18		6		13	
Not obtained	64	68.1	38	61.1	25	56.8
Obtained	30	31.9	24	38.9	19	43.2

TABLE IX. TYPE OF DELIVERY IN MATERNAL MORTALITY CASES IN MINNESOTA
Studies for 1941, 1950, and 1951, Respectively

	1941		1950		1951	
	No.	Per-cent of Regis-tered Pa-tients	No.	Per-cent of Regis-tered Pa-tients	No.	Per-cent of Regis-tered Pa-tients
Undelivered	23		17		21	
Spontaneous	39	43.8	24	46.2	16	44.5
Operative	50	56.2	28	53.8	20	55.5

40 per cent of the deaths occurring are still preventable. The number and variety of faults in management comprise a long list. In the majority of instances the disastrous outcome was not the result of lack of some specialized knowledge or ability on the part of the physician, nor to lack of facilities, but rather to failure to follow principles of care familiar to all physicians.

The following examples taken from the 1951 survey may serve to illustrate this point. One patient died of a ruptured uterus resulting from attempted podalic version and extraction following a prolonged obstructed labor at term. The obstruction was due to the presence of a large sarcomatous mass in the pelvis which was missed because a pelvic examination was not done. No fewer than seven patients were delivered by accouchement forcé, a procedure which is probably never indicated. Six of the seven died from hemorrhage as a direct result of the procedure. Three patients died as a result of complete lack of attention to fluid and electrolyte balance. Two of these were suffering from hyperemesis gravidarum while the third died on the eighth day after delivery by cesarean section. Despite the fact that the patient vomited frequently, had a negligible oral intake of food and fluid, and was subjected to gastric aspiration for more than three days, she was given no sodium, potassium or chloride until just before death and never more than 1,000 cc. of fluid parenterally daily.

Pelvic measurements are still obtained in only one-fourth of the cases (Table VII), and serology during the prenatal course in considerably less than one-half (Table VIII). Surely the value of these procedures does not need re-emphasizing, yet the frequency with which they are done in the cases being studied is increasing only very slowly.

As indicated in Table IX, the relative incidence of operative deliveries is not changing significantly although the absolute incidence is. Approximately 40 per cent of the operative deliveries studied in the last two years of the survey were considered to be not indicated. More importantly, in 1951, 45 per cent were considered contributory to death.

Results of 1951 Survey

Table X contains the details regarding cause of death as determined by the Committee in the cases studied in 1951. Much of the information is self-explanatory but a few points require elaboration. It will be seen that hemorrhage was

TABLE X. PRIMARY CAUSES OF MATERNAL DEATHS IN MINNESOTA
1951 Study

Cause of Death		No.	Percent
Obstetric hemorrhage		13	22.8
Postpartum			
Lacerations of cervix and/or lower uterine segment with atony	9		
Intra partum	2		
Ruptured uterus	1		
Intra-abdominal hemorrhage (ectopic)	1		
Infection		6	10.5
Pulmonary embolism	4		
Septic abortion	1		
Generalized peritonitis of undetermined source	1		
Toxemia		4	7.0
Eclampsia superinposed on hypert. vasc. disease	3		
Eclampsia without pre-existing hypertension	1		
Chorionepithelioma		3	5.2
Anesthesia		3	5.2
Spinal	2		
Inhalation	1		
Dehydration and electrolyte imbalance		3	5.2
Hyperemesis gravidarum	2		
Post cesarean section	1		
Air embolism		2	3.5
Amniotic fluid embolism		1	1.9
Congenital heart disease with failure		1	1.9
Not determinable		3	5.2
Shock of unknown etiol. (? G.I. tract hem.)	1		
Sudden death late labor (? amn. fl. emb.)	1		
Threatened abortion—autopsy negative	1		
Non-obstetrical complications		18	31.6
Trauma	4		
Bulbar poliomyelitis	2		
Acute alcoholism	2		
Panmyelophthisis (chloramphenicol)	1		
Bilateral pneumothorax	1		
Probable rupture cerebral aneurysm	1		
Cerebral hemorrhage (hypersplenism)	1		
Pulmonary tuberculosis	1		
Malignant melanoma	1		
Carcinoma stomach	1		
Not determinable	3		
Totals		57	100.0

by far the leading cause of death. Almost one-half of the deaths due to hemorrhage were accounted for by the manual dilatations of the cervix previously referred to, none of which were indicated. Two of the remaining three patients who bled to death from lacerations of the birth canal had been given subcutaneous obstetrical pituitrin before delivery. One of the two cases of intrapartum hemorrhage involved a patient with a twin pregnancy who was sent back to bed after delivery of one twin. She died three and a half hours later of shock due to massive hemorrhage externally and into the uterus resulting from partial separation of the placenta. The second patient was delivered by an Indian midwife at home. The patient expired of hemorrhagic shock before the placenta was delivered.

The number of deaths due to infection continue to drop. As noted in Table X, pulmonary embolism accounted for two-thirds of the cases in this category.

TABLE XI. ANALYSIS OF MATERNAL DEATH CERTIFICATES IN MINNESOTA
1951 Study

	No.	Percent
Complete and correct	18	31.6
Complete but incorrect	8	14.0
Incomplete and incorrect	13	22.8
Incomplete, otherwise correct	18	31.6
Totals	57	100.0

TABLE XII. ANALYSIS OF BIRTH AND STILLBIRTH CERTIFICATES IN MINNESOTA
1951 Study

	No.	Percent	Percent of Those Required
Complete and correct	18	31.6	48.6
Complete but incorrect	14	24.6	37.8
Incomplete, otherwise correct	5	8.8	13.5
Not required	20	35.1	
Totals	57	100.1	99.9

A notable change between the 1950 and 1951 results concerns the toxemias. Toxemia was the leading cause of death in 1950, accounting for 19 per cent, whereas in 1951 the proportion had dropped to 7 per cent.

Failure to appreciate the intolerance of pregnant women to ordinary doses of anesthetic agents for spinal anesthesia accounted for two deaths. One patient received 150 mgm. of procaine and the other 18 mgm. of pontocaine. Both expired promptly of respiratory paralysis and shock.

Tables XI and XII are included to indicate the great need for improvement in the accuracy and completeness with which birth and death certificates are filled out. In eleven of the twenty-one incorrect death certificates, or 19.3 per cent of the total, the cause of death as determined by the Committee's investigation showed that the cause recorded on the death certificate by the physician concerned was incorrect.

Summary and Conclusions

1. A brief general summary of the results of the Minnesota Mortality Study for the period April 1, 1951, through March 31, 1952, has been presented. Certain comparisons are made between these results and those of the preceding year and with the results of a similar study carried out in Minnesota in 1941-1942.

2. There were fifty-seven deaths associated with pregnancy or the postpartum period in the 1951-1952 period among 80,099 live births for an over-all mortality rate of 0.712 per 1,000 live births. The rate when deaths due to non-obstetric complications are excluded was 0.45. This represents a further reduction as compared with the previous year's study and compares favorably with that of the whole United States. In 1951 the unofficial United States maternal mortality rate excluding non-obstetric complications was 0.71.

3. It is assumed that the remarkably low maternal mortality rate achieved means that efficient obstetric care is being administered by the vast majority of Minnesota physicians engaged in practicing obstetrics.

4. A significant reduction in the proportion of deaths that were considered preventable has taken place since 1941. Still more improvement is possible, since 42.1 per cent of the 1951 deaths were considered preventable.

5. There has been an increase in the percentage of deaths associated with pregnancy but due to non-obstetric causes—from 17 per cent in 1941 to 31.6 per cent in 1951. Thus, there is a significant decrease in deaths due to obstetric causes.

6. The percentage of adequate consultations obtained in 1951-1952 was more than three times that obtained in 1941.

7. Complete pelvic measurements were obtained in only 27.3 per cent of registered patients who died. This situation must be quickly improved. Serology was obtained in 43.2 per cent.

8. Of the delivered patients who died, 55.5 per cent were delivered by operative means. Of the operative deliveries, 40 per cent were considered not indicated and 45 per cent contributed to the patients' deaths.

9. As judged by the standard of minimum requirements for adequate obstetric care as adopted by the Maternal Mortality Committee, prenatal care was faulty in 86.3 per cent, care in labor and/or delivery was faulty in 74.5 per cent, and portpartum care was faulty in 57.6 per cent of the cases studied. The faulty care contributed to the patient's death in 22.7 per cent as regards prenatal care, 53.5 per cent as regards labor and delivery, and 45.5 per cent as regards postpartum care.

10. The primary cause of death is tabulated. Hemorrhage was the leading cause of death in 1951 with almost one-half of the deaths due to hemorrhage being accounted for by accouchment forcé. It is long past time for this procedure to be given up.

11. Adequate autopsies were performed in 40 per cent of the cases studied in 1951. This proportion is approximately five times the frequency with which autopsies were done in 1941.

12. Only 31.6 per cent of the death certificates and 48.6 per cent of the birth certificates reviewed were complete and correct. In 19.3 per cent, the cause of death as recorded on the death certificate was incorrect as determined by the Committee's investigation. The significance of this fact is clear. The validity of data obtained from unchecked death certificates is open at least to serious question.

13. The Committee would like to point again to the fact that in many of the hospitals in which these maternal mortalities are occurring, simple history taking, careful general and obstetric physical examination, and basic laboratory work are grossly deficient. That this is related to the end result for the patient is clear. The Committee would urge again that the medical and administrative staffs of such hospitals owe a duty to their patients to enforce the carrying out of these studies without exception. The minimum standards for adequate obstetric care as set down by the Committee are recommended.

14. The experience of the Maternal Mortality Committee would suggest that there are numerous sources of serious error in the usually reported maternal mortality rates. Case finding which depends on death certificate reports is incomplete and diagnoses from this source are grossly inaccurate. It is impossible to evaluate the accuracy of the division into so-called obstetric and non-obstetric deaths when only death certificate information is available.

15. The Committee is tempted to point out the justification for pride in the efficiency of obstetric care in Minnesota which these data indicate. It has overcome this temptation since there is clearly much to be improved and since pride and progress are uneasy bedfellows.

Reference

1. Maternal Mortality Committee, Minnesota State Medical Association: Minnesota maternity mortality study, April 1, 1950 to March 31, 1951. Minnesota Med., 36:609, 1953.

INTERVERTEBRAL DISC LESIONS IN THE TEENAGE GROUP

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LOW BACK PAIN and mid-back pain with or without radiating pain into the leg is a fairly frequent complaint of both boys and girls in the teenage group. When a patient complains of lumbar or lumbosacral type pain one expects primarily to find evidence of some disease, such as tuberculosis, neoplasm or epiphysitis. When a patient in the teenage group, in addition to the back pain, complains of lower extremity radiation, one is highly suspicious of intervertebral disc type lesion. Radiating pain is not necessarily always present or associated with an intervertebral type lesion and in our series it is fairly common to have a well-established radiographic evidence of intervertebral disc pathology with no complaint of pain extending into either lower extremity. We have recently reviewed a rather large series of low back complaints in patients varying from sixteen to twenty years, nineteen of whom have come to laminectomy for an intervertebral disc lesion.

One of our greatest handicaps in the understanding and treatment of intervertebral disc lesion, both in teenagers and adults, is our limited knowledge of the cause of disc degeneration. There are many theories as to the cause of progressive degeneration in normal intervertebral discs, none of which have been definitely accepted by orthopedists. Because of the activity of both boys and girls in the teenage group it is quite common to attribute any progressive degenerative condition to repeated trauma or to one single episode of trauma. In spite of the fact that several of our intervertebral disc cases occurring in teenagers have been in association with athletic participation, we are rather impressed with the fact that the amount of precipitating trauma in this age group is rather slight.

All of us have seen a great many cases of low back pain in the teenage group which may have been associated with some minimal type lifting injury or athletic injury which would give rather marked difficulty for ten days or two weeks and then would spontaneously improve on bed rest and symptomatic treatment. A great many of these cases have a tendency to recur periodically but never reach the point of becoming bad enough to justify a spinogram or intensive work-up. I feel that in a great many of these cases there is progressive disc degeneration which perhaps represents the early phase in the large number of adult patients whose x-rays show long-standing degenerative disc changes. A very high percentage of these back complaints in teenagers respond very well to symptomatic treatment, including well-fitting wide lumbosacral belts. Certainly this age group, as well as adults should have a long period of conservative management before a laminectomy is considered. All of our patients who eventually require laminectomy were first given an extended period of initial bed rest, heat, sedation, and support.

I feel that in teenagers as well as adults with disc lesions and with unilateral sciatic radiation, only a small percentage actually obtain much improvement from traction. If traction is to be attempted it should only be for a trial period and if there is no definite improvement within forty-eight hours it should be discontinued. In the type of disc where a large free fragment is forced out of the intervertebral space and locked under the posterior ligament traction will definitely be of no value and will many times produce an increase of both back and leg pain. If, after the period of rest and conservative treatment there has been no improvement or insufficient improvement to allow return to activity further work-up is necessary. We feel that a spinogram is indicated in all these patients to more accurately localize the lesion and eliminate possibility of an unrecognized double disc.

I would like to briefly review nineteen cases of patients who eventually have come to laminectomy. Of these eight were girls and eleven were boys, the youngest of whom was sixteen and the eldest had presenting symptoms at the age of nineteen. Several writers have pointed out that in children and teenagers intervertebral disc lesions seem to be rather unusual in their lack of complaints of numbness, parasthesia, and muscle weakness. We have been unable to find any variation in presenting complaints or physical findings between the teenage group and the adult group with similar lesions. Some writers have stressed the high incidence of lumbosacral disc lesions in the younger group but here again in our series eight cases had an L-4-5 defect; ten cases had an L-5-S-1 defect and one patient, a girl of seventeen, had at the same time a large defect at L-5-S-1 on the right and at L-4-L-5 on the left. The history of an injury period here again is quite variable. Only three had the beginning of back and leg complaints after athletic participation. Eight had a completely insidious onset of pain associated with no injury of any type. Two, a pharmacy student and a high school student, first had difficulty when bending over to tie their shoes. Four had difficulty after some minor lifting injury. One had an initial complaint of back and leg pain after sneezing and the last, the girl with the double-disc lesion, first had difficulty after falling a distance of approximately eighteen inches from a kitchen stool. On examination the physical findings in these patients were not unusual. The subjective complaints of shooting and radiating pain which was made worse by coughing, sneezing and straining, were the most common. In five patients out of the nineteen who had no shooting type, radiating pain, a herniated disc was suspected because of the rather long period of severe incapacitating back pain. All patients but three showed a very definite change in reflexes and five out of the nineteen had a definite change in sensation. None showed appreciable atrophy or definite muscle weakness.

Several authors have mentioned the finding of unusually stringy, or gelatinous, material obtained from the intervertebral disc space. This may be true in very young children with whom we have had no experience but in our cases the pathological specimens have been very little different from those in the adults. In five or six patients with large bulging discs which have been opened, a single fragment has been freely removable. The remaining material in the disc space has had a rubbery consistency and we have had the feeling that this represents an unripe or incompletely degenerated form of disc disease. It is in these patients that the prognosis is rather reserved. We make every effort to thoroughly curette the disc space and remove all such material but there is always the possibility of further separation of further degenerating disc material which may later give rise to a secondary protrusion from the same disc space. In the one patient of the nineteen who has had recurrent difficulty, a second laminectomy established the presence of more degenerated material at the same level and on the same side in rather large amounts. Except in these cases where an unripe disc is established at laminectomy we feel the prognosis in teenage patients is rather good. All patients are given a follow-up consisting of developmental back exercises and a gradual return to activity. Several of the patients have been able to return to rather strenuous athletic participation. One of our patients, against advice, was able to play first-string basketball at a Minnesota college three months after the laminectomy. Another patient has been able to satisfactorily play Big Ten football and has had no complaints after hard contact of line work.

Conclusion

In conclusion, I feel we should remember that true intervertebral disc lesions occur fairly frequently in the teenage group. Their treatment is chiefly conservative and only a small percentage require eventual laminectomy. The patients' presenting complaints and physical findings differ very little from adults. The prognosis in the patients not requiring laminectomy is rather good and in those requiring laminectomy the determining factor is the pathologic finding at operation. In cases in teenagers with complete separation of free fragment discs the prognosis is probably better than in adults with similar lesions.

THE FAMILY PHYSICIAN AND PUBLIC HEALTH

Health departments have traditionally worked with the full co-operation of the medical profession. The family physician is the front line of any public health endeavor. In order for a community health program to succeed, the practitioner must give full co-operation and active rather than passive support. Experience has shown that the more extensive the public health program a community enjoys, the greater the demand made by the public on services not only for the treatment of illness but also for health supervision and for preventive services.—Vlado A. Getting, M.D., J.A.M.A., Sept. 26, 1953.

Laboratory Aids to Medical Practice

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RADIOACTIVE ISOTOPES IN DIAGNOSIS AND TREATMENT

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NATURALLY occurring radioactive isotopes, such as radium and radon, have served medicine usefully for several decades. The discovery, in 1933, by Joliot and Curie that stable elements could be converted into artificially radioactive ones opened up undreamed possibilities that physicians are only beginning to realize.

The unstable (radioactive) isotopes or variants of naturally occurring elements achieve stability by means of reorganization of their nuclear structure. This reorganization is accompanied by emanations of various types, which include alpha, beta and gamma rays. As an example, one radioactive isotope of iodine (I^{131}) emits both beta and gamma rays; in so doing, its chemical properties are transmuted from those of iodine to those of xenon. The superscript number is used to identify the isotope, thus, I^{127} is the normal, stable form of iodine, while I^{131} is an isotope which weights 4 atomic units more. The various radioactive isotopes undergo nuclear reorganization at speeds varying from fractions of a second to billions of years. By convention these rates are expressed as the time required for the element to undergo half its radioactive degradation; thus, I^{131} has a "half life" of 8 days. In honor of Marie and Pierre Curie, codiscoverers of radium, the unit of radioactivity is the curie, which is approximately equal to the emanation from 1 gm. of radium. A thousandth and a millionth of a curie are designated as a "millicurie" (mc) and a "microcurie" (μc).

Radioactive isotopes are being used in medicine for therapeutic purposes and for tracer studies. The goal in treatment is to find an isotope with the following characteristics: (1) some degree of predilection for a certain organ or diseased cell, (2) sufficient strength of radiation to kill the diseased cells without serious injury to the patient as a whole; (3) no chemical toxicity and (4) a short half-life, so that the hazard of radiation is limited to days or at most weeks. To be able to concentrate a radioactive substance within a diseased portion of the body affords a means of intimate, continuous radiation without the interposition of normal tissues between the source and the target zone. Iodine, for example, tends to concentrate within the acinar epithelial cells of the thyroid gland; since these cells are unable to distinguish between stable I^{127} and radioactive I^{131}, administration of the latter leads to an internal source of radiation within hyperplastic or malignant thyroidal cells.

Radioactive colloidal gold (Au^{198}) settles out on pleural and peritoneal surfaces, affording a method of treatment of metastatic malignant lesions on these surfaces. Although cures by this method of treatment are rare, the relief of pain and diminution of effusion are often gratifying. Au^{198} may be injected directly into malignant tumors of the prostate, cervix or skin. Radioactive phosphorus (P^{32}), like its stable isotope, inclines toward regions of rapid growth, including bone marrow. This material lends itself readily, therefore, to treatment of the overactive erythropoiesis of polycythemia vera.

The other and perhaps more important medical application of radioisotopes is in the tracing of movements of elements within and between compartments of the body. The advantages of this method over direct chemical analyses are twofold. While one can measure chemically with precision the concentration of iodine (I^{127}) down to a few billionths of a gram per milliliter, one can detect radioiodine (I^{131}) in solutions still more dilute by a factor of several million. Secondly, if the radioactive element should emit gamma rays, sensitive counters can detect the element from outside the intact body, because the gamma rays readily penetrate several inches of tissue and pass into the neighboring atmosphere. When a detector of radioactivity is placed over the thyroidal region of the neck and radioiodine is given intravenously, one can measure accurately the rate with which I^{131} concentrates in the thyroid, as well as the absolute amount extracted from the blood. Both these values are proving to be diagnostically useful in disease of the thyroid.

Many other diagnostic uses of radioisotopes, based on measurement of internal radioactivity from outside the body, are being investigated. These include: (1) localization of tumors of the brain, retina, breast and liver; (2) demonstration of block of the spinal canal; (3) evaluation of extravascular concentrations of blood; (4) estimation of the vascularity of skin grafts and (5) measurement of cardiac output and circulation time.

When samples of fluids or tissues can be removed from the body, more refined radioisotopic techniques become available. These methods often have an added meaning when chemical or biologic assay of the stable chemical is made along with determination of radioactivity. Globulins labeled radioactively are being studied by immunologists. Curiously enough, these studies are yielding results somewhat different from those obtained by labeling the same proteins with azo dyes. When serum albumin tagged with radioactive iodine is injected intravenously and allowed to mix thoroughly, the resulting calculated volumes for plasma and whole blood are as accurate as the results of dye methods and are simpler to obtain. Erythrocytes may be tagged with a variety of radioactive elements, which enables one to determine erythrocytic volumes and to estimate the longevity of these cells in various hemolytic states.

To the biochemist and the physiologist, radioactive substances are becoming indispensable accessories, particularly since the radioactive isotope of carbon (C^{14}) has become generally available. Even the bacteriologist has at his disposal bacteria in which radioactivity is incorporated from their nutrient medium.

Since the radioactivity of these various isotopes is potentially dangerous to both the patient and the investigator, only those familiar with the hazards are allowed by the Atomic Energy Commission, which is the sole agency authorizing their use, to handle radioisotopes. By virtue of his training, the radiologist is familiar with the benefits and dangers of radiation; he should be an integral part of the team to handle the isotopes. With expanding use of isotopes in diagnostic procedures, the clinical pathologist will find himself also a member of the team.

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This is the thirteenth in a series of editorial reports sponsored by the Minnesota Society of Clinical Pathologists and designed to foster closer relationships between the practicing physician and the pathologist.

The use of radioactive isotopes again emphasizes, as have several other of these reports, the ever-increasing role played by the laboratory physician in the modern practice of medicine. Although most physicians will not actively handle radioactive materials, they should be aware of the potential hazards associated with such factors as disposal of radioactive excreta from patients who have received radioactive isotopes.

Reprints of this report are available from the author or the chairman of the editorial committee.

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RESIDENCY IN ANESTHESIOLOGY

The Army Medical Service residency program in anesthesiology will be increased in term to three years beginning July 1, 1954. to give candidates additional training, it has been announced by Major General George E. Armstrong, Surgeon General.

The new plan provides for the two years of clinical training required for American Board of Anesthesiology certification and an additional year devoted to research and development training co-ordinated with the basic sciences in anesthesiology.

According to the new plan, selected Medical Corps officers will spend their first year of the three-year residency at Walter Reed Army Medical Center, Washington, D. C., where they will receive fundamental clinical training.

The second year will be presented at the Army Medical Service Graduate School, Walter Reed Army Medical Center, Washington, and will be devoted to research and development.

The last year of residency will be spent at one of four Army hospitals and will present the candidates with a summation of training with clinical and teaching experience. The four hospitals to be utilized in the program are: Brooke Army Hospital, Brooke Army Medical Center, Fort Sam Houston, Texas; Fitzsimons Army Hospital, Denver, Colorado; Letterman Army Hospital, San Francisco, California; and Walter Reed Army Hospital, Walter Reed Army Medical Center, Washington, D. C.

President's Letter

DOCTOR AND PATIENT

The relationship of the doctor to his patient and the resulting public opinion of the profession as a whole should be uppermost in the minds of all doctors these days. In recent months, the entire medical profession has withstood many unpleasant attacks which have actually stemmed from the unethical practices of a small minority of practitioners.

Naturally, such articles bordering on the sensational, make excellent copy and rate high readership for both the author and the publications in which they appear. Medicine and medical news have reached a high point in lay readership, and as a consequence, any material of this nature is fair game for writers, and often results in unnecessary and unfair exploitation of the facts. It is unfortunate that only the substandard aspect of medicine should gain such notoriety.

On the official level, American medicine has reacted in various ways, ranging all the way from bitter indignation to complete disregard. Letters have been written to the editors, complete facts have been submitted, answering articles have been published, and verbal renunciations have been used.

However, would it be too much to ask of American medicine to revert back to the down-to-earth policy of improving its individual members' day-to-day relationships with the people who may hold some of those views—their patients? Physicians can do no better than that, to prove to the American people that they are conducting their practices on the highest possible level.

And, medicine is doing a job of policing itself on such matters. Medical organizations have established mediation boards with the express purpose of culling out those who infringe on medical ethics, and also to study any grievance against a member.

Another comeback to the sensational article writers has come to light recently. It is interesting and gratifying to see that some publications have printed articles pointing out this very fact—that the entire profession should not be condemned because of the unfortunate actions of a few.

With both writers and advertisers contributing, it is to be hoped that increased mutual good will and understanding will develop between doctors and patients.

Among the most recent items of this new pattern showing what American practitioners are really like are these: the January 11 issue of *Life* magazine carried an advertisement on "How to Select a Family Doctor;" the same ad appeared in the February 6 issue of the *Saturday Evening Post*, in February's *Woman's Home Companion,* February's *Parents Magazine* and in February's *Today's Health;* the November 1953 issue of *Better Homes and Gardens* carried an article entitled "When You Need a Doctor in a Hurry—What Will You Do?"; *Rotarian* of December, 1953 ran an article called "Don't Kill Your Doctor;" and *Look* of November 17, 1953 published a piece on how "Mississippi Trains Its Own Country Doctors."

These are only a few of the recent articles and advertisements appearing in national publications, which emphasize the positive side of American medicine. It is hoped that as a result of less sensational and more factual printed material, more and more people will realize that not all physicians adhere to the described unethical practices, and that the profession, as a whole, cannot be rightfully judged by the actions of the minority.

J. Ohage.

President, Minnesota State Medical Association

Editorial

Arthur H. Wells, M.D., *Editor;* Henry G. Moehring, M.D., and John F. Briggs, M.D.

POLIO VACCINATION WITH A PRAYER

WE HAVE rejoiced with the recently announced prospects of a control for poliomyelitis. The series of discoveries from those of John F. Enders culminating in those of Jonas E. Salk reads like a streamlined version of Microbe Hunters. We delight in the thought that the National Foundation for Infantile Paralysis may have been instrumental in hastening these discoveries by many years. We want ever so much to have this scourge of mankind under control as soon as possible and at the same time see to it that those responsible get the proper recognition. However, as physicians, we soon learn after a continuous series of disillusionments to modify our immediate reaction "This is it!" to "This could be it."

In Minnesota, the State Board of Health, as the official public health agency of the state, is in the position where it must assume responsibility for the program of vaccination against poliomyelitis in 1954. The Board has given careful and continuing consideration to this matter since first approached. Several meetings have been held on the program with members of the State Polio Planning Committee; with the Council and other representatives of the Minnesota State Medical Association; with officials of the National Foundation for Infantile Paralysis; and consultation has been had by telephone and correspondence with scientists in other states, Canada, and Washington. The State Board, in addition, called together a special scientific advisory committee which met on January 20 to advise the board as to recommended procedures in the light of the latest available information. Developments since then have likewise been fully considered.

We wish to commend most highly the State Board for its cautious and yet complete approach to this important problem. The medical profession, as a group, has been constantly kept in the picture, and the advice of the best scientists in this field has been obtained. Everyone concerned has shown a desire to give wholehearted endorsement and support to the program of successful polio vaccination in Minnesota; yet throughout, care is being exercised to ensure complete safety of the procedure and the collection of sound data on which to evaluate the effectiveness of the vaccine. When the time comes for final plans, the medical profession will be glad to do its part.

ROUTINE CHEST ROENTGENOGRAMS IN PRIVATE HOSPITALS

IT is a matter of established fact that the most fertile field for the discovery of pulmonary lesions lies in the routine roentgen examination of the chests of patients being admitted to hospitals. It is also unquestionably a fact that hospitals should take all steps possible to protect their employes and patients from infectious tuberculosis. This is because of the responsibility, both moral and legal which the hospital must take. Therefore, it is of the utmost importance that hospitals make this relatively simple examination of all persons becoming associated with them, not only as a patients but also as employes or attendants in any capacity.

Most of the hospitals in Minnesota will be forced to make necessary adjustments for the examinations in their present accommodations. It is important that the examination unit be in as close proximity as possible to the admission area. The greater the distance between these departments the more difficult it is to obtain roentgenograms of patients, particularly of those admitted under trying circumstances, such as persons injured or those in labor. New hospital buildings being planned or constructed may easily make provisions for chest roentgenograms near or adjacent to the admission office. As a matter of fact, it seems that new hospital plants are now being arranged with the roentgen department on the entrance floor for greater convenience.

The type of routine roentgen examination does not have great significance. The method of making a roentgenogram in only one projection can be regarded as no more than a survey regardless of whether miniature or standard sized films are used. In general, it seems that hospitals of 150-bed capacity or less could best use 14 x 17-inch films made in the established roentgen department. On the other hand, larger institutions should be economically better off by investing in a photoroentgen unit. These larger institutions would eventually repay the investment in a photoroentgen unit merely in the saving made on the examinations of new employes and the interval follow-up examinations of these employes.

The Minnesota Radiological Society has recommended that a charge of $1.50 be made to patients being admitted for photofluorograms and that a $4.00 charge be made for 14 x 17 films. These charges are in effect in those institutions employing either method in the Twin Cities. It is felt that these charges make the procedure self-supporting, which is no more than proper. A profit would probably defeat the purpose of the survey. On the other hand, the findings are of sufficient value to the individual being examined to make it worth his while to underwrite his share of the money and effort being expended by those responsible for the project.

It is apparent that processing of the films can best be done in the established roentgen department. It does not appear that there is any hospital in Minnesota sufficiently large to justify the establishment of a separate processing unit. It is cheaper and easier to provide the necessary material for developing and subsequent handling of film to last over a twenty-four-hour period than it is to construct and maintain separate facilities for this work. This also eliminates the need for a self-sustaining staff for the unit. Technicians, often students, can be employed to operate examination units in the larger institutions from 2 to 5 or 6 p.m. daily when most hospital admissions take place. During this time, these employes can also make roentgenograms of the chests of those persons admitted but not examined during the previous twenty-four hours. During the remainder of each day, the admitting office personnel can be made responsible for the procedure. It is a simple matter to train any reasonably intelligent person in the technical aspect of making a chest roentgenogram.

The keeping of records should be as uncomplicated as possible, as long as the records accomplish the primary purpose of the survey, namely—to protect patients and employes from infectious tuberculosis. The hospital *does* have the right to see to it that infectious tuberculosis be recognized or ruled out in any given chest lesion. Also the hospital has the right to insist that any necessary precautions be carried out when infectious tuberculosis is suspected. Beyond that, the individual members of the staff and medical organizations at the staff level and upward are responsible for subsequent diagnosis, observation and treatment of patient having lesions which are non-infectious.

Simplicity of record keeping saves time, money and personnel. It reduces the research value of the project. However, research should be left primarily to subsidized institutions. Hospitals depending for their support upon private fees are obligated to use those fees for private benefits. If the private hospital were to become engaged in a large scale follow-up program, elaborate records would become necessary. Keeping the fact in mind that the survey is made to discover infectious tuberculosis, a long-range follow-up program does not seem to be the responsibility of a private hospital. All positive findings should be reported to local or state health authorities for subsequent action. Such a plan could add greatly to the overall value of the projects under discussion.

It should be of interest to record briefly the most important findings in an admission survey made over a three-year period in one Minnesota private general hospital.* During the three years 17,790 patients had photofluorograms. The interpreter noted 2,307 positive findings. Twenty-six of these patients, previously unsuspected of having disease, are now undergoing institutional treatment for pulmonary tuberculosis. Three hundred sixteen are being observed or treated privately for suspected tuberculosis. Twenty-nine primary thoracic tumors were discovered, and twenty-one metastatic pulmonary lesions came to light. Eleven "coin" lesions were found, the majority of which are known to be static by reference to previous examinations or by subsequent examinations. Exact classification of these eleven lesions cannot be given because they have not been removed. Cardiac and aortic abnormalities accounted for 629 positive findings. Other findings, such as large calcified primary complexes, adhesions, anomalies, scoliosis, old fractures, et cetera, of doubtful or no clinical significance, accounted for most of the remaining positive reports. However, eighty pleural effusions (mostly congestive), two cases of silicosis, a pulmonary cyst and other interesting conditions were seen.

A routine admission film is unquestionably of great value in the study of general hospital patients. In addition to its primary purpose, it frequently provides a short-cut to the final diagnosis and disposition of the patient. The total time of hospitalization is often reduced by a more speedy diagnosis. Discovery of pertinent findings not related to the reason for hospitalization may be made. We believe that routine chest examinations are more important in the detection of disease than routine urinalysis and blood studies.

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*St. Joseph's Hospital, St. Paul, Minnesota.

J. P. Medelman, M.D.

ROUTINE X-RAYING OF PATIENTS ADMITTED TO GENERAL HOSPITALS

THE x-raying of all admissions to general hospitals is an essential element of the tuberculosis control program. Most reports of hospital admission x-ray screening programs cite the discovery of unsuspected cases of disease, and relate an over-all percentage yield of tuberculosis that is higher than that found in the screening of general population groups. This is quite to be expected, for patients coming to hospitals are generally older people, and people with chronic diseases and vague symptoms. It is this group that usually participates poorly in general population surveys, which are directed at finding unsuspected disease in the apparently healthy person. Moreover, since some heart conditions and other chest disorders, as well as tuberculosis, can be detected by admission x-rays, good medical practice would seem to require that chest x-rays be taken on admission as routinely as blood tests and urinalyses.

The program of x-raying all hospital admissions represents an investment that pays multiple dividends to patients, hospital and community. Often, treatment for a previously undetected disease is received by the patient at an earlier date as a result of discovery by admission x-ray. In addition, the risk of infection within the hospital from an unsuspected case of tuberculosis is minimized for fellow patients, staff and personnel. And lastly, the chain of tuberculosis infection within the community is broken when a previously unsuspected case of tuberculosis is detected by the admission x-ray and brought under public health supervision.

Minnesota deserves congratulations on having one of the lowest tuberculosis death rates in the world in 1952. In fact, tuberculosis has not been among the ten leading causes of death in the state for the past three years. The achievement of this enviable record comes as no surprise, in view of Minnesota's vigorous execution of a progressive tuberculosis control program.

It is also gratifying to know that approximately forty of Minnesota's hospitals are routinely x-raying all admissions and that this program has the endorsement of the Minnesota State Medical Association, the Minnesota Department of Health, and the Minnesota Tuberculosis and Health Association. The fact that more than 2,000 previously unreported cases of tuberculosis are still being found each year in the state, emphasizes the necessity of intensifying case-finding efforts. Like the widely accepted techniques of surveying apparently healthy population groups, of x-raying residents and employes of mental and penal institutions, and of examining contacts and suspects, case-finding among hospital admissions is another extremely effective device which merits application. The general hospitals in Minnesota that do not routinely x-ray their patients on admission are urged to explore the possibilities of this important aspect of a tuberculosis control program.

Although x-raying all admissions presents the already heavily burdened general hospital with additional administrative and financial problems, many of these problems can be readily solved; the costs of the service are moderate, and the rewards are many. Coupled with recent improvements in treatment, the routine x-raying of some 13 million Americans entering general hospitals each year, would certainly bring us closer to the control of tuberculosis.

R. J. Anderson, M.D.

Doctor Anderson is Chief, Division of Chronic Disease and Tuberculosis, Public Health Service, U. S. Department of Health, Education, and Welfare.

MATERNAL MORTALITY SURVEYS

NATIONAL mortality rates were high and showed little change from 1915 to 1930. The average rate was 7.0 maternal deaths for every 1,000 live births. During this same period in Minnesota there was an average of 5.2 maternal deaths per 1,000 live births. Prior to the enactment of the Shepard-Towner Act of 1921 maternal health activities were carried on by a few state health departments or state medical associations. In 1917, the New York Academy of Medicine first became interested in the problem of puerperal mortality because death rates from puerperal causes had remained stationary while death rates from other preventable causes had been steadily declining. Our Division of

Child Hygiene, now known as the Section of Maternal and Child Health, was established by the Minnesota Department of Health in 1922. The Maternal Health Committee of the Minnesota State Medical Association was first appointed in 1936.

Because there was little change in the national maternal mortality rates for the previous ten years, the Obstetric Advisory Committee of the U. S. Children's Bureau, of which Dr. Fred L. Adair of Minnesota was a member and later the chairman of the American Committee on Maternal Welfare, in 1926 presented a plan of study of all puerperal deaths to be conducted in certain states in 1927 and 1928. Studies of maternal mortality based on death certificates were known to be insufficiently detailed to give an accurate picture of the situation. Accordingly, the study was to be carried out by means of personal interviews with the physicians attending all deaths from puerperal causes. The study was made in fifteen states, including Minnesota, at the formal request of each state medical society. The interviews were carried out by physicians on the staff of the State Boards of Health who visited the attending physician or midwife as well as the hospital concerned. The report was written under the guidance of the Obstetric Advisory Committee and was the first intensive and accurate study from which valid conclusions could be drawn. It furnished the impetus for subsequent studies which led to the establishment of maternal health committees in various parts of the country.

In 1930, the New York Academy of Medicine through its Committee on Public Health Relations began a detailed "study of the phases of the public health problems of obsetrics as they affect New York City." It was patterned along the lines of the survey made by Adair and his associates—the so-called "Fifteen States Study" and used the same questionnaire so that satisfactory comparisons would be possible. The three-year study was made in co-operation with the Department of Health of New York City and the Obstetrical Society of New York. The final conclusion of this study stated, "The hazards of childbirth in New York City are greater than they need be. Responsibility for reducing them rests with the medical profession." Coming from the medical profession itself and pointing out that two-thirds of deaths due to childbirth could be prevented if the care of the woman had been proper in all respects, the report aroused intense controversy and was followed by a storm of lay and medical criticism. Its publication, however, led to the formation of maternal mortality committees in many local groups and hospitals. A committee on maternal welfare was established by the Philadelphia Medical Society which is still actively functioning. Shortly afterwards such committees were organized by county medical societies of New York City, Cleveland, Boston and other areas.

This activity on the part of the medical profession in co-operation with public health authorities has been a major factor in the marked and continued decline in maternal mortality. Other factors that accelerated the rate of decline included better undergraduate training of physicians, increased postgraduate training and refresher courses in obstetrics, increased rate of hospitalization and more adequate hospital facilities and services, widespread use of chemotherapeutic agents and antibiotics, and increased availability and use of blood. In addition, public health measures such as the education of the public to the importance of early and adequate prenatal care as well as good obstetric care has played a significant role.

Information from the state health departments of all states and territories in 1951 indicated that maternal mortality surveys were being carried out in nineteen states and the District of Columbia. A number of larger cities also carry on similar studies, including Philadelphia, New York City, Syracuse, San Diego, and others. Twelve states send questionnaires to physicians and hospitals. A few limit their surveys to the information available on death certificates. Only eight states, including Minnesota, make a thorough investigation by the personal visit of trained obstetricians, considered by most authorities to be the only effective means of obtaining accurate and worthwhile data. Within the past year the Wisconsin State Medical Society in co-operation with the Wisconsin Department of Health has instituted a similar study. During the past two years the Minnesota Maternal Mortality Committee has received requests for detailed information on the operation and procedures of the study from eight state medical and obstetrical societies.

The first statewide maternal mortality study in Minnesota was carried out from July 1, 1941, through June 30, 1942, by the Maternal Mortality Committee of the Minnesota State Medical As-

(Continued on Page 160)

The Dean's Page

THE LYON LABORATORIES

On February 11, the University of Minnesota will dedicate the new medical building which is being named in honor of the late Elias Potter Lyon, Dean of the Medical School from 1913 to 1936.

This building, which joins Millard Hall and the Anatomy Building along Washington Avenue, contains four stories and a basement. The ground level is left open for a passageway into the court of the Medical School quadrangle.

The first floor of this building is divided into laboratories for histo-cyto-chemical research and is under the direction of Dr. David Glick, a national leader and authority in this field of work. The funds for this unit were provided by the National Heart Institute in order to increase facilities for research and for the training of personnel in this new and important field of micro-chemistry.

The second and third floors are assigned to cancer biology and provide facilities for the work of Dr. John J. Bittner and his associates, Dr. Carlos Martinez, Dr. Marthella Frantz, etc. One of these floors is devoted to laboratories, while the other houses thousands of pure strain inbred mice. Dr. Bittner is internationally known for his discovery of the "milk factor" in the development of hereditary mammary cancer in mice, as well as for other contributions to knowledge about genetic factors in cancers of mice. For this work Dr. Bittner recently received an award, the first made under a special bequest, from Middlesex College Hospital in London for the most important contribution to cancer research made anywhere in the world. The funds for the construction of these two floors were provided by the Minnesota Division of the American Cancer Society.

The fourth floor is devoted to bio-physics, a field of basic medical research of constantly increasing importance. Laboratories on this floor are utilized by Dr. Maurice Visscher and others in the Department of Physiology, and Dr. Karl Stenstrom and his colleagues in Radiation Therapy. The funds for this unit were provided by the National Cancer Institute.

The basement, as soon as funds for its completion can be obtained, will be devoted to air-conditioned animal quarters and associated research laboratories. This will enable us to use for other purposes the roof-house of Millard Hall which has been ill-suited and unsatisfactory for animal quarters.

Altogether this new building provides the Medical School with some long and urgently needed facilities for basic medical research. We invite the physicians of the state to visit this unit when they have an occasion or opportunity to come to the campus.

Harold S. Diehl, M.D.,
Dean of the Medical Sciences

Medical Economics

Edited by the Committee on Medical Economics
of the
Minnesota State Medical Association
George Earl, M.D., Chairman

FTC FIGHTS DECEPTIVE ADVERTISING FOR HEALTH INSURANCE

A nationwide investigation into objectionable and deceptive advertising for hospitalization and medical care insurance has been initiated by the Federal Trade Commission. According to a recent issue of the Washington Report on the Medical Sciences, trade practice rules were promulgated by FTC in 1950, but this is the first large scale inquiry of its kind to be undertaken by the Commission.

Assistance of businessmen, state authorities and the public has been requested by Chairman Edward F. Howrey. Among the cases of alleged violations of trade rules already in FTC's hands are several which it received recently from William Langer, Republican senator from North Dakota, who is chairman of the Senate Judiciary Committee.

According to the Washington Report, "an estimated 800 health and accident companies, relying chiefly on advertising and mail-in coupons for their business, are in this highly competitive field today. There were fifty in 1925. In 1952, net premium payments totaled nearly $1.9 billion, which was an increase of 206 per cent over 1946."

CLAMOR FOR SECURITY REPORTED BY MRS. HOBBY

According to Mrs. Oveta Culp Hobby, secretary of the Department of Health, Education and Welfare, "the American people want more economic security."

Mrs. Hobby spoke to the third annual American Assembly recently, meeting in New York to discuss economic security problems. Mrs. Hobby pointed out that Federal Social Security was one of three foundations for the economic security of the individual, supplementing industrial pension and insurance plans and individual savings. She remarked that the present high rate of individual savings indicated that Social Security had not affected the incentive of individuals to provide for their own future, insofar as they are able.

According to a recent news issue from the Research Council for Economic Security, which reported the *New York Times*' account of the meeting:

> "It was stated that the conferees were in general agreement that the American people wanted more Social Security because 'they realized increasingly how little economic independence they had as individuals.' The consensus of the panels was that the average American had a feeling of insecurity and that such anxiety was justified by past experiences and the complexities of contemporary life. These factors, rather than a softening of national fibre, are responsible for concern over social security programs, the participants are reported to have found."

HOUSE, TOO, LOOKS AT SOCIAL SECURITY

Before the opening of the second session of the 83rd Congress in January, the fact-finding hearing on the Social Security program opened with much less flourish. The House ways and means subcommittee hearings were the preliminary to what promises to bring a very explosive battle over the whole philosophy of the old-age and survivors-insurance system. Evidences of this coming battle have already been seen in political events of the past month or so.

No matter how hot and heavy the battle, the committee, under Representative Carl T. Curtis of Nebraska, brought out some very useful and interesting information about Social Security costs and possible extension of coverage.

President Eisenhower has repeatedly asked for extension of Social Security to cover about 6,500,000 more persons, including self-employed farmers, domestic servants not now covered, and professional persons, with optional coverage to be provided for an estimated 4,000,000 more, including clergymen and employes of state and local governments.

Present System Studied

The Committee spent considerable time studying the present system of Social Security, and brought to light some interesting facts and figures regarding the large amount of old-age-benefit dollars going abroad.

According to a recent issue of *Insurance Economics Surveys,*

> "Records show that the $13,000,000 in Social Security benefits paid to beneficiaries abroad in 1952 exceeded the amount going to beneficiaries in 12 states here at home. These states were listed as Arizona, Delaware, Idaho, Montana, Nevada, New Mexico, North Dakota, South Dakota, Utah, Vermont, Wyoming, and Mississippi, mainly agricultural states where farmers are not covered by Social Security.
>
> "It also brought out that such beneficiaries abroad are in a position to earn unlimited amounts on the side, whereas beneficiaries here forfeit their benefit payment if they earn more than $75 a month. Social security authorities admitted that they were stumped in finding a means of enforcing that stipulation abroad."

The American Medical Association has taken a stand opposing the inclusion of the medical profession in the extension of social security.

PRESIDENT PRESENTS HEALTH PROGRAM

President Eisenhower's State of the Union message to Congress brought forth many statements on general Administration policy on important medical matters. In summary, this is what he said:

Socialization of Medicine: "I am flatly opposed to the socialization of medicine. The great need . . . can best be met by the initiative of private plans. But it is unfortunately a fact that medical costs are rising and already impose severe hardships on many families. The federal government can do many things and still avoid the socialization of medicine."

Research: "The federal government should encourage medical research in its battle with such diseases as cancer and heart ailments, and should continue to help states in health and rehabilitation."

Hill-Burton Program: "The present . . . act should be broadened to assist in the development of adequate facilities for the chronically ill . . . of diagnostic centers, rehabilitation facilities and nursing homes."

Reinsurance of Health Plans: "The war on disease . . . needs a better working relationship between government and private initiative. . . . A limited government reinsurance service would permit the private and non-profit insurance companies to offer broader protection to more of the many families which want and should have it."

Rehabilitation: "The program for rehabilitation of the disabled especially needs strengthening. . . . This program presently returns each year some 60,000 handicapped individuals to productive work. Far more disabled people can be saved each year . . . if this program is gradually increased."

Military Dependents: "Pay alone will not retain in the career service . . . the necessary numbers of long-term personnel. I strongly urge, therefore, a more generous use of other benefits important to service morale. Among these are more adequate living quarters, and medical care for dependents."

Medical Tax Deductions: ". . . we propose more liberal tax treatment for dependent children who work, for widows or widowers with dependent children, and for medical expenses."

Veterans: "The internal reorganization of the Veterans Administration is proceeding with my full approval. When completed, it will afford a single agency whose services, including medical facilities, will be better adapted to the needs of those 20 million veterans to whom the Nation owes so much." (There was no further reference to VA in the address.)

Social Security: "I ask that this extension (to 10,000,000 more persons) soon be accomplished. This and other major improvements . . . will bring substantial benefit increases and broaden the membership of the insurance system, thus diminishing the need for Federal grants-in-aid. . . ."

PLAN WOULD REINSURE VOLUNTARY HEALTH INSURANCE

The reinsurance of voluntary health insurance plans, referred to above, was asked of Congress by the Administration. The heart of the program probably would be a corporation set up with a federal appropriation for its original working capital. Once under way, its operations would be supported by payments from the member health insurance organizations of a percentage of their gross income from premiums. In some respects, it would be similar to the Federal Deposit Insurance Corporation for bank deposits.

According to a recent issue of the AMA Washington Letter, the reinsurance corporation device follows the general lines of the 1950 Wolverton bill. Mr. Wolverton was then a minority member of the House Interstate and Foreign Commerce Committee which must handle such legislation. Now he is chairman and in a position to effectively push his idea. The Eisenhower plan is understood to include the following three major objectives of the Wolverton bill:

"1. Make it possible for health insurance plans to offer catastrophic or comprehensive coverage by having the federal corporation pay one-third or three-fourths of each survivor's claim in excess of $500 or $1,000.

"2. Make it possible for—or even require—the plans to offer coverage to individual subscribers.

"3. Require all participating plans to scale the cost of premiums in relation to family income. Families at the $5,000 or $6,000 level would pay the maximum, with those earning less paying less."

Other pertinent factors involved in this reinsurance plan are:

"Participating groups would pay about 2% of gross premiums collected to support federal corporation. . . . A limit would be placed on out-state subscribers. . . . Also a limit on over-the-schedule payments to physicians or hospitals. . . . Benefits would have to be clearly defined and meet standards set by the corporation. . . . Patients would have to be allowed free selection among participating hospitals and physicians. . . . Every licensed physician would be eligible to participate. . . . The Wolverton plan excludes commercial companies but the present administration program would admit them."

OREGON MEDICAL SOCIETY OPPOSES REINSURANCE

In line with the American Medical Association's opposition to the Administration plan for reinsurance of voluntary non-profit health insurance, the Oregon State Medical Society has already passed a resolution against such a proposition.

Quotes Supreme Court

Basing its opposition on the belief that reinsurance would actually subsidize the plans and therefore bring federal control, the Oregon organization states:

"The administration has declared that its proposed subsidies to voluntary health insurance plans do not involve 'government regimentation and control' in spite of the fact that the United States Supreme Court (1942) has declared that 'it is hardly lack of due process for the government to regulate that which it subsidizes.'"

The resolution further declares:

". . . the intervention of the federal government in the form of subsidies or otherwise in the operation of the voluntary health insurance plans is unwarranted in the face of the outstanding record of these plans which already have extended their benefits satisfactorily to increasingly large segments of our people, and are increasing the scope of their benefits as rapidly as is consistent with financial soundness. . . ."

Woman's Auxiliary

RAMSEY COUNTY REPORTS ON PROJECTS

The Women's Auxiliary to the Ramsey County Medical Society has completed several major projects since the beginning of activities for the year last fall.

The doctors' wives co-operated with the Fourth District Minnesota Nurses' Association in sponsoring panel discussions in various high schools throughout Saint Paul for nurse recruitment. Doctors' wives sat in on many of the panels, and aided in the transportation of speakers to and from the meetings. Mrs. Wallace Gleason served as chairman.

At the Board meeting in November, 1953, the Auxiliary made sixty cancer dressings.

The Ramsey auxiliary also co-operated in the annual Christmas Seal sale, helping to staff downtown booths with thirty-eight women. Total sales were $136.22. The auxiliary aided in the sponsorship of the annual tuberculosis essay contest among Saint Paul school children. Mrs. George Snyder served on the judging panel. The contest was won by Barbara Tauer of St. Joseph's school and by Ray Blacik of St. Agnes' school.

The January meeting was held at the Saint Paul College Club on January 25. Mrs. M. D. Hilker served as program chairman. The program emphasized civil defense and featured an excellent discussion by Mr. Donald Wood, executive secretary of the Association of Twin City Hospitals. Two films were shown: "One Survival" and "Emergency Action to Save Lives." At this meeting, the winners of the tuberculosis essay contest appeared to read their essays and also to receive $10 gift bonds from the Auxiliary.

AUXILIARY MEETS IN CROOKSTON

The Women's Auxiliary to the Red River Valley Medical Society met at the home of Mrs. D. E. Stewart, Crookston, Friday evening, January 8, 1954. The meeting followed a dinner at the Hotel Crookston with the Red River Valley Society. It also occurred on a night of wind and snow which forced many out-of-town visitors to remain in Crookston overnight.

Mrs. Stewart reported on subscriptions to *Today's Health,* and Mrs. J. F. Norman, of Crookston, gave the report on current legislation. Mrs. Norman is legislative chairman of the group.

Mrs. C. L. Oppegaard told about the regional meeting of the State Auxiliary organization at Detroit Lakes, and Mrs. A. R. Reff, of Crookston, reported on the orthopedic clinic held in Crookston in October.

Among out-of-town guests were Mrs. G. A. Sather, of Fosston; Mrs. C. L. Roholt, of McIntosh; Mrs. Clifford Stadem, of Twin Valley, and Mrs. B. R. Kinkade, of Ada.

MINNESOTA STATE BOARD OF MEDICAL EXAMINERS

230 Lowry Medical Arts Bldg., Saint Paul, Minnesota

E. M. Jones, M.D., Secretary

PHYSICIANS LICENSED FEBRUARY 13, 1953

January, 1953, Examination

Name	*School*			*Address*
ALARI, Heino	Eberhard-Karls U. Tubingen, Germany	MD	1947	Northwestern Hosp., Minneapolis, Minn.
ANDERSON, Hugh Vaux	U. of Oregon	MD	1952	3425 S.W. 9th, Portland, Ore.
BEDDOW, Ralph Marlowe	U. of Oregon	MD	1950	102-110 2nd Ave. S.W., Rochester, Minn.
CRISP, Jr., Elmer Ernest	Northwestern U.	MD	1950	102-110 2nd Ave. S.W., Rochester, Minn.
DAVIS, Charles Truman	U. of Tennessee	MD	1950	102-110 2nd Ave. S.W., Rochester, Minn.
DENKER, Arthur Gilman	U. of Oregon	MD	1952	63 E. Ash, Lebanon, Ore.
DUNN, William Joseph	U. of Illinois	MD	1948	102-110 2nd Ave. S.W., Rochester, Minn.
ELSTNER, Howard L.	U. of Pittsburgh	MD	1951	102-110 2nd Ave. S.W., Rochester, Minn.
ERICKSON, Robert Dell	U. of Minnesota	MD	1953	Wykoff, Minn
FISHER, Don Harvey	Northwestern U.	MD	1951	102-110 2nd Ave. S.W., Rochester, Minn.
GALLO, Bela	Peter Pazmany U. Budapest, Hungary	MD	1945	Lake Bronson, Minn
GOSSELIN, Gilles	U. of Montreal	MD	1949	102-110 2nd Ave. S.W., Rochester, Minn.
HARDY, Paul Edward	St. Louis U.	MD	1951	Ancker Hosp., St. Paul, Minn.
HEALY, John Russell	Jefferson Med. Col.	MD	1949	102-110 2nd Ave. S.W., Rochester, Minn.
JACKS, Quentin Douglas	U. of Mantitoba	MD	1940	102-110 2nd Ave. S.W., Rochester, Minn.
JARVIS, Bruce William	U. of Minnesota	MD	1953	1869 Marshall Ave., St. Paul, Minn.
LEPPO, Niilo Erkki Albert	U. of Helsinki Helsinki, Finland Candidate of Medicine Licentiate of Medicine		 1931 1936	503 Med. Arts Bldg., Duluth, Minn.
MacMILLAN, Angus Hugh	Queens U.	MD	1950	102-110 2nd Ave. S.W., Rochester, Minn.
McPHERSON, Alan Given	U.of Manitoba	MD	1951	102-110 2nd Ave. S.W., Rochester, Minn.
MANSTEIN, George	U. of Pennsylvania	MD	1941	102-110 2nd Ave. S.W., Rochester, Minn.
MYERS, Richard Lyon	St. Louis U.	MD	1951	102-110 2nd Ave. S.W., Rochester, Minn.
OLAVS, Olga	U. of Latvia Riga, Latvia	"Physician"	1942	1009 Nicollet Ave., Minneapolis, Minn.
PRICE, Mary Lomas	U. of Aberdeen Aberdeen, Scotland	MB SB	1947	102-110 2nd Ave. S.W., Rochester, Minn.
SATERSMOEN, Jr., Theodore	U. of Minnesota	MD	1948	Veterans Adm. Hosp., Dearborn, Mich.
SIBLEY, John Adams	U. of Chicago	MD	1946	102-110 2nd Ave. S.W., Rochester, Minn.
SKILLERN, Scott Dorsey	U. of Pennsylvania	MD	1951	102-110 2nd Ave. S.W., Rochester, Minn.
STREITZ, John McDonald	U of Minnesota	MD	1952	Veterans Adm. Hosp., Minneapolis, Minn.
VAN PROOYEN, Cornelia Mary	Washington U., Mo.	MD	1951	Mpls. Gen. Hosp., Minneapolis, Minn.

Reciprocity Candidates

Name	*School*			*Address*
BEST, Etta Wright	U. of Louisville	MD	1949	102-110 2nd Ave. S.W., Rochester, Minn.
CARROLL, John Joseph	Creighton U.	MD	1947	117½ S. 1st St., Montevideo, Minn.
COOK, Clayton Stevens	Tulane U.	MD	1949	102-110 2nd Ave. S.W., Rochester, Minn.
FELDAKER, Mauri	Washington U., Mo.	MD	1950	102-110 2nd Ave. S.W., Rochester, Minn.
GOFF, John Robert	U. of Rochester	MD	1951	102-110 2nd Ave. S.W., Rochester, Minn.
GUNN-SMITH, Allan Murray	U. of Minnesota	MD	1950	431 10th Ave., Two Harbors, Minn.
HEIST, Erwin William	U. of Iowa	MD	1951	Jaguum Hosp., Lutheran Mission, Madang, Terr., New Guinea
HUGHES, Sidney Osborne	U. of Pennsylvania	MD	1947	172 Main St., Winona, Minn.
HYMAN, Jr., Orren Williams	U. of Tennessee	MD	1951	102-110 2nd Ave. S.W., Rochester, Minn.
JESSE, Richard Henry	U. of Nebraska	MD	1949	Truman, Minn.
KEYS, John R.	U. of Cincinnati	MD	1950	102-110 2nd Ave. S.W., Rochester, Minn.
KUNTZ, Aloysius Leo	Tulane U.	MD	1951	102-110 2nd Ave. S.W., Rochester, Minn.
LEMMON, Mark Leonard	U. of Texas	MD	1948	102-110 2nd Ave. S.W., Rochester, Minn.
O'LEARY, James Michael	Jefferson Med. Col.	MD	1946	102-110 2nd Ave. S.W., Rochester, Minn.
OPFELL, Richard William	U. of Iowa	MD	1949	102-110 2nd Ave. S.W., Rochester, Minn.
PERRY, Jr.,John Francis	U. of Texas	MD	1947	U. of Minn. Hosp., Minneapolis, Minn.
SANDERS, Edward Joseph	Creighton U.	MD	1949	102-110 2nd Ave. S.W., Rochester, Minn.
SLEDGE, Claire Blount	U. of Oklahoma	MD	1948	102-110 2nd Ave. S.W., Rochester, Minn.
SPITTEL, Jr., John Allen	U. of Maryland	MD	1949	102-110 2nd Ave. S.W.. Rochester, Minn.

Name	*School*		*Address*
TOON, Robert Wallace	U. of Oregon	MD 1944	1251 Med. Arts Bldg., Minneapolis, Minn.
TROUP, Richard Henry	Northwestern U.	MD 1950	102-110 2nd Ave. S.W., Rochester, Minn.

National Board Candidates

Name	*School*		*Address*
ALLEN, George Seaver	U. of Rochester	MD 1950	Mineral Spgs. San., Cannon Falls, Minn.
BAGLEY, Russell Willis	Tufts Med. College	MD 1948	871 S. Wilder St., St. Paul, Minn.
BEEMAN, Edward Arthur	Boston U.	MD 1947	102-110 2nd Ave. S.W., Rochester, Minn.
BENJAMIN, Robert Bedell	U. of Minnesota	MD 1952	18th & Taylor, Hood River, Ore.
BOUCHELLE, McLemore	Harvard U.	MD 1943	6th Ave. & 9th St. N., Virginia, Minn.
BUCKLEY, Joseph John	New York Med. Col.	MD 1946	500 Union St., Minneapolis, Minn.
CHAPIN, Lemuel Edward	Temple U.	MD 1951	102-110 2nd Ave. S.W., Rochester, Minn.
GORDON, Donald Lloyd	New York Med. Col.	MD 1947	102-110 2nd Ave. S.W., Rochester, Minn.
LIPINSKI, Stanley Wnuk	Northwestern U.	MD 1951	Hibbing, Minn.
SILVER, Lawrence	New York Univ.	MD 1950	Hosp. of Rockefeller Inst. for Med. Research, 66th St. & N. Y. Ave., New York 21, N. Y.
SVIGALS, Robert Elliott	New York Med. Col.	MD 1951	102-110 2nd Ave. S.W., Rochester, Minn.
VAN BUSKIRK, Charles	Albany Med. Col.	MD 1947	U. of Minn. Hosp., Dept. Neurology, Minneapolis, Minn.
WEBB, Jr., Wilson Davenport	New York Med. Col.	MD 1947	102-110 2nd Ave. S.W., Rochester, Minn.
YOUNG, John Van Winkle	Northwestern U.	MD 1951	102-110 2nd Ave. S.W., Rochester, Minn.

PHYSICIANS LICENSED MAY 22, 1953

April, 1953, Examination

Name	*School*		*Address*
AMBRUS, Laszlo	Eberhard-Karls U. Tubingen, Germany	MD 1948	462 S. Robert St., St. Paul, Minn.
AMBRUS, Milka	Eberhard-Karls U. Tubingen, Germany	MD 1948	Midway Hosp., St. Paul, Minn.
ANDERSON, Jack G.	Marquette U.	MD 1951	Mpls. Gen. Hosp., Minneapolis, Minn.
BALEISIS, Peter	U. of Dusseldorf	MD 1949	700 W. Broadway, Minneapolis, Minn.
BARLOW, Loren Call	Northwestern U.	MD 1951	102-110 2nd Ave. S.W., Rochester, Minn.
BOZANIC, Milos S.	Ludwig Maximilian U. Munich, Germany "Arzt"	1948	St. Joseph's Hosp., St. Paul, Minn.
CONNAUGHTON, Patrick Alex	St. Louis U., Mo.	MD 1951	Mpls. Gen. Hosp., Minneapolis, Minn.
DATESMAN, Robert Wallace	U. of Pennsylvania	MD 1951	USAH, 3444 ASU, Camp Stewart, Ga.
DE WEESE, Jr., Robert Cornell	U. of Colorado	MD 1952	St. Cloud Nat. Bk. Bldg., St. Cloud, Minn.
FEINBERG, Milton	U. of Illinois	MD 1941	2215 Plymouth Ave. N., Mpls., Minn.
FOLEY, III, Philip James	Northwestern U.	MD 1951	102-110 2nd Ave. S.W., Rochester, Minn.
GLENNY, Jr., William Robb	U. of Minnesota	MD 1953	Box 308, Osseo, Minn.
GREENWOLD, Warren Eldon	U of Chicago	MD 1946	102-110 2nd Ave. S.W., Rochester, Minn.
HEPBURN, Allan Lockwood	U. of Alberta	MD 1948	102-110 2nd Ave. S.W., Rochester, Minn.
JOHNSON, Marvin Waldemar	U. of Minnesota	MD 1953	Milaca, Minn.
KARLINSKY, William	U. of Manitoba	MD 1945	644 Niagara St., Winnipeg, Man., Can.
KARNOPP, Katherine	U. of Oregon	MD 1952	Glen Lake San., Oak Terrace, Minn.
KONIG, Theodore John	U. of Minnesota	MD 1952	Veterans Adm. Hosp., Minneapolis, Minn.
LASZEWSKI, Franz von Zelberschwecht	Westphalen Wilhelms U. Munster, Germany	MD 1929	St. Joseph's Hospital, St. Paul, Minn.
LYSYJ, Anatol	Ludwig Maximilian U. Munich, Germany	MD 1950	624 W. 54th St. S., Minneapolis, Minn.
McCULLOCH, Charles Fraser	Queens Univ.	MD 1945	102-110 2nd Ave. S.W., Rochester, Minn.
MORISSETTE, Gaston	Laval Univ.	MD 1950	102-110 2nd Ave. S.W., Rochester, Minn.
PLACE, Virgil Alan	Johns Hopkins U.	MD 1948	102-110 2nd Ave. S.W., Rochester, Minn.
PROSHEK, Lumir Charles	U. of Iowa	MD 1949	Mpls. Gen. Hosp., Minneapolis, Minn.
RICHARDS, Albert MacDonell	U. of Minnesota	MD 1948	Heart Hosp., U. of Minn., Mpls., Minn.
RYZEN, Nicholas	U. of Tennessee	MD 1951	Anoka State Hosp., Anoka, Minn.
SABANAS, Alvina Olga	U. Vytautas the Great Kaunas, Lithuania	MD 1939	102-110 2nd Ave. S.W., Rochester, Minn.
SMITH, Dale Compton	U. of Kansas	MD 1945	102-110 2nd Ave. S.W., Rochester, Minn.
SOUIRES, Franklin John	U. of Manitoba	MD 1945	102-110 2nd Ave. S.W., Rochester, Minn.
THOMPSON, Richard Eugene	Northwestern U.	MD 1951	NPBA Hosp., Glendive, Mont.
TORKELSON, Leonard Beck	U. of Minnesota	MD 1953	St. Joseph's Hosp., St. Paul, Minn.
TSAI, Shih Hao	National Med. Col. Shanghai, China	MD 1941	Glen Lake San., Oak Terrace, Minn.
VOIGHT, Allan Earl	U. of Oregon	MD 1951	Mpls. Gen. Hospital, Minneapolis, Minn.
VREELAND, Oliver Henry	U. of Oregon	MD 1952	102-110 2nd Ave. S.W., Rochester, Minn.
WESTPHAL, Paul Harry	U. of Minnesota	MD 1953	Gettysburg, S. D.
WOYDA, William Charles	U. of Minnesota	MD 1953	Box 609, Elk River, Minn.

Reciprocity Candidates

Name	*School*		*Address*
BOSSARD, John Wesley	U. of Maryland	MD 1951	102-110 2nd Ave. S.W., Rochester, Minn.
BOYD, George Knox	U. of Pittsburgh	MD 1951	102-110 2nd Ave. S.W., Rochester, Minn.
BURGERMAN, Arthur	Indiana U.	MD 1948	102-110 2nd Ave. S.W., Rochester, Minn.
BUTLER, Jr., Lewell Colbert	Louisiana State U.	MD 1946	102-110 2nd Ave. S.W., Rochester, Minn.
CAYCE, John Hill	U. of Texas	MD 1947	102-110 2nd Ave. S.W., Rochester, Minn.
CLINGER, Edwin James	Ohio State U.	MD 1946	102-110 2nd Ave. S.W., Rochester, Minn.
COOPER, Milton	Cornell U.	MD 1948	102-110 2nd Ave. S.W., Rochester, Minn.
DURAN, Robert Jackson	U. of Oklahoma	MD 1947	102-110 2nd Ave. S.W., Rochester, Minn.
FYKE, Jr., Frazier Earl	U. of Pennsylvania	MD 1947	102-110 2nd Ave. S.W., Rochester, Minn.
HUEBERT, Dan W.	U. of Kansas	MD 1946	26 Washington, Hutchinson, Minn.
JACKSON, Robert Carl	U. of Wisconsin	MD 1945	102-110 2nd Ave. S.W., Rochester, Minn.
JOHNSON, Robert Hugo	U. of Nebraska	MD 1952	210 3rd St. S.W., Little Falls, Minn.
KILMER, Warren Lee	Indiana U.	MD 1952	State Bk. Bldg., Inter. Falls, Minn
LAW, William McConnell	Med. Col. of Va.	MD 1948	102-110 2nd Ave. S.W., Rochester, Minn.
LEE, Philip Randolph	Stanford U.	MD 1948	102-110 2nd Ave. S.W., Rochester, Minn.
LOCHRIDGE, Jr., Edwin Payne	U. of Georgia	MD 1949	102-110 2nd Ave. S.W., Rochester, Minn.
LOWE, James Cecil	U. of Tennessee	MD 1943	102-110 2nd Ave. S.W., Rochester, Minn.
MacLEAN, Donald Beacom	Wayne Univ.	MD 1950	102-110 2nd Ave. S.W., Rochester, Minn.
PRICE, Jr., William Edmund	U. of Oklahoma	MD 1946	412 Delaware St. S.E., Minneapolis, Minn.
PURO, Henry Evert	Standford U.	MD 1949	102-110 2nd Ave. S.W., Rochester, Minn.
QUINONES, Rafael Enrique	U. of Tennessee	MD 1949	102-110 2nd Ave. S.W., Rochester, Minn.
REDELFS, John Wright	U. of Nebraska	MD 1945	102-110 2nd Ave. S.W., Rochester, Minn.
RICKMAN, James H.	U. of Louisville	MD 1946	102-110 2nd Ave. S.W., Rochester, Minn.
RYAN, Robert Frank	Stanford U.	MD 1947	102-110 2nd Ave. S.W. Rochester, Minn.
SCHWARTZ, Carl Alfred	U. of Pennsylvania	MD 1947	102-110 2nd Ave. S.W., Rochester, Minn.
SCOTT, Jr., Charles	Emory Univ.	MD 1949	102-110 2nd Ave. S.W., Rochester, Minn.
SMITH, Jr. Fred Newman	Tulane Univ.	MD 1947	102-110 2nd Ave. S.W., Rochester, Minn.
TAYLOR, Harry Edwin	Temple Univ.	MD 1949	102-110 2nd Ave. S.W., Rochester, Minn.

National Board Candidates

Name	*School*		*Address*
EDELMANN, Robert Benedict	Long Island Col. Med.	MD 1947	102-110 2nd Ave. S.W., Rochester, Minn.
GARAMELLA, Joseph John	Georgetown U.	MD 1945	Veterans Adm. Hosp., Minneapolis, Minn.
HARTRIDGE, Virginia Bishop	Woman's Med. Col. of Pa.	MD 1950	102-110 2nd Ave. S.W., Rochester, Minn.
O'NEIL, Richard Lawson	Boston Univ.	MD 1947	102-110 2nd Ave. S.W., Rochester, Minn.
WALDMANN, Edward Bernard	Creighton Univ.	MD 1948	102-110 2nd Ave. S.W., Rochester, Minn.
WANNAMAKER, Lewis William	Duke Univ.	MD 1946	Dept. Ped., U. of Minn., Hosp., Minneapolis, Minn.
WINN, Jr., William Edward	Harvard Univ.	MD 1949	102-110 2nd Ave. S.W., Rochester, Minn.

PHYSICIANS LICENSED JULY 17, 1953

June, 1953, Examination

Name	*School*		*Address*
ANDERSON, Richard William	U. of Minnesota	MD 1953	St. Joseph's Hosp., St. Paul, Minn.
ARNESON, Genevieve A.	U. of Minnesota	MD 1953	U.S.P.H.S. Hosp., New Orleans, La.
BAKKE, Arnold Clifford	U. of Minnesota	MD 1953	St. Joseph's Hosp., St. Paul, Minn.
BASINGER, Harold Preston	U. of Minnesota	MD 1953	Williamsport Hosp., Williamsport, Pa.
BEARMAN, Morton	U. of Minnesota	MD 1953	Ancker Hosp., St. Paul, Minn.
BERG, Arnold Martin	U. of Minnesota	MD 1953	Mpls. Gen. Hosp., Minneapolis, Minn.
BOWLIN, Paul Frederick	U. of Minnesota	MD 1953	Oakland Naval Hosp., Oakland, Calif.
BRETZKE, Carl Otto	U. of Minnesota	MD 1953	Greenbush, Minn.
CAMMOCK, Earl Elarth	U. of Minnesota	MD 1953	Harborview Hosp., Seattle, Wash.
CARR, William Johnson	U. of Minnesota	MD 1953	338 LaSalle Bldg., Minneapolis, Minn.
COUNTRYMAN, Ethlyn	U. of Minnesota	MD 1953	San Francisco Hosp., San Francisco, Cal.
CROUCH, Boyden L.	U. of Kansas	MD 1946	102-110 2nd Ave. S.W., Rochester, Minn.
DEMARAIS, Francis Emery	U. of Minnesota	MD 1952	2508 Colfax Ave. S., Minneapolis, Minn.
DOBSON, Mervin Wilbert	U. of Manitoba	MD 1944	Medical Center, Mankato, Minn.
DOXSEE, George Charles	U. of Iowa	MD 1952	30 N.W. 1st St., Chisholm, Minn.
DUNN, Robert Clarence	U. of Minnesota	MD 1953	St. Joseph's Hosp., St. Paul, Minn.
FILIPOVICH, Orest Nicholas	U. of Minnesota	MD 1953	Mpls. Gen. Hosp., Minneapolis, Minn.
FRENCH, Bayard Taylor	U. of Iowa	MD 1952	2229 3rd Ave. E., Hibbing, Minn.
FUSARO, Ramon Michael	U. of Minnesota	MD 1953	Mpls. Gen. Hosp., Minneapolis, Minn.
GODFREY, H. Wilson	U. of Minnesota	MD 1953	Mpls. Gen. Hosp., Minneapolis, Minn.
GOODCHILD, William Russell	U. of Minnesota	MD 1953	Mt. Sinai Hosp., Minneapolis, Minn.
GRANQUIST, Richard David	U. of Minnesota	MD 1953	St. Luke's Hosp., Duluth, Minn.
HABERLAND, Lyle Freeman	U. of Minnesota	MD 1953	Sacramento Co. Hosp., Sacramento, Cal.
HAGEDORN, Jr., Max Boykin	Loyola U.	MD 1949	Veterans Adm. Hosp., Minneapolis, Minn.
HALVERSON, William Gearey	U. of Minnesota	MD 1953	St. Mary's Hosp., Duluth, Minn.

Name	*School*		*Address*
HANSON, Daniel James	U. of Minnesota	MD 1953	Mercy Hosp., Toledo, Ohio
HANSON, Harold Walden	U. of Minnesota	MD 1953	St. Joseph's Hosp., St. Paul, Minn.
HEID, James K.	U. of Minnesota	MD 1953	Madigan Army Gen. Hosp., Fort Lewis, Wash.
HEINZERLING, Carl Robert	U. of Illinois	MD 1953	Mt. Sinai Hosp., Minneapolis, Minn.
HOPPERSTAD, Jule Jerome	U. of Minnesota	MD 1953	St. Mary's Hosp., Minneapolis, Minn.
HOYT, Charles Sherman	U. of Minnesota	MD 1953	Valley Forge Army Hosp., Phoenixville, Pa.
HUFF, John Stephen	U. of Minnesota	MD 1953	Memorial Hosp., Phoenix, Ariz.
JOHNSON, Gordon Erik	U. of Minnesota	MD 1953	St. Luke's Hosp., Duluth, Minn.
JUDE, James Roderick	U. of Minnesota	MD 1953	Johns Hopkins Hosp., Baltimore, Md.
KADESKY, Harold Baird	U. of Minnesota	MD 1953	2300 W. 50th St., Minneapolis, Minn.
KALLENBACH, Rudolf Werner	U. of Minnesota	MD 1953	703 Armstrong Blvd. S., St. James, Minn.
KANE, John Francis	Marquette U.	MD 1952	117½ S. 1st St., Montevideo, Minn.
KAPLAN, Martin Jack	U. of Minnesota	MD 1953	Michael Reese Hosp., Chicago, Ill.
KARON, Everett Howard	U. of Minnesota	MD 1953	U. of Minn. Hosp., Minneapolis, Minn.
KATKOV, Harold	U. of Minnesota	MD 1953	Mpls. Gen. Hosp., Minneapolis, Minn.
KENT, James Roy	U. of Minnesota	MD 1953	St. Luke's Hosp., Duluth, Minn.
KETOLA, Tauno Everett	U. of Minnesota	MD 1953	Highland Alameda Co. Hosp., Oakland, Cal.
KIRSCHBAUM, Thomas Harry	U. of Minnesota	MD 1953	U. of Minn. Hosp., Minneapolis, Minn.
LARSON, Earl Russell	U. of Minnesota	MD 1953	King Co. Hosp., Seattle, Wash.
LENTON, John David	Creighton U.	MD 1953	Creighton Memorial-St. Joseph's Hosp., Omaha, Neb.
LEUZINGER, Donn Ellis	U. of Minnesota	MD 1953	U.S.P.H.S. Hosp., Seattle, Wash.
LINDALL, Dale Regnar	U. of Minnesota	MD 1953	Tripler Army Hosp., Honolulu, Hawaii
LINNELL, Leonard S.	U. of Minnesota	MD 1953	St. Luke's Hosp., Duluth, Minn.
LITIN, Robert Beryl	U. of Minnesota	MD 1953	Santa Clara Co. Hosp., San Jose, Cal.
LITTLE, Bruce Raymond	U. of Minnesota	MD 1953	Santa Monica Hosp., Santa Monica, Cal.
MAGUIRE, Gerald Eugene	U. of Minnesota	MD 1953	Sacramento Co. Hosp., Sacramento, Cal.
MARK, Aaron Louis	U. of Minnesota	MD 1953	Mpls. Gen. Hosp., Minneapolis, Minn.
MARTIN, Jr., George Burnie	U. of Illinois	MD 1950	117½ S. 1st St., Montevideo, Minn.
McFARLANE, Donald Ray	U. of Minnesota	MD 1953	Mpls. Gen. Hosp., Minneapolis, Minn.
McMAHAN, Robert Oliver	U. of Minnesota	MD 1953	U. S. Naval Hosp., Oakland, Cal.
MEAGHER, Thomas Arthur	U. of Minnesota	MD 1953	King Co. Hosp. System, Seattle, Wash.
MELBY, James Christian	U. of Minnesota	MD 1953	U. of Minn. Hosp., Minneapolis, Minn.
MELLER, Maurycy	U. of Parma, Italy	MD 1937	First Nat. Bk. Bldg., Brainerd, Minn.
MILLER, Harold Dawes	U. of Minnesota	MD 1953	Mpls. Gen. Hosp., Minneapolis, Minn.
MURN, Thomas Gregory	U. of Minnesota	MD 1953	Mpls. Gen. Hosp., Minneapolis, Minn.
NACHTIGAL, Fred C.	U. of Minnesota	MD 1953	Veterans Adm. Hosp., Long Beach, Cal.
NAHAS, Gabriel	U. of Toulouse, France	MD 1944	315 Millard Hall, U. of Minn., Minneapolis, Minn.
NASH, Eldore Bentley	U. of Minnesota	MD 1953	Memorial Hosp., Phoenix, Ariz.
NEILSON, William George	U. of Illinois	MD 1952	102-110 2nd Ave. S.W., Rochester, Minn.
NORQUIST, Joseph Louis	U. of Minnesota	MD 1953	Walter Reed Army Hosp., Washington, D. C.
ODLAND, Donald Mark	Marquette U.	MD 1943	Mpls. Gen. Hosp., Minneapolis, Minn.
OLSON, Gregory Mathias	U. of Minnesota	MD 1953	Sacramento Co. Hosp., Sacramento, Cal.
PARSONS, Roger Alan	U. of Minnesota	MD 1953	St. Joseph's Hosp., St. Paul, Minn.
PERKINS, Douglass Everal	U. of Minnesota	MD 1953	614 Broadway, Alexandria, Minn.
POPADIUK, Peter	Friedrich Alex. U. Erlangen, Germany	MD 1948	St. Mary's Hosp., Minneapolis, Minn.
PRESTON, Frank Stebbins	U. of Minnesota	MD 1953	St. Vincent's Hosp., New York, N. Y.
PUGSLEY, Robert Daniel	U. of Minnesota	MD 1953	Swedish Hosp., Seattle, Wash.
RAMLOW, Ralph Marlyn	U. of Minnesota	MD 1953	Ancker Hosp., St. Paul, Minn.
RICHMAN, Harold Gene	U. of Minnesota	MD 1953	Barnes Hosp., St. Louis, Mo.
RICHTER, David James	U. of Illinois	MD 1951	6th Ave & 9th St. N., Virginia, Minn.
ROCKWELL, Winthrop R.	U. of Minnesota	MD 1953	Tripler Army Hosp., Honolulu, Hawaii
RUDIE, William Donald	U. of Minnesota	MD 1953	St. Mary's Hosp., Duluth, Minn.
SCHAFER, Thomas Leo	U. of Minnesota	MD 1953	Detroit Receiving Hosp., Detroit, Mich.
SCHMIDT, Jr., Erwin Rudolph	U. of Wisconsin	MD 1952	St. Luke's Hosp., Duluth, Minn.
SCHNEIDER, James A.	U. of Minnesota	MD 1953	Gorgas Hosp., Panama, Canal Zone
SCHREINER, Leon H.	Northwestern U.	MD 1950	102-110 2nd Ave. S.W., Rochester, Minn.
SETZER, Jr., Hobert Joseph	U. of Minnesota	MD 1953	Mpls. Gen. Hosp., Minneapolis, Minn.
SHIELDS, Jack William	Stanford U.	MD 1948	102-110 2nd Ave. S.W., Rochester, Minn.
SISLER, David Allen	U. of Minnesota	MD 1953	Grand Rapids, Minn.
SONTAG, David W.	U. of Minnesota	MD 1953	Veterans Adm. Hosp., Long Beach, Cal.
SUNBERG, Ruth Dorothy	U. of Minnesota	MD 1953	U. of Minn. Hosp., Minneapolis, Minn.
TAYLOR, Robert Owen	U. of Minnesota	MD 1953	San Diego Co. Gen. Hosp., San Diego, Cal.
TEYNOR, Joseph William	U. of Minnesota	MD 1953	U. S. Naval Hosp., Great Lakes, Ill.
THAL, Sam Harold	U. of Washington	MD 1951	102-110 2nd Ave. S.W., Rochester, Minn.
TIFFANY, Francis Buchanan	U. of Minnesota	MD 1953	St. Luke's Hosp., New York, N. Y.
ULRICH, Christian Andrew	Northwestern U.	MD 1949	102-110 2nd Ave. S.W., Rochester, Minn.
VERMUND, Halvor	U. of Oslo, Norway	MD 1943	U. of Minn. Hosp., Minneapolis, Minn.
VORIS, David Clarence	Toulouse, France		102-110 2nd Ave. S.W., Rochester, Minn.

WALDRON, John Fossen	U. of Minnesota	MD 1953	Mpls. Gen. Hosp., Minneapolis, Minn.
WALTER, William Edward	U. of Minnesota	MD 1953	Mpls. Gen. Hosp., Minneapolis, Minn.
WATERS, Alvin W.	U. of Minnesota	MD 1953	Ancker Hosp., St. Paul, Minn.
WATSON, William Richard	U. of Minnesota	MD 1953	Fitzsimons Army Hosp., Denver, Colo.
WEGNER, Marwood Emery	U. of Minnesota	MD 1953	U.S.P.H.S. Hosp., Staten Island, N. Y.
WENZEL, James Brandrup	U. of Minnesota	MD 1953	Memorial Hosp., Phoenix, Ariz.
WILLIAMS, Paul A.	U. of Minnesota	MD 1953	Tripler Army Hosp., Honolulu, Hawaii
WILLIAMS, Richard Albert	U. of Iowa	MD 1952	Rothsay, Minn.
ZELLMER, Richard Edward	U. of Minnesota	MD 1953	U.S.P.H.S. Hosp., Baltimore, Md.
ZEMMERS, Roberts	U. of Latvia Riga, Latvia	"Physician" 1926	St. Luke's Hospital, Duluth, Minn.

Reciprocity Candidates

Name	*School*		*Address*
BEITHON, Paul Jule	U. of Nebraska	MD 1952	Miller Hosp., St. Paul, Minn.
BURLEIGH, Jr., Edward Gilbert	Louisiana U.	MD 1952	More Hosp., Eveleth, Minn.
CALVERT, Wilson Clark	Northwestern U.	MD 1951	102-110 2nd Ave. S.W., Rochester. Minn.
COLLINS, Jr., Robert Aaron	U. of Georgia	MD 1948	102-110 2nd Ave. S.W., Rochester, Minn.
DYER, John Allen	U. of Pennsylvania	MD 1947	102-110 2nd Ave. S.W., Rochester, Minn.
JOHNSON, Morris Blaine	U. of Nebraska	MD 1952	Miller Hosp., St. Paul, Minn.
LEITSCHUH, Robert Bernard	Marquette U.	MD 1946	Sleepy Eye, Minn.
LEWIS, Jr., Royce Clay	Tulane U.	MD 1946	102-110 2nd Ave. S.W., Rochester, Minn.
McCALL, J. Preston	Baylor U.	MD 1948	102-110 2nd Ave. S.W., Rochester. Minn.
McKAY, Walter Edward	Indiana U.	MD 1948	102-110 2nd Ave. S.W., Rochester, Minn.
MOBLEY, Jack Ervin	Vanderbilt U.	MD 1948	102-110 2nd Ave. S.W., Rochester, Minn.
RACE, George Alexander	Temple U.	MD 1943	102-110 2nd Ave. S.W., Rochester, Minn.
SILAS, Ralph Maurice	U. of Michigan	MD 1943	Veterans Adm. Bldg., No. 18, Ft. Snelling, St. Paul, Minn.
SIMPSON, Joseph Ralph	Louisiana State U.	MD 1947	102-110 2nd Ave. S.W., Rochester, Minn.
SMITH, Jr., Ross Haugh	U. of Pittsburgh	MD 1951	102-110 2nd Ave. S.W., Rochester, Minn.
SOMMERNESS, M. Duane	Temple U.	MD 1945	Willmar State Hosp., Willmar, Minn.
THATCHER, Wilbur Clayton	U. of Iowa	MD 1933	Physicians Bldg., Fort Dodge, Ia.
WALLACE, III, Alexander	Baylor U.	MD 1948	102-110 2nd Ave. S.W., Rochester, Minn.
WILLER, Stanley Hermann	Ohio State U.	MD 1948	102-110 2nd Ave. S.W., Rochester, Minn.

National Board Candidates

Name	*School*		*Address*
ALEXANDER, Jr., John Dewey	U. of Pennsylvania	MD 1949	102-110 2nd Ave. S.W., Rochester, Minn.
ALTHAUSEN, Jr., Theodore L.	U. of California	MD 1947	65 S. 10th St., Minneapolis, Minn.
BRIDGE, Allyn Gustave	Yale U.	MD 1948	301 Kenwood Pkwy., Minneapolis, Minn.
De WALL, Richard Allison	U. of Minnesota	MD 1953	1833 2nd Ave., Anoka, Minn.
FARLEY, Frank Giles	Syracuse U.	MD 1945	2229 3rd Ave. E., Hibbing, Minn.
GROCH, Sigmund Noel	Tufts Col. Med. Sch.	MD 1949	102-110 2nd Ave. S.W., Rochester, Minn.
LINDNER, Janus Casimir	Yale U.	MD 1950	505 Nicollet Ave., Minneapolis, Minn.
MARR, Jr., Norval Mason	Cornell U.	MD 1948	102-110 2nd Ave. S.W., Rochester, Minn.
NICHOLSON, Richard Waldo	Harvard U.	MD 1948	Heron Lake, Minn.
RUTLEDGE, John Boyden	U. of Minnesota	MD 1952	1050 Lake Ave., Detroit Lakes, Minn.
SKRDLA, Willard Blake	U. of Nebraska	MD 1948	102-110 2nd Ave. S.W., Rochester, Minn.

PHYSICIANS LICENSED NOVEMBER 13, 1953

October, 1953, Examination

Name	*School*		*Address*
ALLBAUGH, Robert Dean	Washington U., Mo.	MD 1952	Mpls. Gen. Hosp., Minneapolis, Minn.
AMICK, Lawrence Douglas	U. of Iowa	MD 1945	Sac City, Iowa
BONNET, John David	Johns Hopkins U.	MD 1952	102-110 2nd Ave. S.W., Rochester, Minn.
BOTHAM, Richard James	U. of Wisconsin	MD 1952	102-110 2nd Ave. S.W., Rochester, Minn.
BRUNSTING, Jr., Louis Albert	U. of Michigan	MD 1952	102-110 2nd Ave. S.W., Rochester, Minn.
CAMPAGNA, Mario Joseph	U. of Oregon	MD 1952	102-110 2nd Ave. S.W., Rochester, Minn.
CARPER, John Mark	Jefferson Med. Col.	MD 1952	102-110 2nd Ave. S.W., Rochester, Minn.
CHRISTU, Chris Nicholas	Louisiana State U.	MD 1952	102-110 2nd Ave. S.W., Rochester, Minn.
COMESS, Morton Seymour	U. of Illinois	MD 1952	102-110 2nd Ave. S.W., Rochester, Minn.
CUMMISKEY, Paul Vernon	U. of Minnesota	MD 1953	104 S. 4th St., Mankato, Minn.
DEVANNEY, Louis Raynor	Northwestern U.	MD 1939	102-110 2nd Ave. S.W., Rochester, Minn.
DOAN, Robert English	U. of Minnesota	MD 1953	Mpls. Gen. Hosp., Minneapolis, Minn.
DOUGLASS, Jesse Ellsworth	U. of Minnesota	MD 1952	2325 Stinson Blvd., Minneapolis, Minn.
HENDRICKSON, Russell Robert	U. of Minnesota	MD 1953	Cook Co. Hosp., Chicago, Ill.
HIGGINS, John Alden	U. of Minnesota	MD 1953	102-110 2nd Ave. S.W., Rochester, Minn.
HOWARD, David Lloyd George	U. of Toronto	MD 1948	102-110 2nd Ave. S.W., Rochester, Minn.
HUNTER, Samuel Wynne	U. of Rochester	MD 1947	U. of Minn. Hosp., Minneapolis, Minn.
JOHNSON, David Arthur	U. of Minnesota	MD 1953	Mt. Sinai Hosp., Minneapolis, Minn.
LOUGHEED, Lawrence Edward	U. of Manitoba	MD 1951	102-110 2nd Ave. S.W., Rochester. Minn.

Name	*School*		*Address*
MAHLE, James Parker	U. of Minnesota	MD 1953	St. Joseph's Hosp., St. Paul, Minn.
McSWEENY, II, Austin J.	Loyola U.	MD 1949	102-110 2nd Ave. S.W., Rochester, Minn.
MORROW, Jr., George William	U. of Illinois	MD 1952	102-110 2nd Ave. S.W., Rochester, Minn.
MOULTON, Keith Buell	U. of Minnesota	MD 1953	St. Joseph's Hosp., St. Paul, Minn.
NORDLUND, Mildred Eva	U. of Chicago—Rush	MD 1929	Cass Lake, Minn.
NORMAN, Jr., Mark Lewis	Marquette U.	MD 1952	1764 Grand Ave., St. Paul, Minn.
OLWIN, Thomas Kent	U. of Oregon	MD 1952	Ancker Hosp., St. Paul, Minn.
OZOLINS, Marta Birzins	U. of Latvia Riga, Latvia	MD 1927	274 Iglehart Ave., St. Paul, Minn.
PEAKE, Ronald Alan	Indiana U.	MD 1952	102-110 2nd Ave. S.W., Rochester, Minn.
PITTELKOW, Robert Bernard	Marquette U.	MD 1952	102-110 2nd Ave. S.W., Rochester, Minn.
POPOVICH, Dragojla, nee'Ostojich	U. of Belgrade Belgrade, Yugoslavia	MD 1939	Northwestern Hosp., Minneapolis, Minn.
RAILE, Richard Bayles	U. of Utah	MD 1950	1354 E. Maynard Dr., St. Paul, Minn.
RENO, III, George Louis	St. Louis U.	MD 1951	102-110 2nd Ave. S.W., Rochester, Minn.
SAFIRESCU, Sorin Radu	U. of Bologna, Italy	MD 1950	Fairview Hosp., Minneapolis, Minn.
SEYBOLD, Herbert McCelvey	U. of Texas	MD 1951	102-110 2nd Ave. S.W., Rochester, Minn.
STEWART, Allan H.	Geo. Wash. U., D.C.	MD 1952	102-110 2nd Ave. S.W., Rochester, Minn.
SVARE, Gerhart Trygve	U. of Wash., Seattle	MD 1951	102-110 2nd Ave. S.W., Rochester, Minn.
TAINTOR, Ronald W.	U. of Louisville	MD 1952	Ancker Hosp., St. Paul, Minn.
TERRY, Howard Lincoln	Northwestern U.	MD 1944	Anoka State Hosp., Anoka, Minn.
THOMPSON, Murray Cornwall	U. of Western Ont.	MD 1950	102-110 2nd Ave. S.W., Rochester, Minn.
TRAYNOR, Jr., Mack Vincent	Northwestern U.	MD 1950	102-110 2nd Ave. S.W., Rochester, Minn.
VANDEVER, Harry Wallace	U. of Tennessee	MD 1948	102-110 2nd Ave. S.W., Rochester, Minn.
VEINBERGS, Arnolds	U. of Latvia Riga, Latvia	"Physician" 1940	Swedish Hosp., Minneapolis, Minn.
ZIMERING, Sabina Szwarc	Ludwig Maximilian U. Munich, Germany	MD 1950	2741 Chicago Ave., Minneapolis, Minn.

Reciprocity Candidates

Name	*School*		*Address*
BETTS, Charles Samuel	Vanderbilt U.	MD 1951	102-110 2nd Ave. S.W., Rochester, Minn.
BLATTSPIELER, Lucien H.	U. of Nebraska	MD 1946	Worthington, Minn.
CANTRELL, Glenn	U. of So. Calif.	MD-1947	102-110 2nd Ave. S.W., Rochester, Minn.
CHRISTIANSON, Herbert B.	Marquette U.	MD 1939	102-110 2nd Ave. S.W., Rochester, Minn.
CONGDON, Gordon Hall	U. of Chicago	MD 1940	102-110 2nd Ave. S.W., Rochester, Minn.
CREAMER, Dick Oliver	Baylor U.	MD 1950	102-110 2nd Ave. S.W., Rochester, Minn.
DeLAND, II, Frank Howard	U. of Louisville	MD 1952	102-110 2nd Ave. S.W., Rochester, Minn.
ELLIOTT, Dean Cook	Ohio State U.	MD 1946	102-110 2nd Ave. S.W., Rochester, Minn.
ELLWOOD, Jr., Paul Murdock	Stanford U.	MD 1953	U. of Minn. Hosp., Minneapolis, Minn.
FAUCETT, Robert Lynn	U. of Kansas	MD 1944	102-110 2nd Ave. S.W., Rochester, Minn.
FONTANA, Robert Scott	St. Louis U.	MD 1949	102-110 2nd Ave. S.W., Rochester, Minn.
FOX, LeRoy John	Northwestern U.	MD 1945	NPBA Hosp., St. Paul, Minn.
HELMHOLZ, Jr., Henry F.	Johns Hopkins U.	MD 1937	102-110 2nd Ave. S.W., Rochester, Minn.
HONATH, Donald Herbert	Wayne U.	MD 1947	111 W. Main St., Owatonna, Minn.
KIRK, Jr., Thomas Allen	U. of Virginia	MD 1949	102-110 2nd Ave. S.W., Rochester, Minn.
LEAVENS, Milam Edmund	Baylor U.	MD 1948	102-110 2nd Ave. S.W., Rochester, Minn.
LIMBECK, Donald Arthur	U. of Nebraska	MD 1950	State Hosp., Fergus Falls, Minn.
LOES, Louis Augustine	U. of Iowa	MD 1947	1225 St. Germain St., St. Cloud, Minn.
MILLER, John William	U. of Louisville	MD 1951	102-110 2nd Ave. S.W., Rochester, Minn.
MORGAN, Edward Raymond	Tulane U.	MD 1950	102-110 2nd Ave. S.W., Rochester, Minn.
MUDD, Robert Hiden	U. of Pennsylvania	MD 1951	102-110 2nd Ave. S.W., Rochester, Minn.
PIFER, Lawrence Harrison	U. of Indiana	MD 1952	1218 N. 3rd St., St. Joseph, Mo.
REIFSNYDER, III, Wm. Henry	Jefferson Med. Col.	MD 1951	102-110 2nd Ave. S.W., Rochester, Minn.
RITTER, Donald Glenn	Indiana U.	MD 1952	102-110 2nd Ave. S.W., Rochester, Minn.
TERRY, Jr., Howard Richards	State U. of N. Y.	MD 1947	102-110 2nd Ave. S.W., Rochester, Minn.
TURNER, Jr., John Cooper	Tulane U.	MD 1951	102-110 2nd Ave. S.W., Rochester, Minn.
WALKER, Jr., Harry Charles	Med. Col. of Va.	MD 1951	U. of M. Grad. Sch., Minneapolis, Minn.
WALKER, Jr., Jack Andrew	Southwestern Med. Col.	MD 1948	102-110 2nd Ave. S.W., Rochester, Minn.

National Board Candidates

Name	*School*		*Address*
BIRKHEAD, Newton Charles	U. of Pennsylvania	MD 1949	102-110 2nd Ave. S.W., Rochester, Minn.
BOTTI, John Dominic	U. of Pennsylvania	MD 1952	102-110 2nd Ave. S.W., Rochester, Minn.
BUIE, Jr., Louis Arthur	U. of Minnesota	MD 1952	102-110 2nd Ave. S.W., Rochester, Minn.
DOYLE, Jr., John B.	Geo. Wash. U.	MD 1952	102-110 2nd Ave. S.W., Rochester, Minn.
ENSRUD, Earl Richard	Northwestern U.	MD 1952	102-110 2nd Ave. S.W., Rochester, Minn.
FABI, Mario Nestor	Geo. Wash. U.	MD 1950	102-110 2nd Ave. S.W., Rochester, Minn.
FOX, Irwin J.	New York Med. Col.	MD 1951	102-110 2nd Ave. S.W., Rochester, Minn.
GLASS, Bernard	Ohio State U.	MD 1950	102-110 2nd Ave. S.W., Rochester, Minn.

Name	*School*		*Address*
GREENFIELD, Duane Leonard	U. of Maryland	MD 1946	833 S. Phillips Ave., Sioux Falls, S. D.
HADER, Marvin	New York Med. Col.	MD 1952	102-110 2nd Ave. S.W., Rochester, Minn.
HELME, William Beach	Loyola U.	MD 1951	102-110 2nd Ave. S.W., Rochester, Minn.
KOGL, Richard Conrad	U. of Minnesota	MD 1953	Veterans Adm. Hosp., Minneapolis, Minn.
LEIGH, James Arthur	Bowman Gray Sch. of Med.	MD 1952	606 N. 3rd St., East Grand Forks, Minn.
MAGNESS, John Lower	Geo. Wash. U., D. C.	MD 1950	102-110 2nd Ave. S.W., Rochester, Minn.
MISRAHY, George Albert	Harvard U.	MD 1948	State Hospital, Hastings, Minn.
O'HOLLERAN, Lloyd Stephen	Creighton U.	MD 1952	102-110 2nd Ave. S.W., Rochester, Minn.
PEMBERTON, Henry Walter	Geo. Wash. U.	MD 1952	102-110 2nd Ave. S.W., Rochester, Minn.
PETERSEN, Deane Alfred	U. of Nebraska	MD 1946	1409 Willow St., Minneapolis, Minn.
RANDALL, Alan Douglas	Tufts Col. Sch. Med.	MD 1950	102-110 2nd Ave. S.W., Rochester, Minn.
ROTHWELL, Walter Spray	Harvard U.	MD 1947	102-110 2nd Ave. S.W., Rochester, Minn.
SHRINER, John Franklin	Tulane U.	MD 1950	1826 Clearmont St., Mobile, Ala.
STEINMETZ, Rodney Dunlap	Northwestern U.	MD 1952	102-110 2nd Ave. S.W., Rochester, Minn.
TWEEDDALE, Duane Newton	Creighton U.	MD 1951	102-110 2nd Ave. S.W., Rochester, Minn.
UPDIKE, II, Edwin Hoyt	Columbia U.	MD 1949	102-110 2nd Ave. S.W., Rochester, Minn.
WEISBART, Myron Herbert	Columbia U.	MD 1950	Veterans Adm. Hosp., Minneapolis, Minn.
WILLIAMS, Winfield Tyler	U. of Minnesota	MD 1952	Veterans Adm. Hosp., Minneapolis, Minn.

MEDICAL STUDENT ENROLLMENTS

Says the *Journal of the American Medical Association*† disclosing the 53rd annual report on medical education of its Council on Medical Education and Hospitals, "enrollments in the country's 72 medical and seven basic science schools during 1952-53 totaled 27,688, or 2.3 per cent more than the 27,076 enrolled during 1951-52. The 5,668 students graduated during the last year exceed by 279, or 4.4 per cent, the previous record established in 1947, when at the termination of the wartime accelerated program several schools graduated more than one class. The estimated number of graduates for 1953-54, based on enrollments reported for senior classes in schools, is even greater—6,831." There was, however, a slight decrease in the size of the entering freshman class for the first time in five years—7,425, or 16 less than the record class of 1952-53.

In addition to those regularly enrolled as full-time students of medicine, medical schools had enrolled a total of 140 part-time and special students working toward an M.D. degree, and 407 students in internships that were a part of the degree requirements of the medical schools at which they were educated. There was also a total of 1,734 students from the United States enrolled in 72 foreign medical schools, located in 22 different countries.

There were 1,463 women in medical and basic science schools during 1952-53, eight less than last year, the report said. The women comprised 5.3 per cent of all students, as compared with 5.4 per cent last year. Women in freshmen classes totaled 399, against 394 last year. They constituted 6.1 per cent of all applicants to medical schools this year, compared with 5.6 per cent last year. The actual number of women graduates increased slightly—363, or 5.5 per cent of the total, as contrasted to 351 last year. The record number of women graduates—612, was set in 1949.

A total of 715 Negro students, 2.6 per cent of all enrollments, was reported.

The number of veterans enrolled in medical and basic science schools declined for the second year. There were 7,942 veterans enrolled, comprising 28.7 per cent of the total student body, as compared with 11,436, or 42.2 per cent, last year. There were 36 women veterans.

For the fourth consecutive year, the number of applicants for admission to medical schools continued to decrease, the report disclosed. During the year 1952-53, 16,763 individuals applied for admission, a decrease of 3,157 from 1951-52, and 5,516 less than the number who applied in 1950-51.

"This report has emphasized in recent years that the difficulty of gaining admission to medical schools has been grossly exaggerated," it was stressed.

"The statistics presented here amply confirm this exaggeration. The situation in the past year, when there were 2.6 applicants for each place in medical schools, was approximately comparable to the year 1929-30 when there were 2.6 applicants for each available place.

"One major factor is the disappearance of the large backlog of veterans who completed their premedical training following the termination of World War II. Another factor, one that is certain to cause serious concern among medical educators, is the sharp decrease in the number of applicants with superior academic records.

"While, again, one can only speculate as to the cause, it seems likely that this drop is in no small measure due to increasing competition for such talent from other professional fields."

Medical faculties and medical students alike show a continuing interest in education for family or general practice, the report pointed out. A high proportion of the medical schools sponsor one or more programs specifically designed to induce students to this type of practice.

The report also disclosed that physicians of this country are cognizant of the fact that medical education is a continuous process and that it is their responsibility to keep abreast of advances in the field of medicine. During 1952-53, 64,608 physicians attended 1,341 postgraduate courses.—Editorial, *New York State Journal of Medicine*, February 1, 1954.

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†J.A.M.A., 153:105 (Sept. 12) 1953.

American Medical Association

Proceedings of the House of Delegates

Clinical Meeting

St. Louis, Missouri, December 1 to 4, 1953

The Clinical Meeting of the American Medical Association's House of Delegates, December 1 to 4, 1953, in St. Louis. Missouri, was notable for an absence of the controversies and stormy debate that have characterized some of the meetings of recent years.

The delegates were chiefly concerned with the application, readjustment and implementation of programs and policies already initiated. They embarked upon few new undertakings, but to policies already adopted they gave strong support, and dissenting voices were few.

Several restatements of national policies were made for purposes of clarification. Among them were the Association's policies with respect to medical care for dependents of military personnel, also medical aspects of any program for universal military training which in the future may be passed by Congress. A new emphasis was likewise placed upon the established policy for extension of voluntary prepayment plans for medical and hospital service and the work of the Council on Medical Education and Hospitals was buttressed, in spite of some expressed dissatisfaction, in its expanded accreditation program and its participation in the so-called "Matching Intern Program."

A resolution clarifying membership status and a number of other progressive changes in the Constitution and By-Laws will be welcomed by the membership. Also the adoption of definite objectives for the new Committee on Alcoholism, initiated last year as part of the Council on Mental Health, and of a medical association policy for participating in a national blood bank.

As a result of the end of hostilities in Korea, the doctor-draft law ceased to be a major issue at this session, but the delegates approved the suggestion that Congress be asked to eliminate the draft of doctors entirely at its forthcoming session.

Dependent Policy Restated

So long as a defense army is maintained, however, the question of medical care for dependents of military personnel is likely to be an issue. Restatement of the policy laid down at the interim session in Denver was especially pointed up by consideration of the report of the Citizen's Advisory Commission on Medical Care of Dependents of Military Personnel, the so-called Moulton Commission, which favors continuing and even expanding such care by military establishments. The statement adopted by the House of Delegates declared that the recommendations of the Moulton Commission "would completely reverse the generally accepted position that civilian facilities, if available and adequate, should be utilized first and that the use of military facilities should be restricted to emergencies and to dependents overseas and in remote areas." The statement will now be sent to the Secretary of Defense, to the chairmen of Senate and House military affairs committees and to the Commission on Re-organization of the Executive Branch of the Government, the Hoover Commission. The fact that President Eisenhower in his State of the Union address to Congress subsequently asked for increased medical service for dependents of military personnel makes it certain that this issue will assume especial importance in the months to come.

The policy adopted with respect to any future program of universal military training which may be passed by Congress is in line with the stand on dependent care and with the vigorous opposition voiced at the June meeting against medical service for veterans with non-service-connected disabilities. All have as their objective the placing of definite limitations on the amount of medical service to be provided by the federal government which may otherwise soon expand to a point where the private system of medical service and the quality of care available to the public in general is seriously threatened.

Medical Students Should be Excluded

If any plans are being laid now for early adoption of universal military training, medical students should be excluded from consideration until after they are graduated from medical school, the delegates believe. Their policy also calls for pre-induction examinations and periodic check-ups during the reserve period to be performed by civilian physicians in order to avoid serious withdrawals from hard-pressed civilian medical services. It calls for rehabilitation and corrective treatment which might be found necessary in pre-induction examinations to be carried out, likewise, by civilian doctors in civilian institutions and it asks that trainees in any contemplated reserve corps be ruled ineligible for veterans' medical benefits unless their disabilities are actually connected with active military service.

The delegates proposed that a civilian board be set up nationally to insure a proper balance between civilian and military needs. In that connection, also, they approved an official request from the association that a physician be retained under the reorganization plan in the position of Assistant Secretary of Defense (health and medical). The position is held at the present time by Dr. Melvin A. Casberg.

In order to carry forward the campaign initiated by the American Medical Association last June against federal medical care for veterans with non-service-connected disabilities, with stipulated exceptions, each constituent association was urged by the delegates to create a special committee on veterans' medical service. Several regional conferences on the subject have already been held and they will be extended to all parts of the country to acquaint doctors with AMA policy, it was announced.

Membership Clarified

Many delegates were happy to have the somewhat confused membership picture in the American Medical Association cleared up, as far as delinquents are concerned. They approved the following resolution as a substitution for a resolution introduced by Dr. George Earl of the Minnesota delegation last June. The latter had proposed that delinquent AMA dues be cancelled for members of any constituent state association which should subsequently make AMA membership compulsory for all its members. The substitute resolution adopted at St. Louis reads as follows:

> Resolved, That any active member of the American Medical Association who failed to pay dues for the year 1950, and who was suspended for such delinquency, may be reinstated during the first six months of 1954 by the payment of 1954 dues, only. Should such an individual fail to pay his 1954 dues by July 1, 1954, he shall continue to be considered delinquent.

A study in membership classifications is now under way by the Board of Trustees, it was announced, which should help to eliminate future membership difficulties.

The proposal to extend medical and hospital insurance protection came in the form of a resolution requesting the Council on Medical Service to be ready with definite suggestions for implementing extensions at the 1954 clinical meeting. The resolution called attention to two important groups of citizens who are largely unprotected at the present time. For these, improved coverage could and should be provided. They are "(a) those individuals who suffer catastrophic or long-continued and highly expensive illness and whose financial resources are not adequate to meet the cost thereof; and (b) those citizens who have retired and are living on small incomes and who are not eligible under presently existing public or private plans."

It was pointed out that the need for such extensions is genuine and also that it represents a soft spot in current plans for insurance protection through which federal subsidies may find a ready entrance.

The delegates also added special emphasis at this session to a standing AMA policy involving all prepaid insurance contracts. The resolution was adopted as an emergency measure, and it specifically condemned all insurance contracts which classify any type of medical service as "hospital service." Pathology, radiology, anesthesiology and physiatry were cited as involved in infractions and violations of this established policy.

In that connection, it should also be noted that recommendations were adopted for careful studies to be made by the Council on Medical Service of all types of union labor medical plans, recognizing both the need for many such plans and the dangers involved in unwarranted extensions of original arrangements and especially emphasizing the fact that medical ethics apply to them with the same force as to any insurance contract.

Ethics Uppermost

Medical ethics came in for discussion and action in many phases and applications. Uppermost in the minds of many delegates were the public charges of unethical practices made against medical men in many popular magazines recently. Their concern and the concern of officers and trustees of the Association was reflected in several resolutions. One of these called upon the Board of Trustees to make a study of the actual extent and seriousness of the unethical conduct alleged in these articles. Another was embodied in an amendment of Chapter IV of the Constitution which was adopted at this meeting pursuant to a request made at the June session. It provides that the Judicial Council may censure or expel service, affiliate and honorary, as well as active members of the association, after due notice, for violations of the principles of medical ethics.

It may be of interest to note here that the resolution introduced by Indiana last June on the subject of federal policies governing the grants-in-aid program for care of crippled children in Indiana has been transmitted to the Department of Health, Education and Welfare where assurances have been given that it will receive prompt and careful attention. The special interest of this resolution, which involves the question of possible charges for those able to pay fees and which applies chiefly to states which do not follow the general grants-in-aid pattern, lies in the fact that it is the one that was hastily and mistakenly siezed upon by *Life* magazine last summer for its ill-natured editorial entitled "Watch It Doc!"

Special emphasis was placed on the principle that physicians may not ethically sell their services or accept a salary for them if they may be exploited in so doing. Decision as to the propriety of any given salary arrangement must depend, however, upon individual facts and circumstances of the case. In response to many inquiries, the Judicial Council re-emphasized the accepted principle that it is unethical for a physician to have a financial interest in a pharmacy in the area in which he conducts his professional activities and where he profits directly or indirectly from the sale of devices or remedies prescribed for his patients. The principle also applies in the case of rental of space to a pharmacy by a physician if the rental is for a percentage on a sliding scale of income.

Actual revision of the Principles of Medical Ethics is under way by the Judicial Council and the Council on Constitution and By-Laws, of which Dr. Louis A. Buie of Rochester is chairman, but waits upon results of studies and questionnaires now being circulated to the membership. Included in changes now under consideration is the proposal made at the June meeting for a change in recommendations for billing procedures where more than one physician participates in the care of the patient. Certain other preparatory steps have been completed and were approved, also, including a change in format and a change in the title which formerly read "Education Information Not Advertising" to read "The Relationship of the Physician to the Media of Public Information."

Bills Approved

Among bills due to come before Congress in 1954 were several which received official sanction of the delegates at this session. One was the Jenkins-Keogh bill which opposes compulsory social security coverage for self-employed persons. The delegates favored, instead, income tax deductions as proposed in the bill, which will permit such persons to build up their own coverage. Another favors transfer of functions, responsibilities and duties of the Department of the Interior and the Bureau of Indian Affairs as they relate to health to the United States Public Health Service; also authorization of admission to United States Public Health hospitals of persons committed by state courts who are Service beneficiaries or narcotic addicts. Still another strongly favored deduction without limitation of medical and dental expenses for income tax purposes. Finally, the delegates reaffirmed their support of the Bricker Resolution (Senate J. Res. 1), limiting treaty-making powers of the President.

Among resolutions presented for the first time at this meeting were several of general interest and importance. One of them called upon the Board of Trustees to make a detailed study of educational standards and requirements of osteopathic, chiropractic and naturopathic schools. This one was adopted and referred to the Board.

Another asked each constituent state association to create its own Committee on Rural Health. A resolution was also adopted asking that all state and territorial medical bodies get in touch with the President's Commission on Intergovernmental Relations which will soon examine the entire grants-in-aid program and offer assistance.

Three resolutions from groups of orthopedists asking that the word "Rehabilitation" be dropped from the title of the Council on Physical Medicine and Rehabilitation were referred to the Board of Trustees for study and a report in June, 1954. The contention of the orthopedists is that rehabilitation is an essential part of their professional function while the function of the Council is largely the investigation of appliances.

Three resolutions, all from Rhode Island, asking that the current "matching plan for interns" in which the Council on Medical Education and Hospitals now participates, be abandoned and that steps be taken to insure a more equitable distribution of interns to smaller hospitals, were not adopted. Extensive committee hearings did not disclose any evidence that the matching plan was depriving small hospitals of interns, and a committee has already been set up, the delegates were informed, to study the whole problem of intern procurement.

In that connection, it should be noted that a resolution asking that the current practice of listing acceptable hospitals, which subscribe to the matching plan, separately from the acceptable hospitals which do not subscribe to the plan, was not adopted either. Instead, the reference committee suggested that the Council prepare one additional list in which non-subscribers are integrated with subscribers. Still another resolution asking that teaching care in veterans' hospitals be given only to veterans with service-connected disabilities failed of adoption, as did a resolution asking that oral prescriptions be permitted for codeine preparations which have little or no habit-forming effect. The latter was refused because of the possibility that usable narcotics might be extracted from large amounts of these preparations.

Malpractice Study Requested

The Board of Trustees was instructed to make a special study of the malpractice insurance situation all over the country, looking to eventual adoption of measures to make certain that this essential type of protection will continue to be available.

Income of the American Medical Education Foundation now totals $1,000,174.00, the delegates were told. This total represents all contributions since the Foundation was started in 1950. Total contributions are now 200 per cent of the total of 1952 and contributions to it from doctors, alone, are 50 per cent greater than in 1952. In addition, the doctors gave $782,885 direct to sixty-five of the seventy-nine medical schools of the United States (the remaining fourteen failed to make a report).

This last amount was given by 22,273 individual contributors. Among other individual contributions mentioned to the delegates was a gift of $2,000 from the American College of Radiology; also the $10,000 a year contributed for the last three years by the Woman's Auxiliary to the American Medical Association. An additional $22,630 has been contributed by state auxiliaries.

Dr. Louis H. Bauer, immediate past president of the American Medical Association, has succeeded the late Dr. Elmer L. Henderson to the post of director of the Foundation. Dr. Edward L. Turner, secretary of the Council on Medical Education and Hospitals, has been designated secretary-treasurer in place of Dr. Donald G. Anderson who resigned recently to become dean of the University of Rochester School of Medivine. Dr. Anderson will continue on the board of directors.

Report from WMA

Drs. Gunnar Gundersen of Wisconsin and Edwin S. Hamilton of Illinois, both trustees of the American Medical Association, were the American delegates to the World Medical Association meeting at the Hague, September 1 to 6, 1953. They reported that an attempt on the part of various government and judicial agencies to draft an international code of medical law had met with vigorous opposition from the doctors' session. The WMA declared officially that only medical men are qualified to adopt a code for medicine; that such a code must be based upon medical ethics and that the WMA, itself, has already adopted its own international code of medical ethics which, incidentally, has been approved by the House of Delegates of the American Medical Association. A chapter on the doctor's duty to society is now being prepared for addition to that code. WMA served notice that it will never accept a code drawn up by jurists or government agencies. The American Medical Association must continue to support the world organization, the delegates were told, because it is the only international body which can speak for the practicing physicians of the world. Dr. Bauer was re-elected secretary-general of the association, with Dr. Austin Smith of Chicago taking the place of Dr. Henderson on the Council, and Dr. Gundersen replacing the late Dr. R. L. Sensenich. The editorial board of the association's *Bulletin* includes Doctors Paul Cibrie, secretary of the French Medical Association; Hugh Clegg, editor of the British Medical Journal; Lorenzo Garcia-Tarnel, secretary of the Spanish Medical Association, and Dr. Smith, who acts as executive editor of the publication. It is of interest to note that the first World Conference on Medical Education met in London in advance of this meeting. Dr. Victor Anderson, of Rochester, is vice president of this conference and attended the meeting.

The report of the general manager, Dr. George Lull, showed a membership of 117,063 in the Association, representing an increase of 7,323 over 1952, according to the reference committee on officer reports, and showing the continued confidence of the profession in the American Medical Association. All nine professional publications of the Association show an increase in circulation, including the *Journal of the American Medical Association,* which currently circulates 167,000 copies weekly. *Today's Health,* on the other hand, circulates 260,000 monthly, of which only 26,000 go to doctor-subscribers, and it shows a deficit of $150,000 for last year. There is a serious question whether publication of *Today's Health* can be continued but it was noted, also, that if doctor-subscribers could be increased to 100,000, the deficit could be erased.

The delegates heard from the new president, Dr. Edward J. McCormick, and from Mrs. Leo J. Schaefer, president of the Woman's Auxiliary to the American Medical Association. Dr. Chester S. Keefer, newly appointed secretary for health in the Department of Health, Education and Welfare, was there, also, to make his first appearance before the delegates, and Dr. Leslie M. Fitzgerald, president of the American Dental Association, was among a group of distinguished guests who brought greetings from other organizations.

The address of the president stressed some very real social and economic problems of the profession.

Public Relations Discussed

The public relations of medicine in America today are not good, Dr. McCormick said, and expensive public relations programs are not going to do much to improve them. State and county medical organizations are the ones that will have to do the work, he declared, and he urged every delegate to go home and campaign for strong grievance committees and the prompt expulsion of all who disregard the principles of medical ethics.

> "This is a grass-roots problem," Dr. McCormick said. "Good public relations cannot be bought. They must be earned by exemplary conduct and genuine service in the public interest.
>
> "We have a policing job to do. Our excellent work in all branches of medicine and the public service are easily forgotten. But not unethical practices of any kind. What we can do is to seek perfection always. In so doing we shall find good public relations. There is no other answer."

Dr. McCormick warned the delegates, also, that the greatest battle to preserve our American system of medicine is yet to come. Already this battle is shaping up in demands for medical care of veterans; in new demands for medical care plans by union labor; in the current system of drafting doctors to care for dependents of service men; in the growing demands for federal subsidies to pay for voluntary insurance. He sees it in statements of elected officials who still believe it is good politics to encourage federal handouts of all kinds and who "by inuendo and by word, magnify the medical needs of the people and pay little heed to the many more important problems affecting American men and women."

Since American medicine is actually the best in the world, Dr. McCormick suggested to popular writers and orators that they devote more of their attention to the far greater problems affecting the health and welfare of the people, such as housing, sanitation, juvenile delinquency. nutrition, un-Americanism in many institutions, dishonesty among public officials, gambling, gangsterism and other cancers on our national life "including the collectivists, socialists, communists and those who, having lost sight of God, have forgotten that we are a great nation because the Constitution was written by men of deep faith in the Supreme Being, faith in opportunity, faith in private enterprise."

Dr. Keefer declared that the major question before the profession is this:

> "How can we continue to produce and promote the finest medical care in the world today?
>
> "Our needs," he said, "are for improved medical education; for co-ordination and extension of facilities and personnel; for geographical extension of services and for bringing the cost within the reach of more people.
>
> "These needs will be met in our free society by the combined effort of the laymen, and of members of the medical and health professions working closely together with a sympathetic understanding in their local communities.
>
> "We need experimentation," Dr. Keefer declared further, "in order to find the answers to many of our problems. Experimentation should be carried out by the profession with the full co-operation of the people of the community and the maximum decision should be made at the community level."

Meetings Well Attended

The Council on Scientific Assembly reported plans well developed for the next annual session in San Francisco, June 21 to 25, 1954, and the next clinical meeting scheduled for December in Miami. It was noted that the winter meetings have been well attended, and the importance of these, as well as the annual meetings, has prompted a consolidation and reorganization of the council activities. A plan was approved for transferring the council from its status as a committee of the House of Delegates to a committee of the Board of Trustees with a permanent office at Association headquarters in Chicago. The council was enlarged and the Bureau of Exhibits was incorporated into its structure.

One of the important events of the winter meeting is the selection of the recipients for the annual General Practitioner of the Year award by the Board of Trustees. The choice this year was Dr. Joseph I. Greenwell, an eighty-year-old active practitioner from New Haven, Kentucky, who has practiced for the last fifty-three years within a 25-mile radius of New Haven. Dr. Greenwell has delivered almost 4,300 babies and, of these, 80 were born last year. He has served as a councilor from the fourth district of the Kentucky State Medical Association for thirty years and is a past president of the Nelson County Medical Society. He is also acting health officer for Nelson County and consulting physician for the Louisville and Nashville Railroad and Gethsemane Abbey. Of his twelve children, eight are living. Two were lost in World War II.

Several Minnesota physicians played an important part in the St. Louis session. Among them were Drs. William L. Benedict, of Rochester, who is also chairman of the Section on Ophthalmology, Dr. Frank J. Elias, of Duluth, and Dr. O. J. Campbell, of Minneapolis, all of whom served on reference committees.

J. A. Bargen, M.D.
O. J. Campbell, M.D.
George Earl, M.D.
F. J. Elias, M.D.
Delegates to the American Medical Association

MATERNAL MORTALITY SURVEYS

(Continued from Page 144)

sociation in co-operation with the Minnesota Department of Health. Also co-operating in the study were the Department of Obstetrics of the University of Minnesota and the Minnesota Hospital Association. The maternal mortality rate was 2.0 per 1,000 live births and the report concluded that three-fourths of the deaths were preventable. The Committee's report for 1951 shows that the rate has declined to 0.4 per 1,000 live births and that only about one-third of the deaths studied were considered preventable.

Editorial comment on Minnesota's maternal mortality surveys in the January 1953 issue of the *American Journal of Public Health* is of interest. "In only a sixth of all the states is there a concerted and continuing effort to improve maternal mortality experience through investigation, reporting, and consultation. Minnesota has succeeded in reducing maternal deaths from 2 to 0.6 per 1,000 births (1950)—in one decade—and aims to do still better. Does this story suggest anything to the other five-sixths of the states?"

A. B. Rosenfield, M.D., M.P.H.

◆ Reports and Announcements ◆

AMERICAN COLLEGE OF SURGEONS

A four-day Sectional Meeting of the American College of Surgeons featuring clinics and specialty programs will be held at the Hotel Fontenelle in Omaha, Nebraska, March 1-4, 1954. The medical faculties of Creighton University, the University of Nebraska, and seven local hospitals are co-operating in preparations for the meeting.

Eminent scientists and clinicians from many medical centers will participate in the program which will be of unusual and timely interest.

Morning clinics will be held at the teaching hospitals and at the hotel there will be special sessions in general surgery, ophthalmology, obstetrics and gynecology, otolaryngology, orthopedic surgery, pediatric surgery, thoracic surgery, and urology.

All members of the medical profession are invited to attend. Fellows of the College, members of the Junior Candidate Group, interns and residents are admitted without registration fees. Other doctors are charged a $5.00 fee, all of which goes toward paying a portion of the expenses incurred by visiting speakers.

SYMPOSIUM ON RECENT ADVANCES IN THE STUDY OF VENEREAL DISEASES

The Sixth Annual Symposium on Recent Advances in the Study of Venereal Diseases will be held in the auditorium of the Department of Health, Education, and Welfare, Washington, D. C., April 29 and 30, 1954, according to Dr. James K. Shafer, Chief of the Public Health Service's Division of Venereal Disease.

The sessions are open to all physicians and workers in allied professions who are interested in participating These symposia usually draw hundreds from all parts of the country and are the occasion for exchange of the latest available information by some of the outstanding authorities in the field of venereal disease. Topics discussed at this symposium will cover many aspects of venereal disease control including basic and clinical research, serology, epidemiology, treatment, program operation, and professional education.

AMERICAN HEARING SOCIETY'S ANNUAL COMPETITION KENFIELD MEMORIAL AWARD

Competition for the Kenfield Memorial Scholarship, awarded annually by the American Hearing Society to a prospective teacher of lip reading, will open March 1. Application blanks may be obtained by writing to the society's national headquarters, 817 14th St. N.W., Washington 5, D. C.

Deadline for returning completed applications is May 1. They are to be mailed to Mrs. Eleanor C. Ronnei, care of the New York League for the Hard of Hearing, 480 Lexington Ave., New York 17, N. Y. Mrs. Ronnei is chairman of the American Hearing Society's Teachers Committee. The winner will be announced during National Hearing Week, May 2 to 8.

Funds for the scholarship were subscribed in 1937 in memory of Miss Coralie N. Kenfield, San Francisco, California, who was nationally known for her advanced methods in teaching lip reading.

Winner of the annual award is entitled to take a teacher training course in lipreading from any school or university in the United States acceptable to the Teachers Committee. The scholarship is to be used within one year from the date of award.

A satisfactory applicant for the award must be a well-adjusted individual with a pleasing personality, legible lips, good speech and voice, and no unpleasant mannerisms. Graduation from college with a major in education, psychology and/or speech is a requirement.

Specifications for a hard of hearing contestant include thirty clock hours of private instruction in lipreading from an approved teacher or sixty hours of lipreading in public school classes under an approved teacher. Rules for competition state that an applicant shall plan to teach lipreading with or without other types of speech or hearing therapy.

THE ASSOCIATION FOR RESEARCH IN OPHTHALMOLOGY—MIDWESTERN SECTION

The annual meeting of the Midwestern Section of the Association for Research in Ophthalmology was held Sunday, February 7, 1954 at the University of Chicago Medical Schools, Chicago.

The program for the meeting included the following papers:

Further Contributions to the Enzymology of Corpus Vitreum. *Albert Zeller,* Northwestern University, Chicago.

Metabolic Studies of Lens Epithelium in Vitro. *Bernard Schwartz,* Betty Danes, and *P. J. Leinfelder.* University of Iowa, Iowa City.

The Liebmann Effect in Binocular Perception. *Maressa Hecht Orzack* and *T. F. Schlagel, Jr.,* Indiana University Medical Center, Indianapolis.

Uveitis. A Preliminary Report. *Howard D. Ostler,* University of Iowa, Iowa City.

Studies in Flicker Fusion of the Eye. *Garth J. Thomas,* University of Chicago.

Variations in Quality of Target for Flicker Fusion Fields. *Claude Trapp* and *Paul W. Miles,* Washington University, Saint Louis.

Some Effects of Yttrium 90 Upon the Posterior Ocular Segment. *Frank W. Newell, Paul V. Harper,* and *Anne M. Koistinen,* University of Chicago.

After-Image Transfer Tests in Anomalous Correspondence. *A. K. Hansen,* University of Iowa, Iowa City.

Preliminary Studies in the Aqueous Following Radiation of the Eye. *David Schoch* and *Irving Puntenney,* Northwestern University, Chicago.

Seton Procedure in Glaucoma. *Edward Pushkin, A. C. Biegel* and *Martha Folk,* University of Illinois, Chicago.

The Electroretinogram in Choroideremia. *George W. Bounds, Jr.,* and *Theodore Johnston,* University of Iowa, Iowa City.

Electroretinography in Artificial Partial Avitaminosis A. *Robert J. Davis,* University of Iowa, Iowa City.

FELLOWS ASSOCIATION OF THE MAYO FOUNDATION

Election at the annual business meeting of the Fellows Association of the Mayo Clinic, held at Plummer Hall, Rochester, December 9, resulted as follows: Dr. H. C. Habein, Jr., president; Dr. John O. Godden, vice president, formerly secretary; Dr. S. E. Sivertson, secretary; Dr. Edward B. Waldman, treasurer.

GP ACADEMY ANNOUNCES OUTSTANDING PROGRAM

Attention of general practitioners is called to the Sixth Annual Scientific Assembly of the American Academy of General Practice to be held in Cleveland, Ohio, March 22 to 25, 1954.

A lectureship and exhibit program designed especially to give the general practitioner diagnostic techniques and procedures which apply to everyday practice has been arranged for this unique three-day session.

Speakers, who are top authorities in their fields, include among others, Sir Alexander Fleming of London, discoverer of penicillin; Dr. E. J. McCormick, president of the American Medical Association; Dr. Howard Rusk, outstanding authority on rehabilitation procedures; Doctors George Crile, Jr., and George H. Curtis of the Cleveland Clinic; Dr. Clarence S. Livingood of the Henry Ford Hospital in Detroit; Dr. Sol Katz, associate editor of GP, official publication of the Academy; and Dr. John C. Krantz, Jr., of the University of Baltimore. More than forty-five scientific exhibits will supplement the scientific program which is under the general supervision of Dr. John F. Mosher of Coeymans, New York.

Social events will include a president's reception, music and dancing and a fashion show, coffee hour and book review for visiting women.

BLUE EARTH COUNTY MEDICAL SOCIETY

Election of officers of the Blue Earth County Medical Society was held December 14 at Mankato with the following elected: Dr. R. Wynn Kearney, president; Dr. J. J. Eustermann, vice president; Dr. William S. Chalgren, secretary-treasurer; Dr. L. M. Hammar, delegate to the Minnesota State Medical Association.

FOUR-COUNTY MEDICAL SOCIETY

The Four-County Medical Society and Auxiliary met at the Mayo Foundation House for afternoon and evening sessions, January 6. Talks given in the afternoon were on "Treatment of Hypertension," "Current Use of Antibiotic Drugs," "The Orthopedic Examination of the Back and Hip," with a demonstration, and "The Management of Frequently Encountered Skin Disorders."

After dinner the women toured the new Mayo Clinic building, while the doctors attended a meeting held in conjunction with the regular staff meeting of the Mayo Clinic.

GOODHUE COUNTY MEDICAL SOCIETY

At a dinner meeting held at Red Wing, December 3, the following officers were elected to head the Goodhue County Medical Society: Dr. Ezra Bridge, Mineral Springs Sanatorium, president; Dr. R. V. Sherman, vice president; Dr. George M. B. Hawley, secretary-treasurer.

ST. LOUIS COUNTY MEDICAL SOCIETY

Election of officers of the St. Louis County Medical Society took place at the Kitchi Gammi Club, Duluth, December 11. The following officers were elected: Dr. S. H. Boyer, Jr., installed as 1954 president; Dr. K. R. Fawcett, Duluth, president-elect for 1955; Dr. Walter S. Neff, Virginia, vice president, Virginia; Dr. F. R. Kotchevar, Eveleth, member of the judiciary committee; Dr. Paul Reed, Virginia, member of the economics committee, and Dr. Clarence N. Jacobson, Chisholm, and Dr. Neff, delegates.

STEARNS-BENTON COUNTY MEDICAL SOCIETY

Stearns-Benton Medical Society met at the Granite Bowl, St. Cloud, December 17, electing the following officers: Dr. Henry Reif, St. Cloud, president-elect for 1955; Dr. John Kelly, Cold Spring, president—elected last year; Dr. William Autry, vice president; Dr. E. W. Anderson, secretary.

TRI COUNTY MEDICAL SOCIETY

Doctors and their wives from Kandiyohi, Meeker and Swift Counties met at the annual dinner of the Tri County Medical Society, held in Willmar, December 17. Lakeland Medical Center and Willmar Clinic staffs acted as hosts, with Dr. Roger P. Michels, Willmar, presiding. Musical numbers included one on the violin by Dr. Robert Peterson with Dr. L. J. Opsahl at the piano. Dr. Peterson and Dr. Opshal with Dr. A. M. McCarthy gave a humorous specialty number.

Dr. Paul Giddens, president of Hamline University, spoke on the danger that confronts academic freedoms today, and also pointed out the need for greatly increased facilities in teaching and school rooms for the oncoming tremendous increase in school population.

MINNESOTA STATE BOARD OF HEALTH

Dr. F. W. Behmler, Morris, was re-elected president of the Minnesota State Board of Health at the annual meeting of the board held in Minneapolis, January 12. Prof. Herbert Bosch, University of Minnesota, was named vice president, and Dr. A. A. Chesley was reelected secretary and executive officer. Dr. Behmler has been a member of the State Board of Health since 1940.

Of General Interest

Dr. John W. Ferree, director of community service and education, New York City, and Rome Betts, executive director of the American Heart Association also of New York, were in Minneapolis December 16 to discuss the 1954 Minnesota Heart Fund program set for February. They conferred with **Dr. W. S. Neff,** Virginia, on community service; with **Dr. Karl Anderson,** Minneapolis, on cardiac problems in industry, and **Dr. John Briggs,** Saint Paul, on professional education. They also met with the State Medical Association's executive committee during the day, and in the evening with the general membership at a meeting at the University of Minnesota.

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Yellow Medicine and Renville Counties qualified for tuberculosis accreditation recently. Attended by approximately ninety persons from the two counties, a banquet was given for the occasion December 7, at the Romberg Hotel, Sacred Heart. To qualify for accreditation a county must have a tuberculosis death rate averaging ten or less per 100,000 population for a five-year period. Yellow Medicine County had a 3.7 and Renville a 5.8 rate for the years 1948 to 1952. The county must also have a low infection rate which is established by sampling high school seniors through a tuberculin testing program.

Dr. M. S. Nelson, serving for many years as president of the Yellow Medicine County Tuberculosis Association; **Dr. Lewis S. Jordan** and **Dr. Kathleen Jordan** of the Riverside Sanatorium, Granite Falls, and Clara Thorpe, the organization's secretary for many years, were presented with framed certificates in recognition for the work they have done in combatting tuberculosis. The awards were made by Marguerite Breen, public relations officer for the Minnesota Tuberculosis and Health Association.

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Dr. Pao-shu Chen, Formosa, and Dr. Andreas Angara, the Philippines, visited the Hennepin County League for Planned Parenthood recently for information they might use to help their countrymen. **Dr. Marian Grimes,** planned parenthood clinician, explained the operations of the clinic, and gave the two doctors data on birth control. Dr. Chen, public health student at the University of Minnesota on a World Health Organization scholarship, said there was a great need of birth control in Formosa, where, though the area is the same size as Maryland and Delaware combined, has a population four times that of the two states. Dr. Angara, studying public health at the University of Minnesota on a year's scholarship from the Foreign Operations Administration, was primarily interested in the "rhythm" method of planned parenthood for the underpopulated Philippines. Both doctors said the greatest problems in health, in their respective countries, were sanitation—water supply and waste disposal.

Dr. John Grant, Sauk Centre surgeon, specializes in "organs" in his spare time. He builds pipe organs, which he says he learned how to build when he read a few books and talked to some "guys in the business" during his medical school days. After the reading and advice from "the guys in the business," he began buying parts and assembling the pipe organs. His last venture is a 1,300-pipe organ at the Church of the Good Samaritan at Sauk Centre which he built with the help of the pastor, Rev. Nor Schoenheider. The organ, built from parts secured from churches in Minneapolis, St. Paul, Excelsior and Willmar, was dedicated December 20.

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Among Fellows of the Mayo Foundation receiving degrees at the Commencement exercises at the University of Minnesota December 10 were the following: **Dr. Robert C. Bahn,** Ph.D. in pathology; **Dr. Homer R. Warner,** Ph.D. in physiology; **Dr. Dean P. Epperson,** Ph.D. in surgery; **Drs. Earl F. Beard, Fred Z. Havens, Jr., Alexander M. Minno, Waldemar N. Person, John D. Ruff** and **Robert G. Tompkins,** M.S. in medicine; **Dr. Gilbert A. Reese,** M.S. in ophthalmology; **Dr. Eran O. Burgert, Jr.,** M.S. in pediatrics; **Dr. Milton E. Kurth,** M.S. in plastic surgery; **Dr. Charles F. Smith,** M.S. in radiology; and **Drs. Mitchell S. Madison, J. D. Mortenson, Harold W. Neuman** and **Donald N. Vivian,** M.S. in surgery.

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Dr. Gordon Uhley, Crookston, is taking a year's leave of absence from the Northwestern Clinic, Crookston, in order to do graduate work in urology with Dr. S. D. Creevy at the University of Minnesota, Dr. Uhley's alma mater.

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Dr. Marie Bepko, Cloquet, has apparently mastered the art of budgeting time. She is physician, partner with her husband, **Dr. R. H. Puumala,** homemaker, and active in several professional and civic organizations.

Born in Chicago of Czechoslovakian parents, Dr. Bepko graduated from the University of Illinois, College of Medicine, and interned at the American Hospital during which time she married Dr. Puumala, who was at this time resident at University of Illinois Research Hospital. After their first child was born Dr. Bepko continued her preparation by taking a six months' residence in psychiatry at the state hospital in Elgin, Ill. The family moved to Cloquet in 1935 where the two doctors have since practiced.

Though both doctors have travelled extensively, they have not taken their vacations together until last summer, when with their two children, they motored to Anchorage, Alaska, via the Alcan highway. Besides her duties to her family, Dr. Bepko is vice president of the Pan American Women's Alliance; life member of the American Medical Women's Association; president of the Minnesota State Medical Women's Association; active in community activities which include Parent-Teacher work, projects of the Business and Professional Women's Club, and other organizations.

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Dr. J. W. Bratholdt and **Dr. L. J. Larson,** Watertown, moved their offices into the newly constructed Bratholdt Clinic December 12. The clinic is located across the street from the Bratholdt Hospital.

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Dr. Harold M. Solvason, formerly located at Hennepin and Franklin Avenues, Minneapolis, has opened a new office at Fifty-fourth and Nicollet, Minneapolis. Dr. Solvason is a graduate of the University of Minnesota School of Medicine.

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Dr. Francis J. Braceland, Hartford, Connecticut, formerly of the Mayo Clinic, is a member of one of the divisions of the task force on Federal Medical Services of the Hoover Commission. The activities of the division is to gather information from government agencies and other sources before any conclusions are reached or any recommendations given to the Commission.

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Dr. Edward Zupanc, formerly of Granville, Wisconsin, who has served with the Armed Forces for two years in Japan, will open an office in Duluth, specializing in pediatrics.

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Dr. Swan Ericson, Le Sueur, has retired after practicing medicine in the community for the past thirty-one years. Dr. Donald A. Limbeck. graduate of the University of Nebraska, Medical School, is taking over Dr. Swanson's practice.

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Dr. Edward Q. Ertel, Ellendale, recently contributed a microscope to the Ellendale High School. The gift included a wood carrying case, glass bell protective cover, and immersion lens.

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Awarded for his work in establishing radiological teaching programs in various medical schools in India, **Dr. Leo G. Rigler,** Minneapolis, received the gold medal of the Radiology Society of North America December 23. Dr. Rigler, head of the University of Minnesota's Radiology Department visited India early last year as a member of the World Health Organization specialists' team.

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Dr. William Feigal, Falls Clinic, Thief River Falls, addressed the monthly meeting of the Roseau County Medical Society, December 11. Together with his subject, "Urology in Northern Minnesota," he also reviewed x-rays, showing pathological findings in this area with their treatment and clinical course.

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Dr. David R. Johnson and **Dr. Lyle A. French,** Minneapolis, spoke on the topic, "Cerebral Hemispherectomy for Intractable Seizures," at the regular meeting of the Minnesota Society of Neurology and Psychiatry, at the Town and Country Club, St. Paul, January 12. **Dr. Frank Kiesler,** also Minneapolis, spoke on "The Patient who Can't be Cured."

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Dr. D. H. Garlock, Bemidji, has recently been certified as a Tree Farmer in the state and national tree farm program. His 155-acre farm east of Lake Bemidji was approved by the tree farm committee of Keep Minnesota Green.

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Dr. F. H. Magney, Duluth, was given the Silver Beaver Award. high scouting honor, at the annual business meeting of the North Star Council, Boy Scouts of America, at Duluth Chamber of Commerce quarters December 7. Dr. Magey was cited for long service (since 1935) in keeping the boys fit "in matters pertaining to health."

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Dr. Donald H. Honath, Detroit, Michigan, has joined **Dr. A. J. Olson** and **Dr. F. C. Anderson,** at their clinic in Owatonna. Dr. Honath is a graduate of Wayne University College of Medicine; interned at Evangelical Hospital, Chicago, and finished a four and a half-year residency in general surgery at Mt. Carmel Mercy Hospital, Detroit.

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A new series of industrial safety schools sponsored by the Duluth Chamber of Commerce was held in Duluth, December 9. **Dr. John F. Briggs,** Saint Paul addressed the general session on "Optimism in Heart Disease." **Dr. James Tetlie,** Duluth, spoke on eye injuries; **Dr. Kenneth A. Storsteen,** Duluth, on gastro-intestinal disease; **Dr. Robert J. Goldish,** Duluth, on diabetes; **Dr. John Schmid,** Duluth, on industrial dermatitis; **Dr. Walter Kelley,** on burns; **Dr. William Grohs,** hernias; **Dr. Elizabeth Bagley,** "the home, an important industry," at the women's section.

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Dr. R. J. Ripple, Saint Paul, spoke to members of the Washington County Medical Society at the meeting, December 8, at Stillwater, on the problems of resuscitation and anesthesia. Doctors from Forest Lake and Hastings attended the meeting which was followed by dinner.

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Dr. G. M. Hawley spoke on "Ministering to Hospital Patients" at a meeting of the Red Wing Ministerial Association held December 7, at the Baptist parsonage, Red Wing.

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The new Veterans Research Hospital, Chicago—key hospital in the veterans hospital system—has three Minnesota doctors on its staff. **Dr. Richard V. Ebert,** chief of medicine; **Dr. Ben I. Heller,** chief of the metabolic section; **Dr. Craig Borden,** chief of the cardio-vascular section. All three men have faculty appointments at Northwestern University.

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Dr. F. H. Baumgartner, Albany, has resigned from his office as mayor of Albany. For ten years he has spearheaded drives that have brought about improvements in the village, but his responsibilities as physician has increased to such an extent, he was unable to carry on both tasks.

On January 2, **Dr. K. A. March,** Cambridge, was joined in his practice by Dr. Michael Danyluk, originally from Germany. Dr. Danyluk has been on the staff at the Swedish Hospital, Minneapolis, since coming to Minneapolis in 1950. He studied medicine at the University of Munich, Germany.

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Dr. Albert V. Stoesser, Professor of Pediatrics and Allergy at the University of Minnesota, spoke on "Allergy and Your Child's Behavior" at a meeting of the Child Psychology Study Circle, January 4, at the Groveland Park School Auditorium, St. Paul.

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The complete list of new members from Minnesota accepted by the American College of Surgeons for 1953 is as follows:

Grant L. Griebie, Brownton, surgery; Hubert D. Clapp, Crookston, obstetrics-gynecology; Philip F. Eckman, Duluth, surgery; Francis L. Stutzman, Montevideo. surgery; Clarence W. Wasmund, Red Wing, ophthalmology; John W. Kirklin, surgery, Karl A. Lofgren, surgery, and Frank Charles Mann, pathology, all of Rochester; Hanns C. Schwyzer, Saint Paul, surgery, and Jack R. Pierce, Virginia, obstetrics-gynecology; Ivan D. Baronofsky, surgery; Walter P. Eder, surgery; Milton Feinberg, surgery; Davitt A. Felder, surgery; Geddes B. Flagg, ophthalmology; William Harold Ford, surgery; George L. Garske, urology; John K. Grotting, plastic surgery; Carter W. Howell. surgery; Arthur W. Ide, Jr., surgery; C. Walton Lillehei, surgery; and Henry A. Norum, surgery, all of Minneapolis.

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Dr. Ernest O. Hanson, who has been practicing in Carlton, has opened his office in Cloquet recently.

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The Hayfield Medical Center, a $10,000 community project, was ready for occupancy January 15, by **Dr. Harold J. Elliot,** former missionary doctor in Africa, who came to Hayfield last fall.

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Dr. J. E. Ponterio, Shakopee, recently had $140 stolen from his cash box which was kept in his office desk. Filing cabinets and desk drawers had been rifled.

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Dr. Henry G. Moehring, radiologist of the Duluth Clinic, Duluth, is one of the co-authors of a three-volume book entitled "Clinical Roentgenology," published by Charles C Thomas, Springfield, Illinois. The first volume is now being offered for sale.

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Progress is being made on the new Adams Clinic, Chisholm, which will be ready for use sometime in February. **Dr. Clarence Jacobson, Dr. Bayerd French,** and **Dr. George Erickson** will have their offices in the building plus rooms and facilities for x-ray and laboratory work.

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More than 1,050 doctors attended the twelfth annual meeting of the American Academy of Dermatology and Syphilology at the Palmer House, Chicago, recently. Among officers elected was **Dr. John F. Madden,** Saint Paul, elected vice president.

Dr. Solveig Gislason, New Ulm, recently accepted a position on the medical staff at the State Hospital, St. Peter. Dr. Gislason, born in a log cabin near Gardae, North Dakota, attended North Dakota schools—Normal School, Valley City, and University of North Dakota, Grand Forks, and then took a claim in Montana. She lived alone on the ranch riding horseback to the school where she was teaching. Interest in further education sent her to University of Minnesota where she graduated from the Medical School in 1921.

Dr. Gislason has written children's books that have been recognized and displayed at fairs and seminars by educational authorities in foreign countries such as Belgium, India, et cetera. She has operated an antique shop in New Ulm, and has also been active in civic and social affairs, especially as president of the New Uum Recreation Commission.

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Appreciated by the communities he served so long, **Dr. Mose Lane Strathern,** Gilbert, still makes his rounds though he began his practice on the range in 1911. He moved to Coleraine with his bride that year to take up practice at $100 a month; later he moved to Gilbert, which was then one of the boomingest towns on the Range. During the horse and buggy days, the fee for delivering babies was $5, when the fee went up to $15, Dr. Strathern bought an automobile. Last October 22, he with nine other members of the team that played Michigan a six to six game in 1904, had a reunion in Minneapolis. Dr. Strathern was captain of the Minnesota team that year.

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Dr. Alan Challman, of Minneapolis, attended the annual meeting of the Advisory Committee to the Psychiatry and Neurology Division of the United States Veterans Administration held in Washington, D. C., January 18 and 19, 1954.

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From a reader: In a recent issue (November) of your excellent magazine, you announced in your news column, the *three* young physicians named by *Time* magazine as "Newsmakers of Tomorrow!"

You neglected to name **Edmund B. Flink, M.D.,** director of Internal Medicine at Veterans Hospital, and member of the University of Minnesota Medical staff. His recognition was as deserving as the other gentlemen. (*We regret the omission.*—The Editors.)

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Dr. George D. Haggard, Minneapolis, observed his ninety-seventh birthday January 18. At this time he also received a Life Membership Certificate from the Minnesota Medical Association of which he is justly proud. He has been interested in the Hennepin County Medical Society over the years, and to use his own words, he said, "Why I can recall years ago when I was secretary of the county society, we were gratified that we could boast of eighty members—and now the county society has well over 1,000 members."

Dr. Haggard retired completely from his practice only two or three years ago. He is making his home now with his daughter.

HOSPITAL NEWS

The St. Cloud Hospital has named the following medical staff for the coming year: **Dr. Otto Phares**, president; **Dr. E. M. Anderson**, president-elect; **Dr. J. B. Gaida**, vice president; **Dr. Vernon Neils**, secretary. Serving on the executive committee with the new officers are: **Dr. L. M. Evans, Dr. L. A. Veranth** and **Dr. Karl Walfred**, past president. Chiefs of services include: **Dr. H. E. Sisk**, medicine; **Dr. C. F. Brigham, Jr.**, surgery; **Dr. R. N. Jones**, anesthesia; **Dr. F. J. Schatz**, obstetrics; **Dr. C. S. Donaldson**, orthopedics; **Dr. W. T. Wenner**, EENT; **Dr. H. J. Reif**, pediatrics; **Dr. P. E. Stangl**, clinical and pathological laboratories; and **Dr. C. B. Nessa**, chief of radiology.

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The Duluth, Missaba and Iron Range Railway made a contribution of $300,000 toward construction of a new hospital at Two Harbors. The money is to be used by the North Shore Memorial Hospital, Incorporated, which is a non-profit group, organized under Minnesota laws, and backed by the Two Harbors Independent Citizens Committee. **Dr. Allen Gunn-Smith**, spokesman for the hospital indicated the group planned the construction of a fifty-bed hospital that would serve the entire North Shore area.

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Fairmont Community Hospital has the following new officers on the medical staff: **Dr. E. E. Zemke**, president; **Dr. O. E. Wandke**, vice president, and **Dr. Neil Wickerson**, secretary-treasurer.

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The annual meeting of the medical staff of St. John's Hospital, Saint Paul, named **Dr. T. S. McClanahan**, chief of staff, December 15; **Dr. Robert W. Emmons** was named first vice chief; **Dr. P. G. Polski**, second vice chief; **Dr. Jack W. Strand**, secretary-treasurer; **Dr. C. Kenneth Cook**, retiring chief of staff, was named to the executive committee for a three-year term along with **Dr. H. L. Stolpestad.**

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The need for a community hospital in Forest Lake is great, according to a recent editorial in *Forest Lake Times.* The need is also great for public spirited people living in the community to attempt the project, while the need for federal aid is also great. The area designated four years ago as one of the high-priority areas for a thirty-five-bed general hospital. **Dr. Helen L. Knudson**, State Department of Health, indicates that she has been unsuccessful finding people interested in the hospital program.

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Tentative plans for the Douglas County Hospital, Alexandria, were shown by Architect Frank Jackson at a meeting of the Advisory Committee recently. Plans, that call for a new hospital architecture, consists of diagonal wings extending Y-like arms from each corner of the one-story building. The hospital, capacity forty-eight beds, will be located on a 17-acre plot of ground in the southeast section of the city. **Dr. L. F. Wasson**, Alexandria, and **Dr. E. E. Emerson**, Osakis, are among the committee members.

Plans for a fund-raising project to finance building a community hospital at Cloquet was approved at a joint meeting of the Cloquet Civic Affairs Council and the rural-urban committee of the Cloquet Chamber of Commerce recently. A poll was taken by postcard which indicated that 97.3 per cent of the people in the community were in favor of the project.

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New officers of the medical staff of Glenwood Hills Hospital were elected at the annual meeting held December 16, 1953, as follows: **Dr. J. A. Resch**, president; **Dr. John Schumacher**, vice president, and **Dr. Irving Bernstein**, secretary.

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MINNESOTA BLUE CROSS-BLUE SHIELD

At its meeting on December 30, 1953, the Board of Directors of Blue Shield accepted the resignation of Arthur M. Calvin as Executive Director effective as of January 1, 1954. In view of the fact that the increased duties and responsibilities which Mr. Calvin's recent election to the office of President of Blue Cross have imposed upon him may prove too great for his continued active leadership in the executive management of Blue Shield, the Board of Directors after considerable deliberation accepted Mr. Calvin's resignation. In so doing, the members of the Board highly commended Mr. Calvin for his distinctive contribution to the successful organization and growth of Blue Shield. As Executive Director from the inception of Blue Shield six years ago, he has seen it grow to cover 600,000 Minnesotans, and its payments to doctors for services rendered subscribers now exceed $4,000,000 annually.

The Board of Directors, having accepted Mr. Calvin's resignation, appointed Dr. Edwin J. Simons as Executive Director. Dr. Simons will continue the executive management of Minnesota Blue Shield, which is now one of the twelve largest Blue Shield Plans in the nation. Dr. Simons was formerly engaged in the practice of medicine at Swanville, Minnesota, for a period of twenty-five years. For over three years, he has served as Medical Director of Blue Shield, and in the past year, he has also served as its Associate Executive Director.

Blue Shield and Blue Cross will continue to co-ordinate their activities as they have in the past. Blue Cross will continue to provide Blue Shield with such services as enrollment, billing, accounting and statistical services. The two organizations will remain in close co-operation to promote the development of both plans in order that the people of Minnesota may be provided with the best in medical-hospital service and coverage.

One person out of every six covered by Minnesota Blue Cross used hospital care during 1953, an increase over the care used in 1952.

Approximately 942,000 days of hospital care were provided by Blue Cross, an increase of about 40,000 days over 1952. This increase in hospital utilization also necessitated an increase in Blue Cross funds paid out. Almost fifteen million dollars was paid out for Blue Cross subscribers and their families in 1953 compared with payments of $12,500,000 in 1952.

Minnesota Medicine

Journal of the Minnesota State Medical Association, Southern Minnesota Medical Association, Northern Minnesota Medical Association, Minnesota Academy of Medicine and Minneapolis Surgical Society

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THE PRESENT STATUS OF SURGERY FOR AORTIC STENOSIS

ROBERT P. GLOVER, M.D.
Philadelphia, Pennsylvania

THE PROBLEMS associated with the surgical alleviation of aortic stenosis have to date presented far greater difficulties than any of the other stenotic valvular lesions. These problems have been recognized for many years since the first surgical attempt to relieve an obstructed aortic valve was made by Tuffier in 1913.[5] Their exact nature, however, has only been fully appreciated within the past half decade, during the recent renaissance of intracardiac valvular surgery. Although as much if not more time and valiant effort, both experimental and clinical,[2,3,4] has been directed toward this most intractable of valvular lesions as has been devoted to tricuspid, pulmonary and mitral stenosis, equal success has not as yet been forthcoming. There are a number of reasons for this state of affairs.

Anatomically, the valve is the least accessible to exploration, for it lies centrally placed within the confines of the heart. In aortic stenosis, the left ventricular myocardium is greatly hypertrophied, which, together with the tremendous vascular pressure generated within its outflow tract, prevent the use of a subaortic transmyocardial incision for the insertion of finger or instrument similar to that used in pulmonary valvulotomy, because of the ensuing uncontrollable hemorrhage. The valve cannot be effectively reached through an auricular appendage. The position of the ostia of the coronary vessels lying directly behind the valve leaflets makes direct reconstructive surgery hazardous (Fig. 1).

Pathologically, there is an early fusion of the three valve cusps, the margins of which become thickened, rolled, eburnated and infiltrated with calcium very early in the course of the disease. This is due, in part, to the disease itself but more particularly to the location of this valve in the course of the blood stream. The valve lies in a confined space of small diameter and is the recipient of the full head of vascular pressure from the strongest of contracting cardiac chambers—the left ventricle. The factor of vascular *trauma* is, therefore, of great importance for, following initial fibrotic agglutination of the cusp margins, there is a constant and steady deposition of traumatic elements such as fibrin and calcium from which there can be no relief. Thus, calcification develops early and as the leaflets are relatively small in surface area, almost complete immobility and fixation may be rapidly evident. Some flexibility of the cusps at their attachment to the aortic ring may remain for a time but this is less prominent than that seen in the stenotic mitral valve. This factor of *trauma* no doubt partially explains the fact that the anteromedial leaflet of the mitral valve (also called the aortic or septal leaflet) is the seat of the greatest calcification in mitral stenosis, for it lies nearest the aortic ring in the high flow pressure left ventricular outflow tract and also receives the brunt of the vascular ejection at the time of left ventricular contraction (Fig. 2). Therefore, from a pathologic standpoint, surgical intervention in aortic stenosis, to effectively re-

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store a measure of normal valve leaflet motion, must be carried out before extremes of the above described process are reached. To wait longer merely compounds the likelihood of surgical failure, for in consequence of the valvular change, the left ventricular myocardium hypertrophies and dilates, in due time to wear out and become incapable of effective action. As the left ventricle is the cornerstone of the heart and circulation its failure predicates irremediable disaster. This is in contrast to the findings in mitral stenosis, where, despite profound pathological and clinical changes even to the point of prolonged congestive failure and invalidism, the left ventricle retains its indispensable function—actual, compensatory and recuperative—almost to the end.

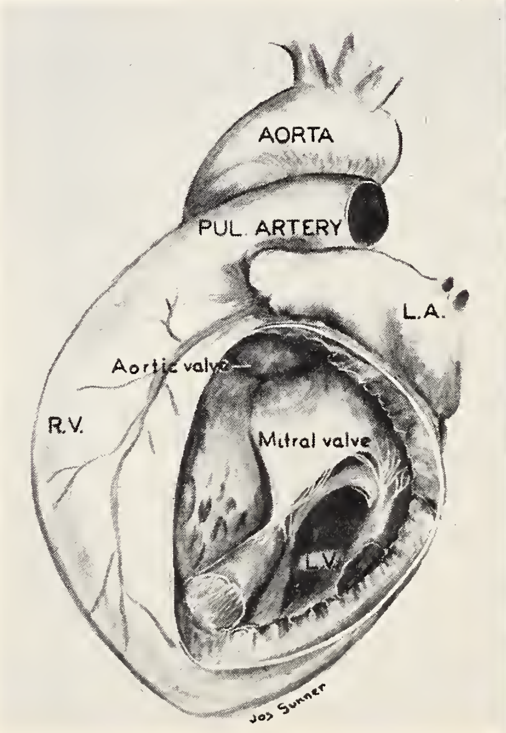

Fig. 1. Note the aortic valve lying beneath the pulmonary artery almost centrally placed within the cardiac mass. Safe access to the valve can be gained through the left ventricular myocardium near its apex.

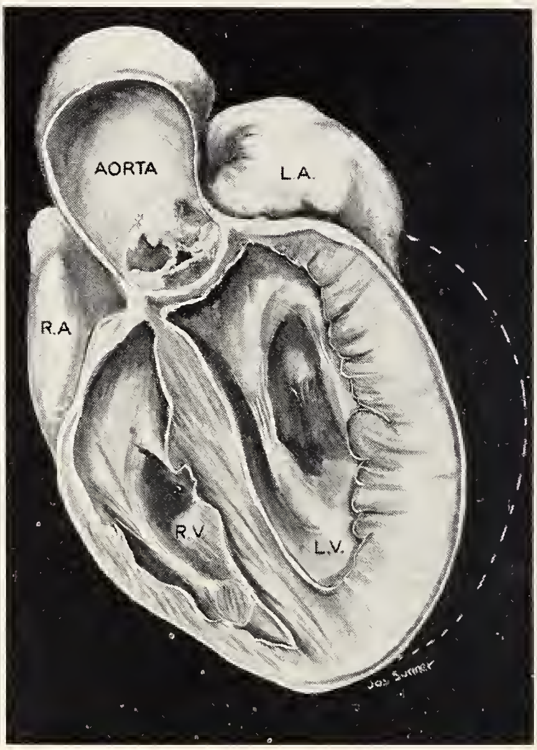

Fig. 2. Early calcification of the aortic valve with progressive concentric hypertrophy of the left ventricular myocardium. Observe the post stenotic dilatation of the ascending aorta.

Physiologically, because of the jet-like ejection of relatively small amounts of blood with each left ventricular contraction, the systolic blood pressure may not reach high levels although it is perhaps sustained and maintained longer than usual. Diastolic pressures, however, are high so that the pulse pressure is small and coronary artery filling, now under greater stress because of the demands of an increased myocardial mass, may be inadequate. As such, these patients are often precarious anesthetic risks, to say nothing of the superimposed danger of surgery.

Surgical Objective

Despite these apparent roadblocks, considerable progress has been made. The experimental work which has been undertaken in an effort to find a satisfactory surgical solution to this problem has been previously reported.[1] Briefly, the present operative approach calls for a left lateral thoracotomy, the introduction of a dilating instrument through a relatively avascular area of the left ventricle near its apex and lateral to the left descending coronary artery. The dilator is directed through the valve by passing it over a previously placed wire guide, the olivary tip of which can be felt in the aorta prior to the insertion of the larger instrument through the myocardium. The dilating head opens in triangular fashion causing the fused commissures of the stenotic valve to separate, partially relieving the stenotic obstruction and restoring a measure of valve action (Fig. 3).

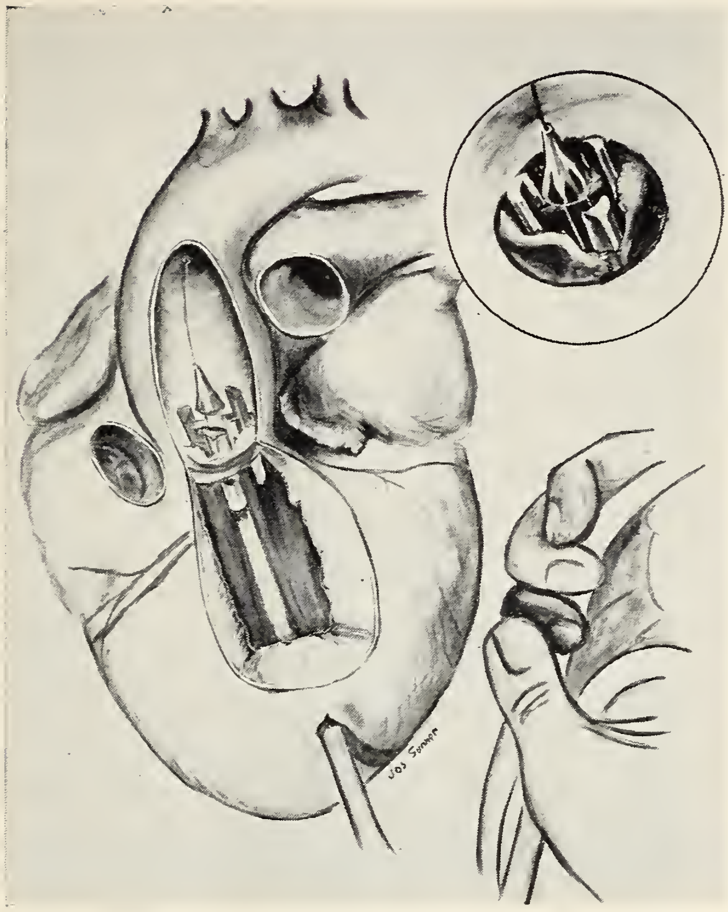

Fig. 3. The aortic dilator inserted through the myocardium of the left ventricle near its apex. This instrument is guided through the aortic valve by threading it over a fine olive-tipped wire previously placed through the valve. The insert depicts the three flanges of the dilating head separating the commissures. The self-rotating head settles automatically into the V-shaped commissures of the fused aortic cusps.

TABLE I.
CLINICAL CLASSIFICATION OF AORTIC STENOSIS

Stage	Symptoms
1	Asymptomatic—murmur only
2	Fatigue, heart consciousness, palpitations, visible pulsations
3	Syncope, Angina—reduced cardiac output
4	Episodic pulmonary congestion
5	Congestive failure

TABLE II. AORTIC STENOSIS

Stage	Results				
	No.	Excellent	Improved	Unimproved	Deaths
1. Asymptomatic—murmur only	0	—	—	—	—
2. Fatigue, heart consciousness, palpitations	2	2	—	—	—
3. Syncope, angina	10	2	6(3*)	1	1
4. Episodic pulmonary congestion	5	—	3	1	1(1*)
5. Congestive failure	5	—	—	—	5(2*)
Totals	22	4	9	2	7

*Combined Mitral and Aortic Commissurotomies.

Clinical Classification (Table I)

The necessity for translating the present rationale of surgery for aortic stenosis into reasonable, practical language has called for the development of a classification for the recognition of clinical states either applicable or unsuitable for surgery at the present time.

The recognizable clinical stages through which the average patient with aortic stenosis will pass are well known, although perhaps not readily defined in the mind of the physician in the light of surgical advances. *Stage 1* is the period during which the typical systolic murmur develops before symptoms result. *Stage 2* marks the onset of an ill-defined period where the patient becomes subjectively aware of his forceful heart action, palpitations develop, easy fatigue becomes noticeable and his attention may be called to the unusual, visible pulsations in his supraclavicular and suprasternal notches. Admittedly, this stage is not specifically characteristic of aortic stenosis *per se,* but when seen with an aortic systolic murmur the combination takes on added significance. *Stage 3* ushers in the obvious period of progressive disability due to a diminishingly effective cardiac output as evidenced cerebrally by spells of dizziness and actual syncope. Myocardially, substernal discomfort, tightness, effort and angina with its characteristic distribution bespeak inadequate coronary flow. With beginning left ventricular failure, *Stage 4* is characterized by episodes of pulmonary congestion and edema at first rather easily controlled by medical measures but shortly to become refractory to even the most vigorous management. *Stage 5* connotes impending disaster and an early demise, for in addition to left ventricular failure, superimposed right heart failure with hepatomegaly, ascites and peripheral edema now labels the heart picture as one of irretrievable damage not to be reversed by any regime—medical or surgical.

Results of Surgery (Table II)

Twenty-two cases have undergone aortic commissurotomy and six of these have been combined with a mitral commissurotomy because of concurrent bivalvular stenosis. One patient (Stage 2) done over three years ago remained completely asymptomatic for two years. During the past twelve months, although working steadily and living a normal life, he has noticed some minor precordial tightness with exercise after meals which had not been present during his first two postoperative years. This patient's aortic commissurotomy was performed with our original dilator which did not have the strength or wide diameter opening of the present more refined instrument which may account for his present status. Clinically, there has been little change in his cardiac findings.

The remaining twenty-one cases have been operated upon in the past eighteen months. The pertinent data can be seen at a glance in Table II. The over-all mortality has been high which may have been influenced somewhat by the inclusion of cases with bivalvular surgery. The observation of paramount importance, however, is that cases done in the early symptomatic phase (twelve cases—Stage 2 and 3) run a risk at present of only 8.3 per cent and even including those in Stage 4, when left ventricular failure with pulmonary edema has entered the picture the mortality rate is a reasonable 11.7 per cent. One has but to consider the seriousness of the disease and the utter hopelessness of its immediate prognosis to appreciate that such results are highly encouraging at this stage of our surgical progress. When true congestive failure has developed (Stage 5) it is doubtful whether much, if any, salvage can be either expected or obtained.

Conclusions

The natural course and progression of rheumatic stenotic valvular disease has dictated the necessity for the mechanical relief of its acquired stricture. Mitral and aortic commissurotomy have become major adjuncts in the over-all and continuing care of victims so afflicted. Provided the patient is referred for surgical intervention early in the course of his symptomatic progression, such a patient can be restored to a high level of efficiency and enjoy a more normal life as a useful and productive citizen. The burden of selecting patients at *an early stage* rests squarely on the shoulders of the practicing physician and this responsibility can no longer be avoided or postponed.

References

1. Bailey, Charles P.; Glover, Robert P.; O'Neill, T. J. E., and Redondo-Ramirez, H. P.: Experiences with experimental surgical relief of aortic stenosis; preliminary report. J. Thoracic Surg., 20:516-541 (Oct.) 1950.
2. Glover, Robert P.: Stenotic valvular disease of the heart: Surgical treatment. Texas State J. Med., 48: 674-685 (Oct.) 1952.
3. Glover, Robert P.: The indications for and the results of surgery for mitral and aortic stenosis. Maryland State J., 2:547-554 (Oct.) 1953.
4. Glover, Robert P.; Bailey, Charles P., and O'Neill, T. J. E.: Surgery of stenotic valvular disease of the heart. J.A.M.A., 144:13, 1049-1057 (Nov. 25) 1950.
5. Tuffier, T.: Etat actuel de la chirurgie intrathoracique. Tr. Internat. Cong. Med., 1913, Londone, 1914, Sect. 7, Surgery, pt. 2, p. 249; La chirurgie du coeur. Cinquienne Congress de la Societe Internationale de Chirurgie, Paris, July 19-23, 1920, Rapports Proces—Verbauz et Discussions Publies par le Docteur, Brussels, Hayez, 1921, L. Mayer, pp. 5-75.

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SEPTAL DEFECTS

A ventricular septal defect probably is the commonest congenital cardiac anomaly. *Although a small ventricular septal defect (maladie de Roger) makes the most noise, it is of the least physiological importance.* The murmur is systolic in timing, may be short or long, is loud, harsh and usually maximal along the lower left sternal border; the heart will be normal in size. When the defect is large or high in the septum, the murmur may be loudest over the pulmonic area and the heart may show some enlargement. This latter type is often misdiagnosed as a patent ductus because of the location of the murmur. Surgical repair of ventricular defects is still in the experimental stage.

In an auricular septal defect the systolic murmur is usually maximal over the base of the heart. This condition usually causes more of a physiological strain than a ventricular septal defect resulting in an enlargement of the right side of the heart and fullness of the pulmonary vessels. Surgical repair of auricular defects is being done, but experience is still limited.

A patent foramen ovale should not be confused with a true auricular septal defect. In patency of the foramen ovale, blood cannot flow from the left auricle to the right auricle because of the valve on the left side. Except under abnormal conditions, blood cannot flow from the right to the left because the pressure in the left auricle is higher than in the right auricle. Therefore, a foramen ovale, although anatomically patent, is functionally closed and clinically of no significance.—From "Diagnosis of Congenital Cardiac Defects in General Practice," American Heart Association.

APPLICATION OF A MECHANICAL HEART AND LUNG APPARATUS TO CARDIAC SURGERY

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IT IS A PLEASURE to be here and to talk about a subject in which I have been interested for many years. The ultimate objective of my work in this field has been to be able to operate inside the heart under direct vision. From the beginning, I have not only been interested in the substitution of a mechanical device for the heart, but also for the lung. We have always considered congenital abnormalities of the heart the most suitable lesions for operative repair. Many of these abnormalities are septal defects. In the presence of a septal defect, shunting the flow of blood around one side of the heart with a pump, will not provide a bloodless field for operative closure of the defect. An apparatus which embodies a mechanical lung, as well as pumps, enables you to shunt blood around both the heart and lungs, thus allowing operations to be performed under direct vision in a bloodless field within the opened heart. Furthermore, an apparatus which embodies a mechanical lung enables you to provide partial support to either a failing heart or failing lung where a major operative procedure is not contemplated. Such an apparatus can also be used as an adjunct during the course of a major operative procedure. This partial support of the cardiorespiratory functions consists in removing venous blood from some peripheral vein continuously, oxygenating the blood and getting rid of the carbon dioxide in it and then injecting the blood continuously in a central direction in a peripheral artery. Of course, such partial circulation, or cardiorespiratory support, requires the use of a mechanical lung in the circuit.

I shall not describe in detail the entire apparatus. I shall merely discuss six aspects of the problem which I consider of fundamental importance. Four of these concern the apparatus itself, and two concern problems which arise on opening the heart and operating within it under direct vision.

The first feature of a mechanical heart-lung apparatus is a suitable pumping mechanism to move the venous blood from the subject, through the apparatus, and back into an artery of the subject. There is no real problem about a pumping apparatus. There are many ways of moving blood through tubing without producing significant amounts of hemolysis. We have used for many years a roller type of pump which does not contain any internal valves. Such pumps are extremely simple. Because of the absence of valves, the blood circuit is easy to clean and there are no stagnate regions where fibrin might be apt to form. There are many other advantages in this type of pump such as the simple and rapid control of the rate of blood flow. The pumps cause no significant hemolysis. In human patients in which we have used the apparatus, hemolysis has always been well below 100 mg. of free hemoglobin per 100 ml. of plasma. In animal experiments, hemolysis is similarly minimal.

The second main feature of a mechanical heart-lung apparatus is the mechanical lung itself. This presents far more difficulties than pumping blood. I am sure that the most efficient apparatus for performing the functions of the lung has not yet been devised. Our present mechanical lung, however, provides a reasonably satisfactory working solution to this problem. The mechanical lung performs the gas exchange required for respiratory function by filming blood on both sides of screens which have a somewhat larger mesh than ordinary fly screens. These screens are made of stainless steel wire and are suspended vertically and parallel in a plastic chamber. As the blood flows over these screens, it takes up oxygen and gives off carbon dioxide. It should be remembered that it is equally important to remove carbon dioxide from the blood as it is to add oxygen. It is easy to observe that sufficient oxygen is being picked up in the apparatus, as the blue blood entering the oxygenator becomes red as it leaves. This can be determined more accurately, of course, by intermittent sampling or by continuous reading with a Wood cuvette and oximeter. On the other hand, there is no way of estimating the carbon dioxide tension by observing the color of

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the blood. We have solved this problem by reading continuously the pH of the blood as it leaves the oxygenator. As there is no significant increase in fixed acids in the blood in the course of these experiments, the pH changes are due practically entirely to changes in carbon dioxide tension. We have an automatic control which keeps the carbon dioxide tension at the desired normal level and which is operated by any change in the continuously recorded pH level of the blood.

A third important feature of any apparatus which temporarily performs the function of the heart and lungs is constancy of fluid volume. The apparatus should at all times hold a constant volume of blood at any rate of blood flow. If the apparatus is not designed so as to hold a rigid volume of blood at all rates of blood flow, blood might accumulate in the apparatus with consequent depletion of the subject's vascular system and a dangerous drop in the subject's blood pressure. Similarly, if the apparatus should hold less blood at any time, there would be an excessive amount of blood in the subject's vascular system. Obviously the tubing in the blood circuit will always hold a constant amount of blood. There are two places in the circuit, however, where the blood volume might vary. One is in the blood reservoirs at the bottom of the plastic chambers which draw venous blood from the subject, and the other is in the thickness of the film of blood on the screens in the oxygenator. Rigid control of the volume of blood at the bottom of these plastic chambers has been obtained by an electronic device invented by Dr. B. J. Miller in our laboratory. This electronic device senses the level of the blood in these chambers and automatically operates the pumps which draw blood from the chambers. Thus when the level of blood tends to rise, the pump automatically operates at a faster rate. When the level falls the pump automatically is slowed. This electronic circuit has proven eminently satisfactory and maintains a rigid volume of blood in these chambers.

In the second place where blood might accumulate, on the screens of the oxygenator, a very simple way to avoid such an increase is by inserting an additional pump in the circuit which draws not only from the bottom of the mechanical lung but also from the tubing carrying venous blood from the subject. This additional pump operates at a rate which is always greater than the rate of blood flow from the venae cavae of the subject. As this pump operates at a fixed rate, the thickness of the blood on the screens in the oxygenator does not vary.

The fourth important requirement of any mechanical heart and lung apparatus is that the apparatus should remove all of the blood returning to the heart through the venae cavae and yet should not apply too great a negative pressure to the orifices of the cannulae because the venae cavae would then be collapsed around the orifice of these cannulae. We have found that the simplest way of obtaining such a smooth blood flow from the cavae is to interpose a suction chamber between the pump and the cannulae in the venae cavae. The degree of negative pressure can be easily regulated in this chamber and a smooth uniform flow of blood is easily obtained.

In summary, then, every mechanical heart-lung apparatus devised to take over temporarily the entire functions of the heart and lungs must comprise four essential features. First, there must be a good method of pumping blood through the circuit which does not cause hemolysis, which can be quickly and easily adjusted to varying flow rates and finally which enables the blood circuit to be easily and thoroughly mechanically cleaned. Second, the mechanical lung must not only fully saturate the blood with oxygen but must maintain the carbon dioxide tension of the blood at a normal level. The latter requirement may be taken care of by an automatic apparatus which continuously reads the pH of the blood leaving the mechanical lung and adjusts the carbon dioxide tension accordingly. Third, the apparatus must hold a constant amount of blood at all rates of blood flow. This can be accomplished by electronic control of the pumps removing blood from plastic chambers so that the pumps operate always to maintain a constant level of blood in the reservoirs at the bottom of the chambers. The thickness of the blood film on the screens in the oxygenator is kept constant by an extra pump which always circulates a constant flow of blood over the screens. Fourth such an apparatus must be able to remove smoothly all the venous blood returning to the heart through the venae cavae without collapsing these veins. We have found the simplest way of accomplishing this is to interpose a negative pressure chamber between the pump and the cannulae in the venae cavae.

There are two problems concerning operations upon the open heart which merit discussion. The first consists in the disposal of the blood returning to the chambers of the heart even though all the blood flow from the venae cavae is diverted to the apparatus. The second problem is the avoidance of air embolism when the heart is opened.

The first problem is not difficult to solve. When the entire functions of the heart and lungs are taken over temporarily by the mechanical heart-lung apparatus, the myocardium continues to receive its normal flow of oxygenated blood by way of the coronary arteries. This blood is returned to the interior of the heart by way of the coronary sinus and the Thebesian veins. This blood must be disposed of so that the operative held can be clearly visualized when the heart is opened. We have accomplished this by aspirating this blood into a special plastic chamber in which any air aspirated is dissipated. The blood collects at the bottom of this chamber and is pumped back into the main extracorporeal circuit free of air.

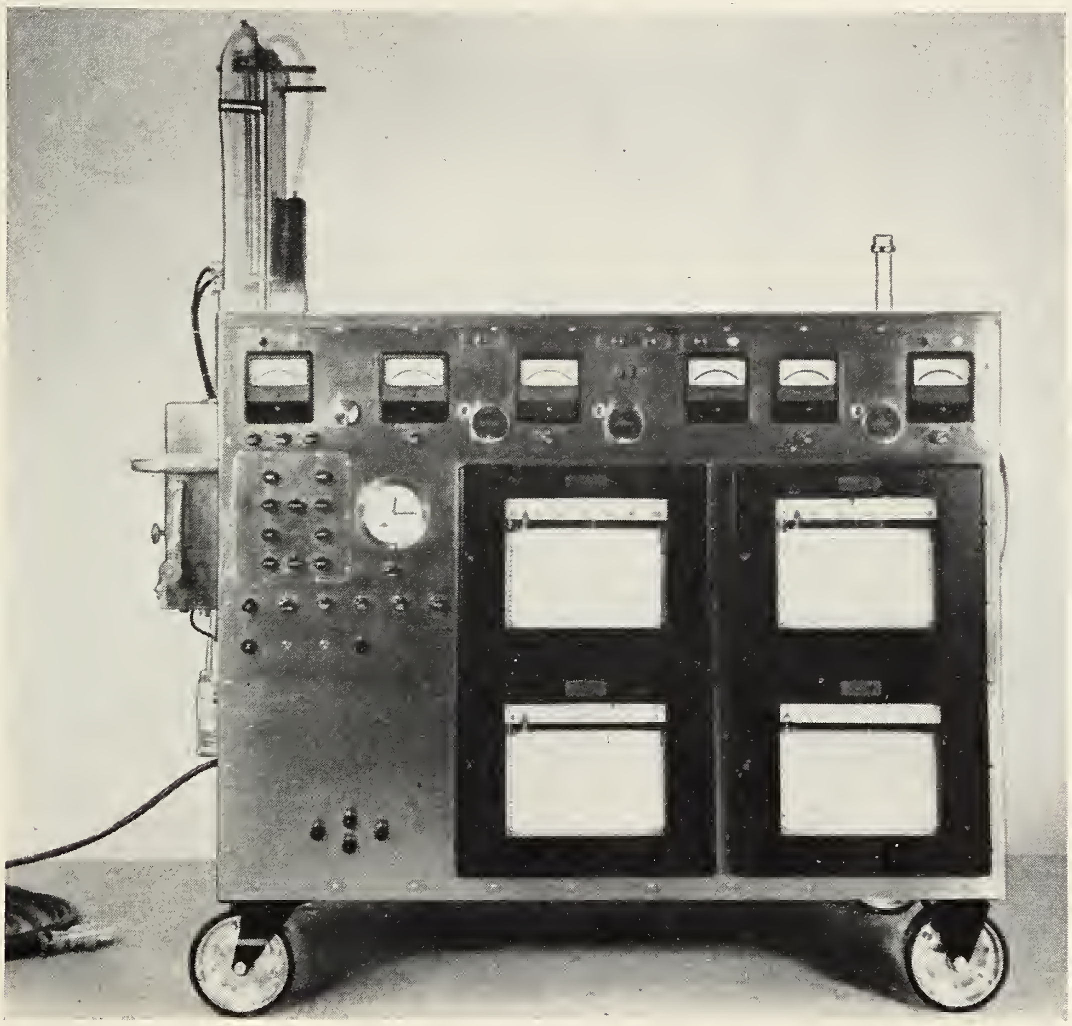

Fig. 1. Front view of apparatus showing the recording and control instruments and the lung suspended above the cabinet on the lett.

Air embolism must be avoided when the heart is opened. If there is no septal defect, operations upon the right side of the heart can be performed without any great danger from air embolism. It is easy to flood the chamber of the heart with blood or salt solution after the operation is completed so as to avoid air embolism. Small amounts of air in the pulmonary arteries are probably not significant. On the other hand, operations on the left side of the heart, or on the right side of the heart in the presence of a septal defect, present a real problem in the prevention of air embolism into the ascending aorta. The immediate result of such air embolism is usually blockage of the coronary arteries with ventricular fibrillation and death. Our solution of this problem is to insert a small plastic catheter through a stab wound in the apex of the left ventricle. Suction is applied to this plastic catheter during the course of the open cardiotomy so that any air or blood entering the left ventricle takes the path of least resistance out through this plastic catheter instead of being

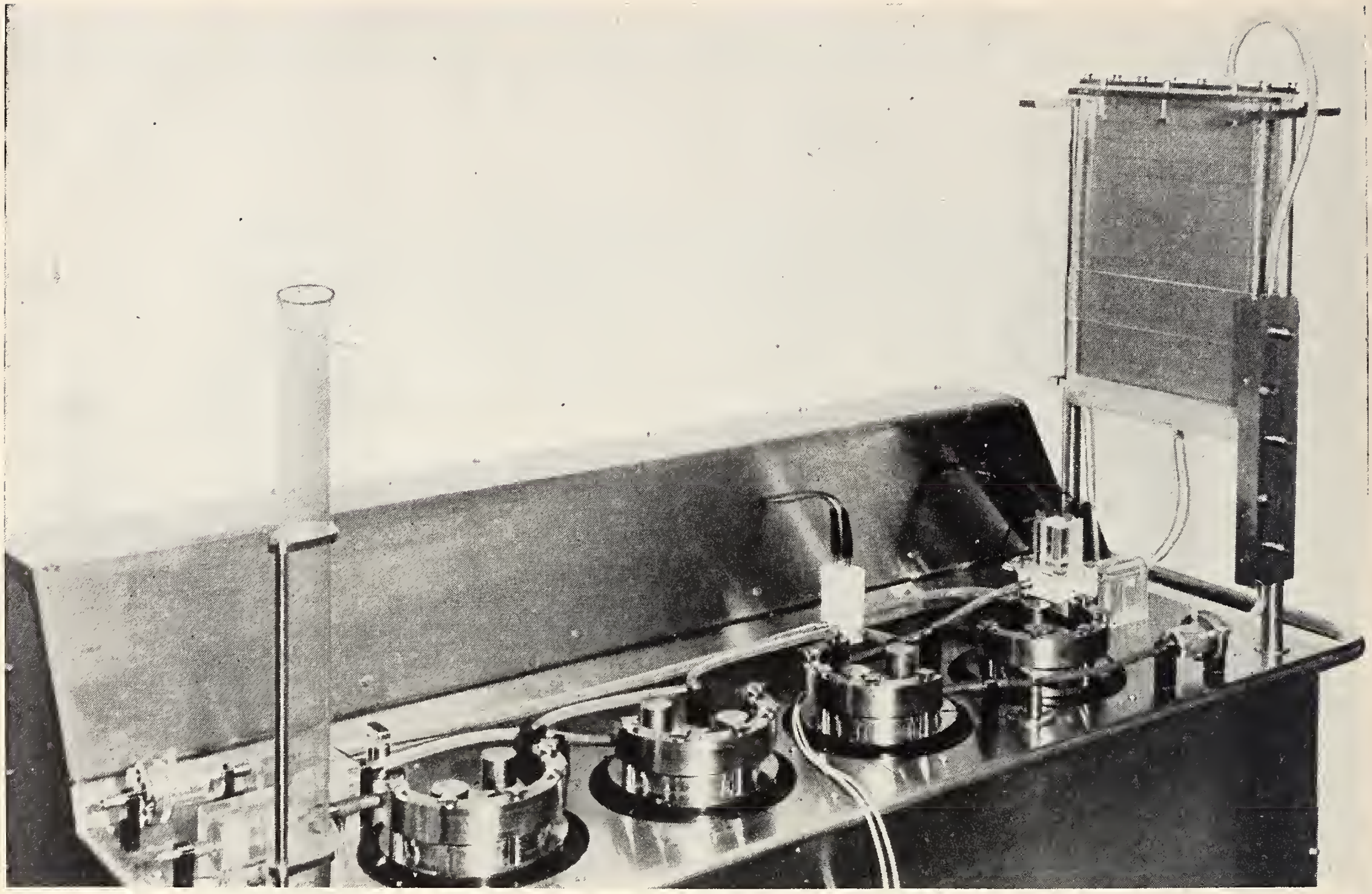

Fig. 2. Oblique rear view of apparatus showing the rotary blood pumps and the battery-type screen lung suspended above the cabinet on the right side.

ejected into the aorta. This plastic catheter is also connected to the debubbling chamber which receives the cardiac venous blood aspirated from inside the heart. Thus this blood is also returned to the circuit, and so to the subject, after the air bubbles are removed. After the wound in the heart is closed, and no further bubbles of air appear in the tubing connected to this catheter, it is removed and the small stab wound in the left ventricle closed by suture. Since employing this method in both animals and patients, we have had no instances of air embolism.

Figure 1 shows the front view of the apparatus which I have described, with the recording and control devices on the front panel. Figure 2 shows the rear view with the roller type pumps on top of the cabinet. Two rollers on a revolving arm pass over the rubber tubing which is clamped in a semicircular position. The rollers move the blood through the tubing and the blood cannot flow back because there is always a roller compressing the tube. To the right of Figure 2 is the mechanical lung which consists of vertical screens suspended in parallel in the plastic case. Blood passes onto the screens through slits at the top of the lung. The blood collects in the bottom of the plastic case as it leaves the screens. The pump returning the oxygenated blood to the subject is automatically controlled by the electronic device which senses the level of the blood at the bottom of the plastic case, through which the blood passes. The lucite block near the oxygenator contains glass and calomel electrodes which continuously measure the pH of the blood. The filter consists of a screen with wires 150 microns in diameter and a 300-micron mesh. We do not know whether such a filter is necessary before returning the blood to the subject. However, we regard it as a good safety precaution in human patients. The tube at the end of the apparatus returns the blood to the patient through a cannulae directed in a central direction in an artery. In human patients we employ the central end of the divided left subclavian artery. The other two tubes are connected with cannula which are inserted into the superior and inferior venae cavae.

Oxygen is blown over the screens suspended in the plastic case. Thus the blood film on the screens is exposed to an atmosphere of pure oxygen. The lung shown in Figures 1 and 2 has six screens. We have a larger mechanical lung with eight screens which are of longer length and which we have used on adult patients. An additional pump draws blood both from the bottom of the oxygenator and from the tubing containing the venous blood coming from the patient. The pump maintains a constant rate of flow through the oxygenator so that there is no variation in the thickness of the films on the screens. The electronic control circuit maintains the blood level in the bottom of the plastic case constant at all rates of flow. The pump removing the blood from this chamber is controlled by this electronic device.

There are two plastic negative pressure chambers. One of them collects the blood from the venae cavae and the other (Fig. 3) is the debubbling chamber in which the blood from the cardiac veins and from the left ventricle is collected. As the blood passes down the sides of this plastic chamber any bubbles of air are dissipated. Last spring we reported the successful repair of interatrial septal defects in animals using a flap of pericardium. We have been prepared to use such a flap of pericardium in human patients. We found, however, as Swan has, that it is quite easy to close such defects with a continuous suture in the open heart under direct vision and that consequently a pericardial graft is not needed.

The inferior vena cava is cannulated by a "tygon" tube passed into the inferior vena cava by way of the right atrial appendage. The superior vena cava is cannulated by a "tygon" tube passed through a stab wound in the right atrial wall. Oxygenated blood from the apparatus is pumped into the aorta through the divided central end of the left subclavian artery. Ligatures passed around the superior and inferior venae cava are tied over the enclosed cannulae. This diverts all the venous blood to the extracorporeal blood circuit. In addition to closing atrial septal defects in dogs with the pericardial graft, we have successfully closed interventricular defects in dogs by direct suture. The defect is exposed by an incision in the anterior wall of the right ventricle parallel to the left anterior descending coronary artery.

There has been a progressive decline in operative mortality in successive series of animals operated upon in our laboratory. Three years ago we had an 80 per cent mortality. In the most recent series this mortality has declined to 12 per cent. By operative mortality we mean any death which could be attributed to the operation and any death occurring the first month after operation. We have successfully temporarily taken over the entire blood flow through the heart and lungs of a dog for as long as one hour and forty minutes with prolonged survival in a healthy condition.

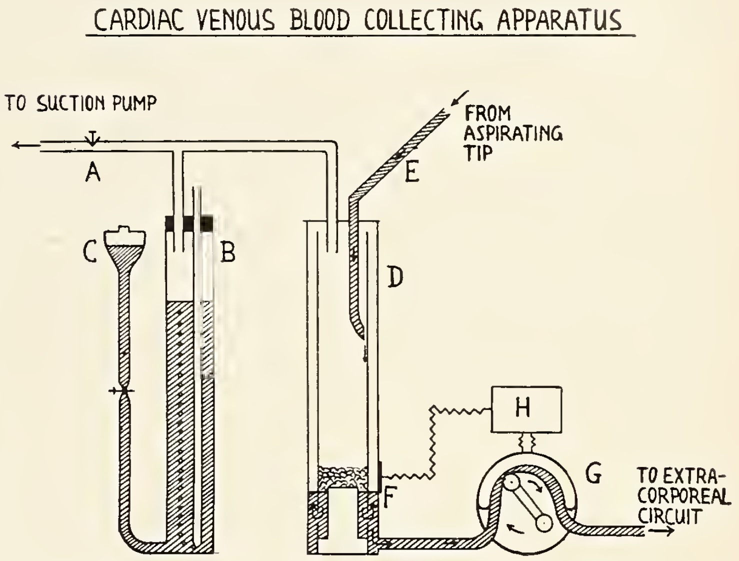
CARDIAC VENOUS BLOOD COLLECTING APPARATUS
TO SUCTION PUMP
FROM ASPIRATING TIP
A
E
C
B
D
H
G
F
TO EXTRA-CORPOREAL CIRCUIT

Fig. 3. Diagram of the cardiac venous blood collecting apparatus. The diagram is self-explanatory. The rotary pump G returns the blood to the main extracorporeal circuit after it has dissipated its bubbles in the inner chamber of cylinder D.

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The average saturation of the venous blood entering the apparatus is 63 per cent. We regard a normal saturation of venous blood with oxygen as the best indication of an adequate blood flow to the tissues. If the saturation of venous blood with oxygen falls to low levels it is obvious that there is an inadequate blood flow to the tissues. We have been successful in maintaining the pH in a normal range. The hemolysis in this group of experiments averaged 35 mg. of free hemoglobin per hundred ml. of plasma. You are all aware that this is an insignificant amount of hemolysis and that a similar degree of hemolysis can occur if blood is forced rapidly through a fine needle. There is only slight increase in the fixed acids, generally in the neighborhood of three millimoles per liter which is within the normal diurnal variation.

Now I suppose what you are all interested in is not how many animals we have successfully operated upon but how many humans we have operated upon. The details of our animal experiments have appeared in two articles recently published. The human patients we have operated upon, using the apparatus, have not yet been reported.

We have used the apparatus to carry temporarily the entire cardiorespiratory functions of four human patients. The first was operated upon a year and one-half ago, and the last in July, 1953, of this year. We have one surviving patient who is quite well in every way with complete closure of an atrial septal defect. The three deaths have all been due to human error and not to failure of the apparatus.

The first patient, who was operated upon a year and one-half ago, was a fifteen-month-old baby that weighed eleven pounds and was in severe congestive cardiac failure. Attempts at cardiac catheterization in this baby were unsuccessful. It was the opinion of everyone who saw this baby that the cardiac abnormality was an interatrial septal defect. We explored the right side of the heart using the apparatus and discovered that no atrial septal defect existed. The child died after operation and at postmortem was shown to have a huge patent ductus arteriosus which had not been recognized at the time of operation. This, of course, illustrates the importance of complete exploration of every heart which is operated upon. We might have saved this child's life if we had closed the ductus.

The second patient was operated upon May 6, 1953. She was an eighteen-year-old girl who had a large interatrial septal defect proved by cardiac catheterization. The patient had been symptom free until about six months before operation, when she began to show symptoms of right- sided heart failure. She was hospitalized three times in these six months. Every time she returned to ordinary activity, she had symptoms of heart failure. Cardiac catheterization revealed an atrial septal defect with a left to right shunt through the defect amounting to nine liters per minute. The patient was connected with the apparatus for forty-five minutes and for twenty-six minutes all cardiorespiratory functions were maintained by the apparatus. She had a large interatrial septal defect which was quite easily closed with a continuous silk suture. The patient's postoperative convalescence was uneventful. She was readmitted to the hospital in July, 1953. At this time, cardiac catheterization showed that the septal defect was completely closed and that there was no evidence of any shunt. The cardiac murmur had completely disappeared and she was in good health. We believe that a transverse incision extending from one axilla to the other opening both pleural cavities through the fourth interspace, and dividing the sternum, gives the best exposure for this type of cardiac operation. The chest wound heals quite solidly and results in an inconspicuous scar beneath the breasts.

The last two patients were operated upon in July, 1953. They were both underdeveloped girls aged five and one-half years. Each of them weighed only about thirty pounds. The first child had a large interatrial septal defect proved by cardiac catheterization. Cardiac arrest occurred after we had opened the chest but before we had cannulated any vessels. The heart became blue and dilated as the chest was being explored. We tried for one hour to establish normal cardiac contractions but were unable to do so. We then rather reluctantly cannulated the superior and inferior venae cavae and the left subclavian artery while an assistant massaged the heart. As soon as the patient was connected with the apparatus the heart action became strong and the color of the heart pink. We then opened the right atrium and repaired five separate defects in the interatrial septum. After the defects were closed and the heart wound sutured, the ligatures around the venae cavae were cut allowing the heart to take over part of the circulation. Whenever we stopped the artificial support by the machine, the heart dilated and began to fail. Partial support by the extracorporeal blood circuit was maintained for three or four hours at the end of which time the cannulas were withdrawn and the chest was closed. The patient's heart, however, dilated and cardiac arrest occurred. Death, of course, in the patient cannot be attributed in any way to the use of the heart-lung apparatus as cardiac failure occurred prior to the use of the apparatus. Perhaps the dilatation of the heart and cardiac arrest was the result of a reversal of the shunt through the interatrial defect due to blood transfusion which was given during the early part of the operation.

The second five and one-half-year-old child had a proven interatrial septal defect by cardiac catheterization. It proved impossible, however, to pass the catheter into the right ventricle. On clinical grounds an interventricular septal defect was thought to exist in addition to the interatrial defect. It was known that the patient had a left superior vena cava which was somewhat larger than the right superior vena cava. The child turned out to have not only a huge interatrial septal defect but also a large interventricular septal defect and a small patent ductus arteriosus. Cannulation proceeded normally in this child and when we opened the right atrium we found it to be flooded with bright red blood returning to the atrium through the tricuspid valve. Ae we could not get a clear field to work and the flow of bright red blood was so excessive, we closed the atrium and removed cannulae. The child died after the operation, which was to be expected due to our failure to correct any of the cardiac defects.

(Motion picture shown)

This motion picture was taken during the course of the operation in which the five separate interatrial defects were sutured. It illustrates the value of being able to visualize the interatrial septum. It seems to me that the four smaller defects might have been missed by the employment of indirect blind methods. The film clearly shows how simple it is to keep the operative field clear of blood and that the heart appears of normal color and is beating well because the myocardium is receiving oxygenated blood from the apparatus through its coronary vessels.

In conclusion, I would like to say that I think the work I have reported is some of the early work in this field and that there is considerably more work to be done. It seems to me that there will always be a place for an extracorporeal blood circuit because it permits a longer safe interval for opening the heart than can ever be obtained by any of the hypothermia methods.

Discussion

Dr. F. D. Dodrill, Detroit, Michigan—You have just heard an excellent review of the mechanical heart work by the leading pioneer in this country. Dr. Gibbon has had vastly more experience than the rest of us in this field, and I am envious of his vast accomplishments in this work.

When I first began this work several years ago, it was not definitely known, nor is it now definitely known, just how this can best be done. It was originally thought by those working in the field one could not bypass either side of the heart without completely bypassing the entire heart and lungs at the same time. As time has gone on and various workers have made definite contributions along this line, it is now apparent that either the right side or the left side of the heart may be bypassed while the opposite side of the heart and lungs continue to perform their functions.

We began a few years ago, therefore, to explore the possibilities of the following types of procedures: (1) bypass of the right heart, (2) bypass of the left heart, (3) bypass of both sides of the heart using the lungs for oxygenation, and (4) bypass of the heart and lungs using the mechanical oxygenator. We have performed more than 100 operations on the experimental animal doing one of these various procedures.

Our apparatus is so constructed that it can perform any or all of these functions. Insofar as the mechanical heart itself is concerned, I feel there are three factors which are important. These are: (1) it should be strong, sturdy and not subject to breakdown, (2) it should be so constructed that all parts coming in contact with the blood can be sterilized by ordinary autoclaving, and (3) it should produce and maintain a pulsatile flow in the vascular system.

Bypass of the Right Heart.—Our experimental work in bypass of the right heart has been highly successful in the experimental animal. This consists in taking the blood from the superior and inferior vena cava, passing it through the right side of our mechanical heart and back into the pulmonary circuit through the artery to the right lower lobe of the lung. By this method, the right side of the heart can be bypassed while the opposite side and the lungs continue to perform their functions. Also, if one wishes, the blood may be taken from the right atrial appendage. By this method not only the superior and inferior vena cava flow, but the coronary sinus flow, as well, can be completely diverted from the right side of the heart. These connections depend upon the type of procedure and the exposure which one is attempting.

In our experimental work on the right-sided bypass, we encounter severe hypotensive reflexes. These hypotensive reflexes are greatly increased by even the slightest degrees of anoxia. This reflex has been referred to as the Bainbridge reflex but it may be possible, that additional reflexes may be present. It is my impression that a great deal of physiological investigation is needed along this line. On the left side, however, these hypotensive reflexes are much less acute and oftentimes in the experimental animal we see no evidence at all of a decrease in the blood pressure during a left-sided bypass. Using such a procedure, any purely right-sided heart disease may be exposed and corrected under direct vision. The examples of such conditions are pure pulmonary stenosis with an intact cardiac septa and diseases of the tricuspid valve also with an intact septa. Figure 1 illustrates the pulmonary valve in a patient with pure pulmonary stenosis during a right-sided bypass. This valve was exposed for a period of twenty-five minutes while the mechanical heart pumped approximately 4½ liters of blood per minute. The left side of the heart continued to perform its function as did the lungs. While the right-sided bypass of the heart seems to be a rather practical procedure, it should be pointed out that there are not many pathological conditions of the right side of the heart alone in which such a procedure can be used. The great majority of patients with pulmonary stenosis have a defect in either the intratrial or interventricular septum and in such a patient a unilateral bypass is, of course, out of the question. There are rare instances of large emboli lodging in the right side of the heart which give some warning before death and it is possible that such large emboli could be removed during a right-sided bypass.

A right-sided bypass is a practical procedure for patients with pure right-sided disease. There is no need at all for bypassing the lungs as well for exposure of a purely right-sided defect. The main advantage of using the mechanical lung along with the mechanical heart is that the connections of the apparatus to the anatomical structures are much easier. However, with a right-sided bypass, the connections are likewise easy and should present no obstacle at all to the operating surgeon. Of all these various procedures which can be done in the way of

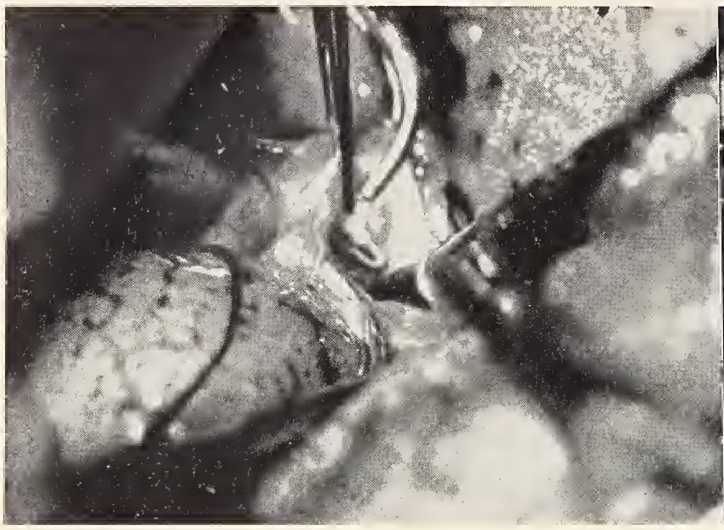

Fig. 1. Stenotic pulmonary valve in a seventeen-year-old boy.

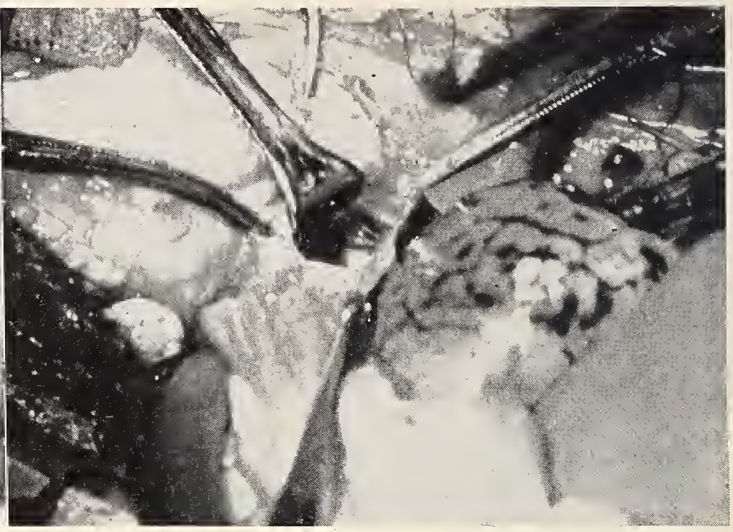

Fig. 2. Open left strium exposing the mitral valve in a fifty-year-old woman.

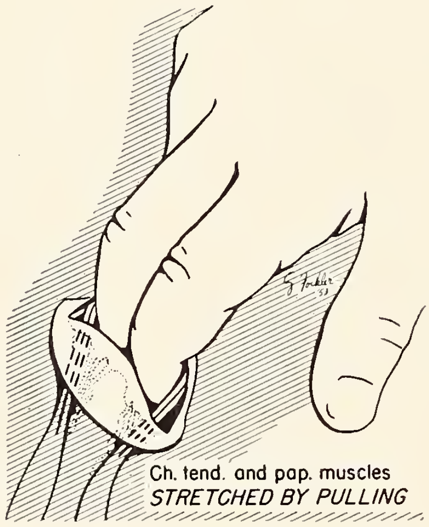

Fig. 3. A method of elongating the chordae tendineae in a patient with mitral regurgitation.

bypassing the entire heart or a portion thereof, I strongly feel that a right-sided bypass alone is a practical procedure which will be useful in years to come. The pathological conditions, however, are rather limited for this type of a procedure.

Bypass of the Left Heart.—A great deal of animal experiments have been done on the left-sided bypass. This consists in taking the blood from the pulmonary veins, passing it through the left side of the mechanical heart and back into the aorta which is its ultimate destination. The experimental work on dogs is extremely gratifying. We have been able to expose the mitral valve in dogs for fifteen consecutive times without a fatality. During the left-sided bypass, the right side of the heart and the lungs continue to perform their functions. During such a procedure, it is necessary to maintain a sufficient systemic blood pressure and preferably to maintain it by a pulsatile flow. The hypotensive reflex which is rather acute on the right side, especially if there exists any degree of anoxia, is not so acute on the left side. In numerous animal experiments, we have not encountered such a reflex at all. We have applied this procedure to a few patients. Figure 2 illustrates the open left atrium with the mitral valve in the depths of the cavity. This patient made an uneventful recovery and is much improved from the surgical procedure. It seems logical to assume that if the mitral valve can be exposed, there is more apt to be an opportunity to improve valve function especially in mitral regurgitation. Figure 3 illustrates the elongation of the chordae tendineae in a patient with mitral regurgtation during a left-sided bypass. Although, the valve and the chordae were not visualized, the arterial pressure pattern shows conclusively that the mechanical heart was completely maintaining the systemic circulation. It has now been over one year since this patient was operated and he is vastly improved, even to the point of almost a complete disappearance of his loud murmur. One additional patient has also been treated in a similar manner with encouraging results. There are several factors in the production of mitral insufficiency; however, the most important one seems to be the shortening of the chordae tendineae and sometimes the papillary muscles as well. The valve leaflet itself may be nearly normal, and if the chordae can be lengthened to the point where it permits valve closure, the regurgitation is markedly diminished or corrected. Oher types of procedures have been done during a left-sided bypass. The aortic valve may be manipulated by inserting a finger into the left atrial appendage down through the mitral valve and upwards through the aortic valve. A patient with aortic stenosis has been so treated and the valve was easily fractured with the finger. Whether or not this procedure will be better than the method of going directly through the left ventricle remains to be seen.

Bypass of Both Sides of the Heart.—A bypass of both sides of the heart using the lungs for their natural functions is a more difficult procedure from the technical standpoint. We have been able to do this, however, quite satisfactorily in experimental animals and have applied it to one patient. Both sides of the heart were completely taken over by the mechanical device. A stenotic pulmonary valve was corrected and the interventricular septum was exposed. Unfortunately, this patient had, in addition to the pulmonary stenosis, marked infundibular stenosis and could not be sufficiently corrected. During the course of the procedure, the systemic blood pressure as well as respiratory function were quite satisfactory. Unfortunately, the patient succumbed on the fourth postoperative day from atelectasis and pneumonitis.

A Combination of Hypothermia and the Mechanical Heart.—These various procedures which I have shown you have been done solely with the mechanical heart. We are now working along the lines of the combination of hypothermia and the mechanical heart. Whether or not various types of intracardiac surgery will be done under hypothermia alone, using the mechanical heart alone or a combination of these two methods remains to be seen. The advantage of combining hypothermia with the mechanical heart is that during hypothermia, the circulating blood volume is markedly reduced and the work of the mechanical heart is vastly decreased. Moreover, with the circulation of such a small volume of blood, only one lobe of the lung needs to be used for respiration. Such a small quantity of blood can be easily circulated through one lobe of the lung while the rest of the lungs are at rest. This vastly minimized the technical difficulties with the use of the mechanical heart alone. It is easy to cannulate one of the upper pulmonary veins, to pass the blood through the left side of the mechanical heart and back into the aorta. The right-sided connections for such a procedure are similar to a complete bypass of the heart and lungs and consists simple of cannulating the superior and inferior vena cava and passing the blood back into the pulmonary circuit. The lung is a very intricate organ. It is not known but that it may perform other functions aside from the uptake of oxygen and the release of carbon dioxide. It seems as though a good hard attempt should be made at using the lung for its natural function before we completely discard it.

Dr. Clarence Dennis, Brooklyn, New York.—After Dr. Maurice D. Visscher and Dr. Owen H. Wangensteen jointly suggested to me, late in 1945, the undertaking of a project to develop an artificial heart-lung apparatus, I very quickly became aware of the activities of Dr. John H. Gibbon, Jr., and his group in Philadelphia. I consulted him at that time, and was very cordially received, shown protocols, shown the apparatus as it had developed up to that time, and welcomed into the group of those working on this project. Between that time and this, there have been associated in the research project with Dr. Karl E. Karlson and me approximately eighteen physicians. Work has been carried out first at the University of Minnesota and more recently at the State University of New York, and has been supported by the United States Public Health Service, the Graduate School of the University of Minnesota, the State University of New York, the Life Insurance Medical Research Fund, and private sources. In our search for an answer to this problem, my associates and I have come to have the very highest regard for the ingenuity, integrity, and cordiality of Dr. Gibbon and his associates. Success could not have come to finer people.

Dr. Gibbon has very carefully listed the qualifications of a satisfactory artificial heart-lung apparatus. We have been concerned with an additional qualification, namely, the ability to render the whole apparatus absolutely free of bacterial contamination. The reason for our preoccupation with this qualification arises from the work of Dr. Russell M. Nelson, who was a member of our research group three years ago. Dr. Nelson showed that our apparatus at that time was contaminated with the paracolon bacillus, and that injection intravenously into normal dogs of a re-suspended fourteen-hour culture of this organism resulted in a symptom complex which had been responsible for most of the deaths prior to that time. This symptom complex developed rapidly and killed some dogs in less than three hours. The affected dogs showed a rapidly progressive metabolic acidosis, an incoagulable state of the blood, development of effusions into the serous cavities, massive gastrointestinal hemorrhage, and death in shock.[3] Satisfactory sterilization of the apparatus removed this symptom complex from our list of problems. The machine which we formerly used could not tolerate autoclaving, and sterilization by formaldehyde proved fairly regularly to be incomplete. Nevertheless, it was possible for us to do a long series of animal perfusions with this old apparatus, with no more than a 10 per cent mortality attributable to the perfusion *per se*.[1]

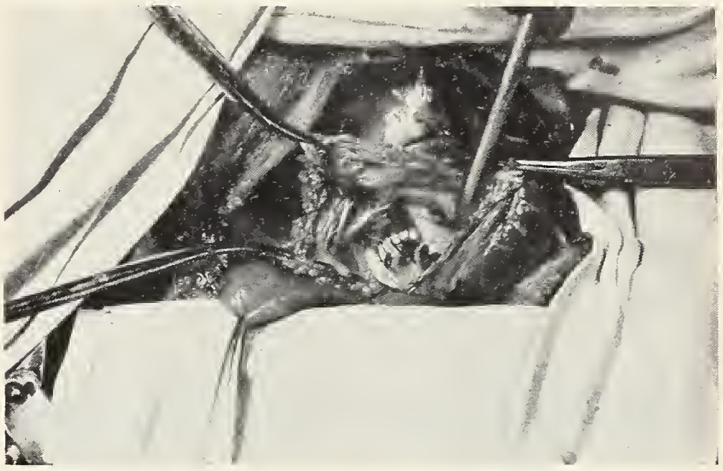

Fig. 1. Closure of inter-atrial septal defect, May, 1951. Closure was easily performed with interrupted silk sutures under direct vision. The patient suffered air embolism before opening the atrium, a consequence of human error in failure to switch on an automatic control mechanism. She died of complication of air embolism after several hours of partial support by the machine.

We gained so much confidence with this apparatus that, in conjunction with Dr. Richard Varco, we perfused two patients in efforts to achieve satisfactory closure of inter-atrial defects.[2] We were unsuccessful in salvaging either of these patients, but it was the consensus that the failure was not due to any intrinsic defect in the apparatus, but rather to human errors. The repair which was accomplished in the second patient is shown in Figure 1. This patient was lost as a consequence of human error, namely, failure to turn on the automatic control apparatus, which had been designed and proven adequate to maintain the desired blood level in the reservoir of the oxygenator. Air embolism occurred because of this oversight. The patient survived approximately eight hours after the procedure, but only by virtue of partial support by the pump-oxygenator, and the closure at the time of autopsy was perfectly adequate. Dr. Gibbon's observation is in agreement with ours, namely that the margins of most inter-atrial septal defects in clinical patients are thick, and firm, and silk closure, therefore, is not usually difficult of accomplishment. Closure in this instance, in the summer of 1951, was very easily accomplished.

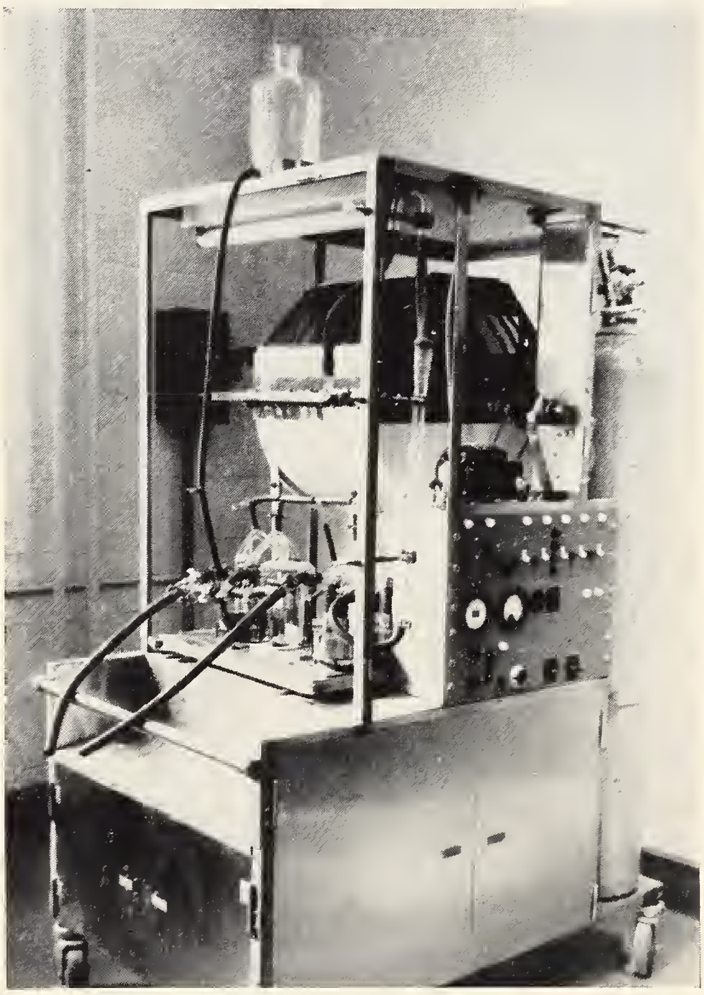

Fig. 2. The present pump-oxygenator apparatus. The entire blood-bearing unit can be autoclaved after assembly. Oxygenation is accomplished by filming on steel mesh discs which are mounted on a rotating horizontal shaft. The pumps are a modification of the Dale-Shuster pattern. Risk of human error has been reduced by use of largely automatic controls.

Because of our concern over the occurrence of human errors and because of our concern with regard to absolute sterility, we have spent the past year and a half in the construction of a new apparatus, the blood-bearing portion of which can be assembled and autoclaved as a unit. This apparatus is not yet complete, but has reached a stage of construction which has permitted us to employ it for approximately two dozen trial perfusions. The apparatus in question is indicated in Figure 2. In this apparatus, oxygenation is accomplished by the filming of blood near the center of a 50 cm. disc of stainless steel mesh which is mounted on a slowly rotating horizontal shaft. It will oxygenate 500 to 600 cc. of blood per minute from half saturation to full saturation and has a volume content of approximately 65 cc. of blood at any given moment. The pumps are a modification of the Dale-Shuster pattern. They are activated by hydraulic pressure, which is produced by a mechanical cam and bellows arrangement below the surface of the table. The apparatus as a whole will handle about 5 liters of blood per minute. It has been calculated that it should be capable of adding in excess of 350 cc. of oxygen per minute and in perfusions in large dogs has been measured to add 210 cc. per minute. We now regularly observe sterile blood cultures at the end of perfusion, and hemolysis produces approximately 1 mg. per cent of plasma hemoglobin per minute of perfusion.

There are many problems which remain and which are the source of considerable concern at the present time. It has been found that our greatest problem is that of postoperative hemorrhage. A technique of protamine titration, which has been employed over a period of several years in our laboratory, indicates that the addition of protamine to the animal is effective for only an hour or two and that additional protamine thereafter must be added until approximately six hours have passed. The platelet count is not appreciably lowered, and this, therefore, is not the reason for our hemorrhagic difficulties. It is suspected that there is excessive trauma to the blood which may be responsible for the degree of hemorrhage which we have experienced, but the low plasma hemoglobin concentrations cast a doubt even on this suggestion. Preliminary studies suggest fibrinolysin as the offender.

Perhaps our major remaining problems with the new machine rotate around the completion of satisfactory automatic controls. We have, at times, utilized the suggestion of Professor Dogliotti of Turin, Italy, that the pumping of blood from the machine to the arterial system of the subject be governed entirely by the arterial blood pressure of the subject. At other times, we have maintained a constant volume in the extra-corporeal circuit. The latter appears to be the preferable arrangement. As far as controls are concerned, the removal of blood from the venae cavae has been simplified by setting the apparatus up in such fashion that the pumps fill by gravity from the vena cava.

There remain several metabolic problems that have to be studied. An occasional perfusion is characterized by a profound loss of circulating plasma sodium. An occasional perfusion is also complicated by development of marked metabolic acidosis, even when there is a negative blood culture. Finally, there is occasionally a profound disappearance of circulating protein, which, as yet, we have not been able satisfactorily to explain.

It is likely that Gibbon has better solutions to many of these problems than we, but all of them must be painstakingly resolved before utilization of pump-oxygenators in clinical surgery can become general.

(References on Page 185)

APPLICATION OF HYPOTHERMIA TO CARDIAC SURGERY

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THIS REPORT contains a brief review of our early work on the physiology of hypothermia. Following that an assessment is made of the present status of research on hypothermia and some aspects of its clinical application to cardiac surgery are discussed.

General hypothermia is being investigated as a means of reducing the oxygen requirements of the body sufficiently to allow exclusion of the heart from the circulation and thus permit intracardiac surgery under direct vision. Ordinary adult laboratory animals cannot be cooled safely to a body temperature much below 20°C. These studies are concerned with changes between 38°C. and 20°C., during surface cooling and rewarming.

In dogs, the oxygen consumption steadily declines with a fall in body temperature, provided the shivering reaction is carefully controlled and arterial oxygen saturation maintained. At 20°C., it is approximately 15 per cent of normal. The picture on rewarming is much the same, with a similar gradual increase in oxygen consumption as the body temperature rises.

In cardiovascular studies, the blood pressure fell slowly to approximately 24°C., below which temperature, the fall became more rapid. Heart rate and cardiac output showed a more progressive fall during cooling. The cardiovascular state returned to normal on rewarming. The venous pressure rises slightly during cooling. If the positive pressure artificial respiration is vigorous and over a long period there is a greater rise in venous pressure. The electrocardiogram shows typical changes and reverts to normal on rewarming.

We have been asked whether low body temperatures may not have a deleterious effect upon cerebral function. In continuous electro-encephalograms in monkeys the waves diminished and there was often no evidence of cortical activity at 20°C. There were no wave forms suggesting cerebral anoxia and normal tracings re-appeared on rewarming. Behavior tests before and after cooling, showed no evidence of cerebral damage. These tests were not repeated following a period of interruption of the circulation.

In the biochemical study there were no serious electrolyte changes noted with our original cooling techniques. Small changes in potassium and calcium were not considered significant, although an increase in potassium sensitivity at low body temperature has already been reported in the literature.

It was known that low body temperatures cause a shift in the oxygen dissociation curve to the left and presumably the oxygen becomes less available to the tissues. In order to offset this, the CO_2 content of the blood, originally, had been increased during cooling and a pH slightly below normal was maintained in order to balance this effect. In a complete study of the acid base mechanism, it was found that the pH of the blood could be varied depending on the rate of positive pressure artificial respiration with pure oxygen during cooling. Contrary to our original premise, the animals appeared to tolerate low body temperatures with greater safety if their pH was maintained above normal. This was judged by a reduced incidence of cardiac arrest on cooling a series of animals to 18°C.

During actual surgery on the heart at low body temperatures it was a difficult situation to find oneself with the chest open at low body temperature and the heart in standstill. It is awkward and impractical to rapidly rewarm an animal with the chest open. Primarily to meet this situation, an electrical artificial pacemaker was designed which stimulates the sino-auricular node through an intravenous electrode producing heart beats with sustained blood pressure. This makes it possible to close the chest and rewarm the animal to a temperature where his own nodal rhythm will return. This electrical pacemaker has been combined with a defibrillator in a single resuscitation unit and has been in use experimentally and clinically for over two years.

As a further development of the technique of

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resuscitation from low body temperatures, a radio-frequency technique of rewarming was designed. It had been felt that this may be more practical in the operating room of hospitals, particularly when dealing with adult patients and would very likely prove an advantage from an esthetic point of view. The chief reason for developing this method for rewarming of animals however, was an attempt to prevent the "rewarming shock" which often developed upon rewarming in water at 40°C., and for which no adequate explanation, as yet, has been given. This is an inductive type of heating in which the animal or patient lies between two insulated coils. This produces a simultanous rewarming of deep and superficial structures and is an attempt to prevent premature dilation of the peripheral vascular bed.

In applying this principle surgically, our earlier results with dogs were only moderately encouraging. In a later series, using monkeys that had been cooled to a body temperature of 17°C., to 19°C., it was found possible to exclude the heart from the circulation and open it for fifteen to twenty-four minutes, with survival in twelve or thirteen experiments. Since these animals showed no changes in personality or behavior after this experience, it was judged that there had been no serious ill effects to the cerebral function. In this series of experiments a small amount of Pentothal anesthetic was given intravenously and they were cooled by being placed in cooling blankets containing coils with circulating refrigerant and 0°C. They were removed from the blankets about 2°C., above the temperature at which the operation was to be performed, as there is always a drift down following removal due to the temperature gradient which exists with surface cooling. With experience this drift can be assessed beforehand as it appears to depend upon two factors, namely: (a) the rate of cooling and (b) the weight of the animal or patient.

Both the right and the left side of the heart were opened in these experiments. They were merely token procedures and no specific intracardiac maneuvers were carried out. The field was dry at that temperature and the coronary inflow, which is a serious technical problem with the extracorporal technique, is at a minimum and usually represented by a slow ooze. This of course is because the arterial blood pressure is not maintained during the period which the circulation is interrupted.

Both the warm water and the radio-frequency technique of rewarming were used and the rate of rewarming in each is about equal. These animals were active within two hours of their return to normal body temperature. There appeared to be a higher incidence of wound infection than one would expect from a similar series of thoracotomies at normal body temperature.

Hibernation.—In all this work, the limiting factor in general hypothermia has been the development of cardiac arrest as either venticular fibrillation or cardiac standstill in adult dogs and other laboratory animals once the body temperature falls below 20°C. Even a body temperature of 20°C., is not completely safe in dogs. On cooling a series of adult dogs at this temperature, with positive pressure respiration, a small percentage will die of cardiac arrest before they reach that level. The statistical results in such a series will depend upon the breed and size of the dog, and in laboratories where the animal cages are not in properly air-conditioned rooms, the results may vary with the season of the year. One may expect better results from cooling, if the animal has been living in a cold environment.

By far the most important factor is age. Young animals display a much better tolerance to low body temperatures and Adolph has shown that infant animals may be cooled to temperatures as low as those that are tolerated by hibernating animals (3° to 5°C.). We have cooled a ten-week-old puppy to 10°C., with survival, so it would appear that animals and very likely humans, lose this tolerance to low body temperatures as they mature.

These facts have afforded a stimulus to investigate ways of recapturing this early characteristic. Evidence is available which indicates that infant humans possess a similar remarkable tolerance to low body temperature which presumably is lost with age and maturity.

For several years our team has tried various biochemical agents and technical procedures in order to make cooling to low temperatures a safe procedure and eliminate the complication of cardiac arrest. These many tedious experiments have met with only a limited improvement in our technique of cooling.

As another approach to the problem, a study of hibernation was initiated. The groundhog will not only hibernate at body temperatures as low as 3°C., but he can be artificially cooled to this level using an anesthetic and a cooling blanket as previously described. At this temperature the circulation was interrupted and the heart opened for one to two-hour periods on six groundhogs with five survivals. In an attempt to discover the secret of this unusual tolerance to low body temperatures, biochemical studies were made of the blood, both at normal body temperatures and during stages of hibernation. These blood samples were taken by special techniques in which the animal was not disturbed. No significant biochemical changes of the order which might explain the above phenomena were found in the groundhog's blood. Thus, his unusual cold tolerance has been attributed to an hibernating gland.

Studies of this gland would indicate that it is an endocrine gland of considerable size, rather than a storage gland, and histologically somewhat resembling the adrenal cortex. There were two large discrete segments of gland in each axilla, but it is also diffusely placed throughout the chest and mediastinum which renders it technically impossible to de-gland the animal. By cooling groundhogs at various seasons of the year and correlating the results with the size of the hibernating gland, a relationship between the activity of the gland and the animals' ability to tolerate very low temperatures, has been found. We have been in the process of extracting this gland for the past two years in the hope of obtaining an active principle which will improve cold tolerance when injected into non-hibernating animals and into human beings.

Present Research in Hypothermia

The present-day experimental approach to the problem of direct vision surgery of the heart is being studied with the use of: (1) extracorporal circulation, (2) hypothermia, (3) a combination of extracorporal circulation and hypothermia. Those studying hypothermia are lowering the body temperature of animals and humans by (a) surface cooling, (b) extracorporal direct cooling of blood, with, or without, the use of a pump. The goal of this research in hypothermia, is to bestow upon ordinary laboratory animals the unusual cold tolerance of hibernating animals that enables them to be cooled safely to very low temperatures. These low temperatures would then allow a longer period of interruption of the circulation and permit the correction of some of the more complicated lesions. At the present time there are only a limited number of conditions which may be corrected by direct vision surgery in five or ten minutes and such a period usually allows very little leeway. In order to obtain longer and safer operating time one has to eliminate the danger of cardiac arrest which develops in adult animals below 20°C., and in young animals at a lower temperature.

One can vary the biochemical state of the animal by various chemicals, cardiac drugs, gland extracts, et cetera. One may also vary the physiological state by technical procedures such as balloon obstruction of the aorta to improve coronary and cerebral flow, irrigation of the coronary arteries during the period of exclusion with a double lumen arterial catheter, the electrical pacemaker, exsanguination and retransfusion, and many other approaches. These procedures often improve the cooling technique and they have reduced but not eliminated the danger of cardiac arrest at these lower body temperatures.

It may be that one may not be able to control ventricular fibrillation and cardiac standstill without a clear understanding of such fundamental problems as oxygen transfer from blood to tissues and nerve conduction at low body temperatures. Infant animals and hibernating animals may have a similar type of tissue metabolism which may require study.

It may be possible to rapidly cool animals to a very low temperature and disregard ventricular fibrillation, with the establishment of a normal heart action on rewarming. This would not be possible in large animals or adult human beings without recourse to the extracorporal circulation.

Clinical Application of Hypothermia

Regarding the clinical application of hypothermia to cardiac surgery, there would appear to be two main conditions where cooling may be useful: (1) to allow intracardiac exposure with obstruction of the circulation and (2) as an adjunct to render current surgical procedures upon the heart more safe.

Doctors Lewis and Varco of this city (Minneapolis) were the first to report a case of intracardiac surgery with hypothermia and it was very pleasant to hear this confirmation of a theory. Several other centers have reported the clinical use of hypothermia for intracardiac surgery since that time. Swan has given further impetus to this work by his experience with experimental hypothermia and his excellent case reports.

Regarding the selection of cases for intracardiac surgery, judging from experimental evidence, one would be well advised to select only infants and children for cooling to the low body temperatures that are required. In order to justify the risk of severe cooling, one must cool to a temperature which allows an adequate period of interruption of the circulation without encroaching too closely on the lethal time limit. With careful cooling of body temperatures to 20°C., the circulation may be interrupted for approximately fifteen minutes. We have found that in infants and children 24°C. to 26°C. is safer, but of course that would allow a shorter period for exclusion of the circulation, perhaps five to ten minutes.

As one examines all of the cases of congenital heart disease in children, from the point of view of surgical correction, they appear to fall roughly into three main categories. (a) There is the very large group of children with congenital heart disease that usually die within the first year of life. In this group one finds tricuspid atresia, transposition of the great vessels, abnormal pulmonary venous drainage, and other similar conditions. These cases will require complicated procedures which cannot be done in a few minutes, but due to their age, they will very likely tolerate low body temperatures better than most cases and with a hopeless prognosis, one feels justified in exposing them to a procedure with a considerable risk. (b) There is the group of ventricular septal defects with, and without overriding the aorta, that will undoubtedly pose a difficult technical problem in many instances due to proximity of the conducting mechanism of the heart. (c) Inter-auricular septal defects and simple pulmonary stenosis, particularly the valvular type, are instances of congenital lesions which are relatively simple to correct and this may be done associated with a short period of interruption of the circulation. The results in cases with valvular pulmonary stenosis will have to be compared with those using the current operative procedure, before this approach can be fairly assessed.

Since age is an important factor in cold tolerance, I should think that the premature patients and infants to a few months of age with congenital heart disease, may be found to demonstrate remarkable tolerance to low body temperatures. If that is the case, one may hope to interrupt the circulation for longer periods of time.

We have commenced our clinical study with infants of the first group in co-operation with Drs. W. T. Mustard and J. D. Keith. The behavior of these infants at low body temperature has been gratifying, but serious obstacles have been met in the technical repair of these complicated cases.

The use of hypothermia as an adjunct to ordinary anesthesia is not new and has been practiced by Dr. Wills Potts for several years. In adults and children facing ordinary cardiac procedures with a high mortality rate due to heart failure or serious arterial oxygen desaturation, the body temperature may be lowered to about 30°C., at which level the oxygen requirements are reduced to about sixty per cent of normal before the hypothermia has seriously reduced cardiac function. This should reduce the risk of surgery. The above figures are based on our oxygen consumption studies and on the fact that in animal work, one finds the pressure fairly well maintained with a gradual fall to about 24°C., at which level it usually drops off rather more quickly. Certainly cooling, both adults and children to 30°C., with proper control of respiration should be a reasonably safe procedure.

In selecting cases for the uses of hypothermia as an adjunct, Mustard and Keith have chosen the very poor risk cases and those that are deeply cyanotic, but for whom a successful operation, by a standard technique, would be expected to yield a good result. Similarly, in adult cardiac surgery, those who are doing mitral valve surgery have found that the mortality rate in the group with right heart failure is high, particularly in the advanced cases with cyanosis and chronic failure. The first such case, in which a significant lowering of body temperature was obtained, was cooled in the sitting position. Her arterial oxygen saturation improved and she tolerated a difficult technical procedure remarkably well. Hypothermia down to 30°C., has proven a worthwhile adjunct in such cases.

One cannot give an opinion as to whether all cyanotic patients undergoing surgery should be cooled. Such a technique in practice may prove cumbersome and time consuming.

One may expect an increased incidence in wound infections with hypothermia. Another complication to be considered is the rewarming shock which has been observed experimentally and may be reduced by the use of our radio-frequency rewarming technique.

Regarding the actual technique of hypothermia, one may suggest rapid cooling in a cold water bath for infants and small children and the use of cooling blankets for larger children and adults. There should be continuous electrocardiogram recordings and rectal temperature.

The infants and children should be anesthetized and intubated before cooling as their cooling period may only be a matter of fifteen to twenty minutes. In the adults, we have cooled them with sedative alone to about 33°C., at which time they are given pentothal anesthesia with intubation and from that point down to 30°C., they are given positive pressure at twenty respirations a minute, in order to blow off excessive CO_2 and maintain a slightly elevated pH. If positive pressure is carried out for too long a period, it is suspected that this may increase the venous pressure and pre-dispose to ventricular fibrillation.

The operative technique has already been mentioned and it is recommended that rapid rewarming be carried out either by warm water at 40°C., or a radio-frequency device to a body temperature of 34° to 35°C. The final three or four degrees rewarming should be carried out in bed with warm water bottles.

Thus, at present, it is felt that hypothermia soon will be made safe for children. Adults cannot be cooled safely to body temperatures which are low enough to permit extensive intracardiac surgery. The answer to the safe cooling of adults may lie in such problems as the release of oxygen to tissues, or in understanding an anerobic type of tissue metabolism possessed at birth which gradually alters with maturity. It is suggested that cyanotic children are already conditioned to low oxygen tensions and very likely possess a slightly different tissue oxidative system. Such children may be expected to cool more safely than those that are not cyanosed.

Regarding the future of hypothermia in surgery, one is still optimistic. Extension of its use to surgery of the aorta is a natural step and many other possible uses such as liver surgery may be found. It requires study and care in its clinical application. Hypothermia as applied to cardiac surgery has, as its greatest asset, its simplicity.

APPLICATION OF A MECHANICAL HEART AND LUNG APPARATUS TO CARDIAC SURGERY (Discussion)

(Continued from Page 180)

References

1. Development of a pump-oxygenator to replace the heart and lungs; an apparatus applicable to human patients, and application to one case. Dennis, Clarence; Spreng, Dwight S., Jr.; Nelson, George E.; Karlson, Karl E.; Nelson, Russell M.; Thomas, John V.; Eder, Walter P., and Varco, Richard L. Ann Surgery, 134:709 (Oct.) 1951.
2. Acute metabolic changes associated with employment of a pump-oxygenator to supplant the heart and lungs. Dennis, Clarence; Spreng, Dwight S. Jr.; Young, LaVonne; Nelson, George E.; Karlson, Karl E., and Pereyma, Constantine. Pages 165-171 in Surgical Forum: Clinical Congress of American College of Surgeons. Philadelphia: W. B. Saunders Company, 1952.
3. Nelson, Russell M.: Metabolic effects of paracolon bacteriemia. Ann. Surgery, 134:885 (Nov.) 1951.

CARDIAC SEPTAL DEFECTS

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Denver, Colorado

EFFECTS of the two cardiac septa are apparently common, and together comprise a group of congenital heart lesions which is possibly the largest single such group. In 20 per cent of patients with congenital heart disease, one or the other septa will contain a patent defect. The significance to the patient of the presence of a septal opening depends almost entirely upon the volume of blood flow which is shunted through the opening. If this volume is large, an important physiologic defect is created which places its strain upon the heart and affects the size and function of the entire circulatory system.

Ventricular septal defects commonly occur as an isolated lesion as well as part of the complex of tetralogy of Fallot. For some reason which I do not clearly understand, it has become the commonly accepted conception, throughout the medical profession, to associate isolated ventricular defects with the name of Roger, and to think of this lesion as a benign defect which does not need treatment. It is true that there do exist defects of the ventricular septum, usually in the mid-portion of the septum, that though relatively small, do give a systolic murmur, which we should properly classify in our thinking as the maladie de Roger and which, in fact, do not cause significant physiologic disturbance since the shunt of blood is very small. On the other hand, it is our feeling that this is the uncommon type of septal defect. By far the more common and important is the high defect which exists immediately beneath the openings of the aortic and pulmonary valves. In this circumstance, the shunt of blood from left to right is great. There is enlargement and hypertrophy of both ventricles, of the pulmonary artery, and of the pulmonary-vascular bed. A pulmonary hypertension gradually asserts itself in order that systemic flow may be maintained. The strain on the heart now becomes increasingly great and death comes to the patients in the late teens, twenties or the early thirties. The situation is, in our opinion, exactly the same as that described by Eisenmenger and passes under his name in many instances. We do not think that the aorta, in fact, does anatomically override, in most instances, or if it does, is it not displaced by the enlarging pulmonary artery? We wonder whether there is any special entity as Eisenmenger's complex, but rather is it not simply a large ventricular septal defect, with the resultant changes in anatomy and physiology which have been brought about by virtue of this lesion. In any event, a large, high ventricular septal defect creates a functional single ventricle, and the outlook for the patient is no different than it would be were the septum essentially absent.

As for the auricular septal defect, this may occur in two locations, depending on the nature of the embryologic arrest. The auricular septum is formed in two phases with the growth first of what is called the septum primum, followed later by the growth of the septum secundum. Depending upon which process is arrested, the anatomical location on the septum of the defect is determined. Those which occur low, over the openings of the valves, present greater technical difficulty, but both high and low defects present the problem of massive left-to-right shunts of blood with the result of a gradually enlarging pulmonary circulation, that is to say, large right auricle, large right ventricle, large pulmonary artery and pulmonary-vascular bed. At the same time, a corresponding decrease in the systemic circulation occurs so that the aorta is small and peripheral circulation is low. It is thought that this may well explain a rather typical gracile and fragile habitus of patients with this lesion since their systemic circulation is so decreased. The shunt through the auricle into the pulmonary circulation is a saprophytic drain upon the circulatory needs of the remainder of the body. Under these circumstances, the heart gradually enlarges until heart failure ensues. Although we do not believe our computations to be exactly accurate, we have seen pulmonary circulatory flows with indices above thirty, while in the same patients the systemic indices were in the neighborhood of two to three. We think that as much as 75 to 90 per cent of the blood entering the left

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auricle may, in fact, escape through the septum into the right auricle. Thus, with auricular septal defects, as with ventricular septal defects, if the lesion is small, the patient may well survive to live a normal life and a normal life span. On the other hand, a very good percentage of these patients come to gradual diminution of cardiac reserve and death in their twenties and early thirties. Because septal defects are common, and because they frequently result in disability and premature death, these lesions are an important challenge to the surgeon to develop safe and adequate techniques for their closure.

As regards ventricular septal defects, the experimental work to date suggests that there is a very critical size which will result in serious physiologic disturbance of blood flow. Holman and Beck[7] were the first, I believe, to study this problem in the laboratory. They noticed that with the creation of defects sufficient to cause a significant shunt of blood, there were marked changes in the size of the heart. They noted the pulmonary artery increased in size until it was quite disproportionate to the aorta, and this was associated with right ventricular hypertrophy. These observations led them to the elaboration of a general thesis, which apparently has stood the test of time, to the effect that the heart and blood vessels place themselves in harmony with the increased volume flow of blood through them. The former, that is the heart, by dilatation and hypertrophy; the latter, the great vessels, by dilatation alone. Eppinger and Gross[4] subsequently studied holes in the septum which measured from 4 to 6 mm. in diameter and by computation determined that from 30 to 50 per cent of the output from the left ventricle went through defects of this size. Subsequently Griffin and Essex[5] determined that a hole in the dog's septum 5 mm. in diameter would close spontaneously, an 8 mm. hole remained and was tolerated indefinitely, but if the lumen were 10 mm., the dog would not survive without digitalization, while one 12 mm. was uniformly fatal within the first eight days no matter what the treatment. This experimental data gives strong support to the clinical impression that the size of the defect and the degree of shunt, is of supreme importance in determining the eventual outcome of the patient.

Experimental methods developed for the closure of interventricular septal defects have so far consisted of attempts to place blindly either autogenous tissues or foreign prosthesis in such a fashion as to occlude the septal opening. Murray[9] described such a method, using strips of fascia lata. Subsequently, Bailey and his co-workers[2] attempted to obstruct the opening by passing pedicled grafts of pericardium, either in the form of tubes or sheets through the septal defect. In their experimental work, both types of pericardial grafts could be placed technically and were tolerated by the heart of the animals who survived operation. In both instances, however, after the passage of a few weeks the grafts melted away and only a strand-like remnant of fibrous tissue remained. Bailey attempted this closure in a few patients with similar results.

Recently, Schumacker[11] has reported a modified method of using pericardial grafts. With this technique, the graft is placed in the form of a tube in a front-to-back fashion within the substance of the septum making the tube traverse the septal defect by means of digital control with a finger through the right auricle into the right ventricle during the placement of the tube. Autogenous blood is now injected into the tube, blowing up that portion which is free within the septal defect into a balloon-like expansion which occludes the opening. In his experimental results, complete closure of the experimental lesion by fibrous tissue resulted from the organization of the clotted blood within the pericardial graft. Schumacker[10] has subsequently modified the technique so that instead of using pericardium, he now uses a tissue made of plastic. He has not yet reported upon the results of his initial clinical trials with this method.

It seems reasonable to believe that the technical problems posed by the interventricular septal defect are somewhat greater than those posed by defects in the auricular septum. The presence of the adjacent conducting tissue of the septum, and the confusion of the immediate environment which includes portions of all four major valves of the heart, have made technical solution to the problems of this particular lesion lag behind that of others. At the present time, I believe it is fair to state that no standardized closure technique of ventricular septal defects is yet available.

Defects of the auricular septum on the other hand, although posing interesting problems, have become amenable to surgical repair at reasonable risk rates. As was proper, the solution to the problem was first obtained in the laboratory. The

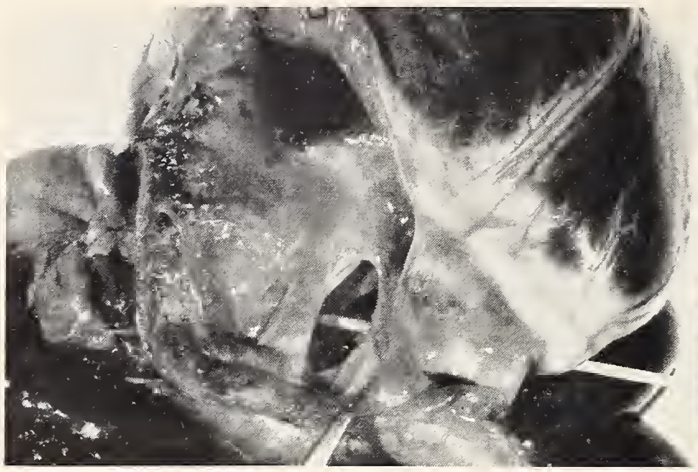

Fig. 1.

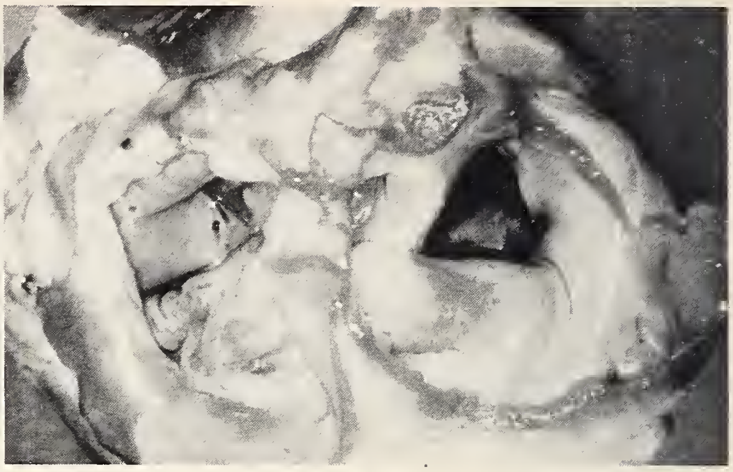

Fig. 2.

first report, in this regard, of real importance was that presented by Dr. Cohn[3] in 1947, wherein he described an extremely ingenious method for transplanting a segment of auricular wall, to the auricular septum, with the thought in mind that such a transplantation might be effective in closing auricular septal defects. Our study[13] began about this time with attempts to create large defects in the auricular septum. This was finally accomplished, after unsuccessful attempts with various closed methods, by the use of the temporary interruption of circulation, opening the right auricle and excising under direct vision the atrial septum. We placed an arbitrary time limit of ninety seconds as the upper safe limit for occlusion but by intermittently resuming and stopping circulation we were able to obtain four and one half minutes of operative time within the right auricle, which proved to be more than adequate to perform this job. Defects created experimentally in this fashion will remain patent and, in Figure 1 we see such a defect two weeks following its creation.

The first technique for closure which we tried involved what we have subsequently termed the principle of the "doughnut" auricle. This concept implied that it would be possible to invert a fair amount of tissue to the center of the septum, thus obliterating the central portion of the auricular lumen, yet at the same time allowing blood to flow around such tissue in such a fashion that adequate blood flow would be maintained. In the first experiments, the tips of the two auricles were inverted and sewn together through the septal defect (Fig. 2). This resulted in an 80 to 100 per cent closure of the size defects that we were dealing with in the experimental laboratory. In 1949 and 1950, this method was given clinical trial in a few patients. Although the patients tolerated the surgery, it was soon clear that we were not obtaining adequate closure of the large clinical defects found in these patients—defects many times larger than the experimental ones. Accordingly, we returned to the laboratory and devised a method for placing a plastic button across the septal defect.[14] The response of the heart to a polyethylene button placed in this fashion was quite interesting. The button becomes covered with fibrin in a few hours. Figure 3 shows a polyethylene button which has been in place within the auricle for twenty-four hours. One half of the button has been exposed in order to show the fibrin layer covering it. In the course of about three weeks this fibrin layer will be replaced by ingrowth of fibrous tissue from the periphery which becomes a smooth, glistening membrane (Fig. 4). The end situation of this technique then, is a plastic button within the lumen of the right auricle, occluding the interauricular septal defect, and held in place by the inverted tip of the left auricle to which it is sewn (Fig. 5). The left auricle is inverted only to maintain fixation, as we have always been concerned lest a foreign body placed within the heart might work its way loose and become migratory. This technique was also tried in a few patients, but here fixation of plastic buttons, adequate in size to occlude the very large septal defects which we encountered, proved to be difficult. At about this time, Bailey and his group[1] published the technique which they had developed, modifying the "doughnut" auricle concept, and utilizing auricle wall instead of appendage. Utilizing finger guidance, the wall of the auricle was sewn, with an interrupted suture, to the rim of the septal defect thus obliterating the lumen through the septum. Also, at about this

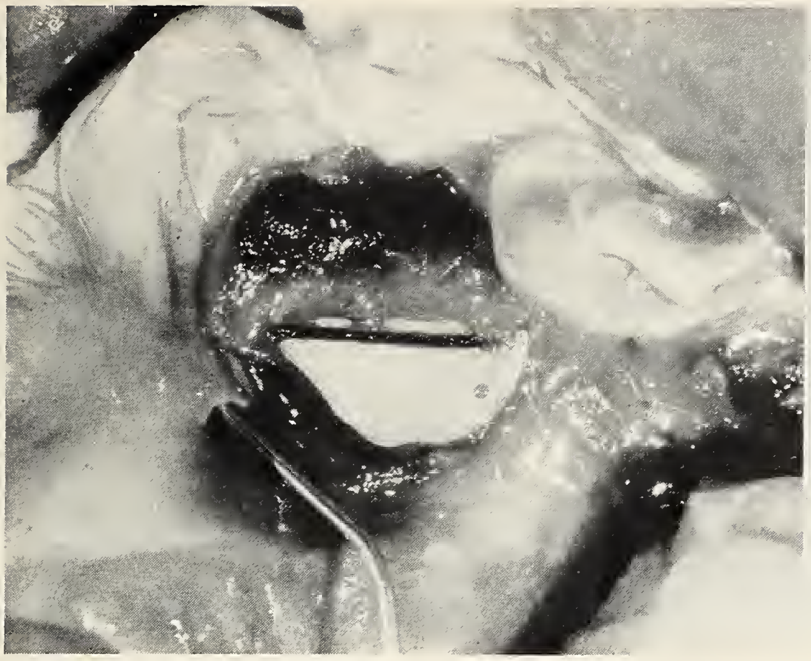

Fig. 3.

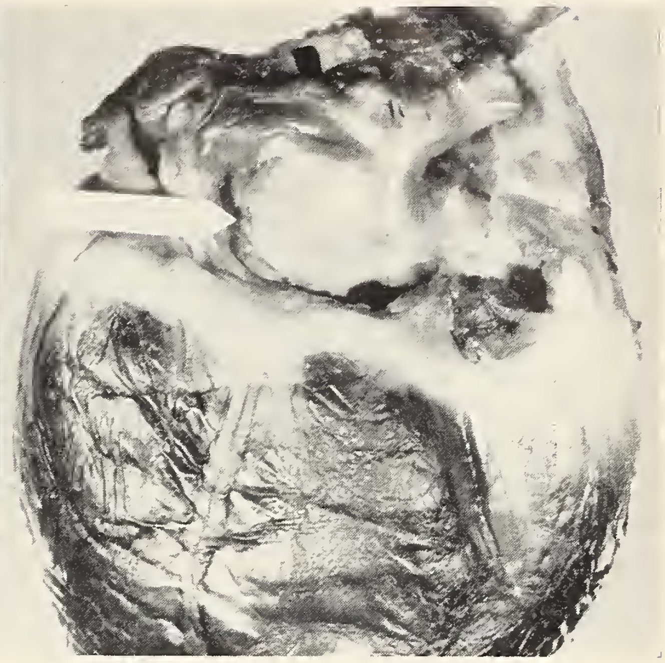

Fig. 4.

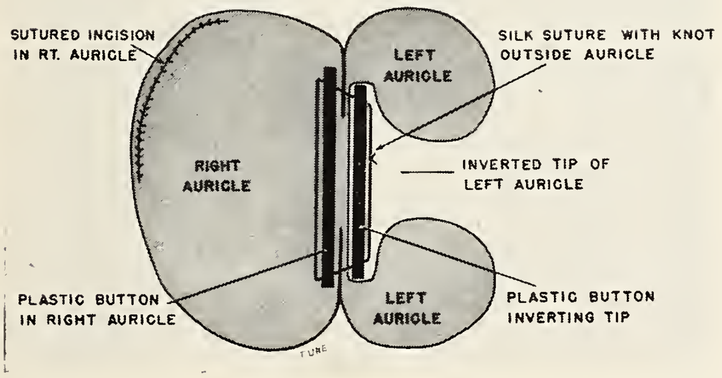
SUTURED INCISION IN RT. AURICLE
LEFT AURICLE
SILK SUTURE WITH KNOT OUTSIDE AURICLE
RIGHT AURICLE
INVERTED TIP OF LEFT AURICLE
PLASTIC BUTTON IN RIGHT AURICLE
LEFT AURICLE
PLASTIC BUTTON INVERTING TIP

Fig. 5.

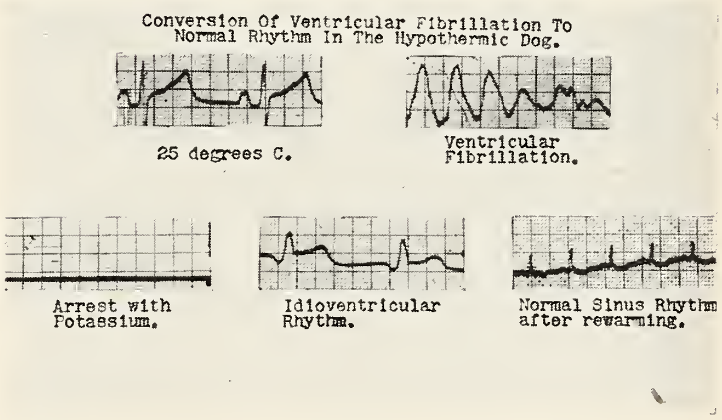

Fig. 6.

time, Gross[6] published his early experience with the use of the well, which allowed him to attempt to sew together at leisure, the rim of the septal defect, feeling with his finger and his instruments through the pool of blood in his well.

At this stage, then, there appeared to be two or three methods for the repair of interatrial septal defects, all of which suffered from the fact that they were performed blindly. At this time, also, in our laboratory, as in many other laboratories throughout the country, interest became focused upon the potentialities of hypothermia in the management of cardiac procedures. It appeared to us that of all intracardiac lesions, the interatrial septal defect was certainly one which should be most amenable to open visual repair. While we were studying methods in the laboratory to make it safer for human clinical application, the report of Drs. Lewis and Taufic,[8] from this great medical center in Minneapolis, of the first attempt which proved to be successful of the open closure of interauricular septal defect, was made. Since that time, we have adopted this method, and our last five patients have been operated with visual closure of their defect.

The application of hypothermia to cardiac surgery has already been throughly discussed. I wish merely to state that we cool our patients in a tub of ice water after they are under the effect of anesthesia. Constant temperature recording together with constant electrocardiograph monitoring is maintained. Experimentally, in the laboratory we found that hyperventilation was of great importance in reducing the incidence of ventricular fibrillation.[15] We found, moreover, that the use of potassium chloride was very helpful in resuscitative measures designed to defibrillate the ventricles in the event it should occur. Figure 6 shows segments of an electrocardiogram taken from an experimental animal undergoing hypothermia, who was made to fibrillate by the placement of a ventricular incision. Potassium chloride injected into the base of the aorta and massaged into the coronary circulation results in standstill which is then followed after cardiac massage by the resumption of rhythmic ventricular contractions. We felt, on the basis of our experimental observations, that it would be safe to attempt to explore the application of hypothermia to clinical surgery.

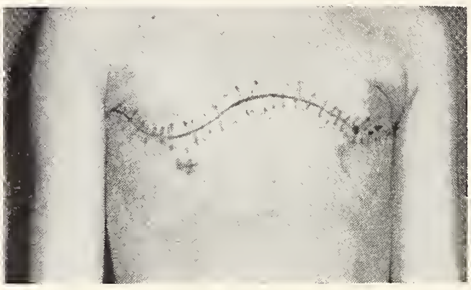

Fig. 7.

Interatrial septal defect was one of three lesions to which the method was applied. With your permission, I would like to describe some of the technical aspects of this procedure which we have found to be of value and importance. In the first patient with closure of atrial septal defect, we placed only a left anterior incision (Fig. 7). Exposure was not as adequate as we would have wished, and from this time on we have routinely placed the anterior bilateral sternum-splitting incision which we had previously used for pericardectomy and the removal of foreign bodies from the heart.[12] This incision gives maximum exposure of the heart and all its chambers, and allows one to manage any cardiac complication which might arise. Although long, this incision is a physiologic one, and, since no portion of the thoracic cage is removed, the patient is comfortable in the post-operative period and breathes well so that pulmonary complications are not common.

After the incision is placed, the first step is the ligation of the azygos vein, and the placement of tapes about the superior and inferior vena cava. The pericardium is now opened widely, and an incision into the auricle, well posterior, about an inch and one half in length, is made over a non-crushing clamp. Thus the incision into the auricle is made before circulatory arrest in order not to waste any of the valuable period of cessation of circulation.

Traction on the tapes around the vena cava is now made, shutting off the entrance of blood to the heart. After a few beats, allowing the heart to empty itself as much as possible, a clamp is placed across the base of the aorta and pulmonary artery. In addition, tourniquets previously placed about the root of the lungs are tightened in order to occlude the pulmonary veins. Thus, all vessels, entering or leaving the chambers of the heart, are occluded. The clamp on the auricular incision is now released, the residual blood in the heart is removed by suction and inspection of the chambers can be made. It is important not to forget to feel the mitral valve, through the septal defect, in order to be sure that this structure has not become involved as in the so-called Lutenbacher syndrome. The atrial septal defect may now be closed by sutures, using interrupted mattress sutures of silk. No matter how large the septal defect may be, in our limited experience, the edges will come together without tension when the heart is collapsed. We believe that when blood is allowed to flow again, the tension will not be increased sufficiently to cause tearout of the sutures. All of the sutures in the septum are placed and tied, with the exception of the last one. The line of closure is now held open by a forceps while the heart is flooded with saline solution forcing all air out of the left side. The final suture is now tied and cut under the surface of the water. More solution is poured in to fill the right side of the heart, and the non-crushing clamp is applied to the auricular incision. The various obstructing clamps to the lung roots, to the aorta and pulmonary artery are now removed, and the tape around the superior vena cava is released. Circulation at this moment is restored, at least partially. One observes the action of the heart and, as time goes by, the beat picks up and the myocardium becomes pinker, the tape around the inferior vena cava is finally released. Circulation is now totally restored. The incision into the auricle may be sutured, with interrupted sutures of silk, at leisure. Finally chest drains are placed in both pleural spaces and the incision is closed.

To date, we have operated on five patients using the open visual technique for closure of interauricular septum. In four of these patients, the operation was successful; the patients have done very well. In our opinion, these patients have complete closure of the septum. The fifth patient failed to survive the operation. He was a twelve-year-old boy who had a history of chronic heart failure, and was on digitalis for several months before operation. Although technically it was possible to suture the septal defect, his heart became distended and he died, in what we took to be heart failure, about two hours later in spite of resuscitative measures. The duration of total occlusion of circulation in these five patients varied from five minutes and fifty seconds to seven minutes and forty-five seconds. No suggestion of cerebral damage has occurred.

At the present time we feel that the safety of the open technique is sufficient to warrant further trial, inasmuch as the technical aspects of the closure of the septal defect is so much superior to any of the blind techniques, no matter how ingenious they may be.

References

1. Bailey, C. P.; Downing, D. F.; Geckler, G. D.; Tikoff, W.; Goldberg, H.; Scott, J. C.; Janton, O., and Redondo-Ramirez, H. P.: Congenital communications: clinical and surgical considerations with a description of a new surgical technic: Atrio-septopexy. Ann. Int. Med., 37:888, 1952.
2. Bailey, C. P.; Lacy, M. H.; Neptune, W. B.; Wheeler, R.; Arvanitis, C. S., and Karasic, J.: Experimental and clinical attempts at correction of interventricular septal defects. Ann. Surg., 136:919, 1952.
3. Cohn, R.: An experimental method for the closure of interauricular septal defects in dogs. Am. Heart J., 33:453, 1947.
4. Eppinger, E. C., and Gross, R. E.: Measurements of the circulation in dogs with an interventricular septal defect. J. Clin. Investigation, 20:461, 1941.
5. Griffin, D. C. J.; and Essex, H. E.: Experimental production of interventricular septal defects: certain physiologic and pathologic effects. Surg., Gynec., and Obst., 92:325, 1951.
6. Gross, R. E.: Surgical closure of interauricular septal defects. J.A.M.A., 151:795, 1953.
7. Holman, E., and Beck, C. S.: The physiological response of the circulatory system to experimental alterations. I. The effect of intra-cardiac fistulae. J. Exper. Med., 42:661, 1925.
8. Lewis, F. J., and Taufic, M.: Closure of atrial septal defects with the aid of hypothermia: experimental accomplishments and the report of one successful case. Surgery, 33:52, 1953.
9. Murray, G.: Closure of defects in cardiac septa. Ann. Surg., 128:843, 1948.
10. Schumacker, H. B.: Personal communication.
11. Schumacker, H. B., and Moore, T. C.: The experimental closure of atrial septal defects. Presented before the 33rd Annual Meeting of the American Association of Thoracic Surgery, San Francisco, March, 1953.
12. Swan, H.; Forsee, J. H., and Goyette, E. M.: Foreign bodies in the heart. Ann. Surg., 135:314 (March) 1952.
13. Swan, H.; Maresh, G.; Johnson, M. E., and Warner, G.: The experimental creation and closure of auricular septal defects. J. Thoracic Surg., 20:542, 1950.
14. Swan, H., and Stewart, B. D.: A modified button technique for the closure of experimental interauricular septal defects. J. Thoracic Surg., 25:397, 1953.
15. Swan, H.; Zeavin, I.; Holmes, J. H., and Montgomery, V.: Cessation of circulation in general hypothermia. I. Physiologic changes and their control. Ann. Surg., 138:360, 1953.

Discussion

Dr. Ivan Baronofsky, Minneapolis, Minn.: It is indeed a pleasure to be asked to discuss "The Surgical Treatment of Interatrial Septal Defects." With all that you have seen and heard this afternoon with regard to the work of Dr. Swan, Dr. Bigelow, Dr. Crafoord, and Dr. Lewis, I feel very much like a discusser of a discusser. My experience in closure of interatrial septal defects is rather limited; however, my interest is great. In 1951 an attempt was made to close an interatrial septal defect in a youngster five years of age. This was done by an attempted inversion of the right atrial wall and then suturing the atrial wall to the rim of the interatrial septal defect. Catheterization studies of this youngster revealed partial closure of the septal defect.

I should rather like to present to you some interesting studies that were carried out by Dr. Carleton B. Chapman and Dr. Robert Fraser at the University of Minnesota Variety Club Heart Hospital.† Drs. Chapman and Fraser studied the hemodynamics of interatrial septal defects in nineteen patients. Clinical features of note are as follows: ages were from seventeen to fifty-one; ten were male and nine female; heart disease was detected early in life in three, in mid or late childhood (7 to 14 years) in seven; at the age of eighteen or later in eight; the chief complaints were shortness of breath in six, pain around the heart in five, palpitation in three, fainting episodes in one, and no complaint in two. Systolic murmurs were present in seventeen cases. Cardiac catheterization was accomplished in all of these cases. The oxygen content of blood samples taken from various sites and the femoral arterial saturation was determined (Table I).

TABLE I.

	SVC	RA	RV	PA	FA	Fem. Art. % Sat.
Mean	12.8	16.9	16.8	16.9	18.8	96.0
S.D.	2.4	2.6	3.1	2.9	2.7	7.6

The existence of functioning communication between the atria was thought probable if the oxygen content of the right atrial blood exceeded that of blood from the innominate vein or superior vena cava by 2.0 volumes per cent. Drs. Chapman and Fraser determined the systemic and pulmonary flows, mean pressure in the pulmonary artery and right ventricle, and right ventricular

(Continued on Page 194)

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SURGICAL TREATMENT OF PULMONARY STENOSIS

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IT is only eight years since Blalock opened the door to surgical alleviation of pulmonary stenosis. Intensive clinical study by the cardiologist has accentuated skill in diagnosis. Roentgenologists have learned the refinements of interpretation of cardiac silhouettes and lung densities. Through angiocardiography, knowledge of the chambers of the heart and its vessels is obtained. Cardiac catheterizations inform us concerning blood flow and interpret the significance of venous and arterial blood mixtures. The anesthesiologist has acquired skill in carrying these cyanotic patients through a long and arduous operation. Pathologists study our errors and stake out hazards along the course. Physiologists and pharmacologists continue to add to our basic knowledge of the heart, its action, its reserve, and its response to drugs.

Innumerable workers in many fields have done much for these children—the victims of cyanotic heart disease.

Our attitude at the moment towards the surgical treatment of the tetralogy of Fallot at The Children's Memorial Hospital, Chicago, is as follows:

If it has been demonstrated clinically that the child has infundibular stenosis with an interventricular septal defect, we approach the condition surgically by a posterolateral incision on the left side through the fourth interspace, provided the child is above approximately one year of age. If the arch is on the left, an aortic pulmonary anastomosis is done, making the opening 4 mms. in diameter, slightly less in very young children. If the arch curves to the right, a subclavian pulmonary anastomosis is done on the left through a similar incision. If the patient is an infant below one year of age and cannot live without surgical relief, an aortic pulmonary anastomosis is done on the right side.

In spite of all possible preoperative studies, it is at times impossible to be sure whether we are dealing with a tetralogy or a pure pulmonary stenosis. If the evidence leans towards tetralogy, a posterolateral incision is made and the pericardium is opened. If by palpation and observation, or pressure studies, the diagnosis is confirmed, a shunt operation is done. However, if the condition proves to be a pure pulmonary stenosis, a valvulotomy is done either in a retrograde fashion by way of the left pulmonary artery or the incision is extended and a transventricular valvolotomy is done.

Whenever a pure pulmonary stenosis is even suspected, a left submammary approach is chosen and, if the diagnosis is confirmed, a transventricular valvolotomy is done. If the patient is found to have an infundibular stenosis, the incision is extended laterally and a shunt operation is done.

What about the transventricular approach to infundibular stenosis as suggested by Brock? We have made a detailed post mortem study of forty-two hearts—some fresh and some in formalin-fixed condition. The results of this study will be published soon in the *American Heart Journal*. The photographs cannot be reproduced here, but it was concluded that about 19 per cent of the hearts had a type of infundibular stenosis which could well have been treated by intracardiac surgery. The fact that we found a smaller percentage amenable to simple intracardiac resection is probably due to the fact that the average age group in our series was far lower than that of other investigators. Eighty-two per cent of this series was comprised of hearts taken from children below three years of age, and obviously, represented the more severe types of tetralogy of Fallot.

Pulmonary stenosis with intact interventricular septum, more commonly labeled pure pulmonary stenosis, is most satisfactorily treated by transventricular valvolotomy as suggested by Sellors and Brock. The results of surgical treatment of this condition are truly spectacular.

A recent study of forty patients by Dr. Gibson at our hospital brings to light a number of interesting facts among which is the age factor. For some unknown reason, our patients have been much younger than those studied by other cardiologists. Nine were under the age of one year at the time of operation. Seven were between one and two years of age, eleven were between two

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TABLE I. MORTALITY FIGURES

A-P or S-P Anastomosis	Tetralogy of Fallot		Percentage
	No.	Deaths	
2 weeks to 3 years	145	23	15.8
3 to 16 years	186	6	3.2
Total	331	29	8.7
	Isolated Pulmonary Stenosis		
23 days to 3 years	19	1	5.2
3 to 15 years	21	0	0
Total	40	1	2.5
	Tricuspid Atresia		
11 days to 3 years	15	8	53
3 to 16 years	5	0	0
	20	8	40

and five years, and the remaining thirteen were between five and fifteen years.

The surgical approach to these patients is as follows:

The chest is entered through the third interspace by a left submammary incision. The pericardium is opened. The use of novocaine in the pericardial sac and in the heart muscle has been discontinued. After placing a couple holding sutures in the wall of the right ventricle, the closed valvolotome is introduced into the right ventricular chamber, opened to appropriate size, thrust through the stenotic valve, closed and withdrawn from the heart. The expanding dilator is then introduced through the same opening and the stenotic pulmonary valve is dilated as widely as possible. The palpating finger on the pulmonary artery will be able to determine by the change in pressure and thrill that the pulmonary constriction has been properly opened.

If the patient, particularly a small child, is especially cyanotic and if its heart is greatly enlarged, holding ligatures are placed as above and the small knife valvolotome is quickly thrust through the constriction. The moment the stenosis is partially relieved the child's color changes and the heart action improves. It is then safer to proceed with the expanding valvolotome and dilator.

Mortality figures appear in Table I. It is at once obvious that when dealing with the tetralogy of Fallot, if the child's condition is such that it can survive to age three years, it has over 95 per cent chance of surviving surgery. Mortality must necessarily continue high in infants whose condition is so bad that immediate operation offers the only hope of life. Infants with tricuspid atresia do poorly after surgery. Results of a shunt operation in general are very satisfactory to the parents and the child but, of course, leave much to be desired from a medical standpoint.

The surgical mortality of pure pulmonary stenosis—one death in forty cases—is 2.5 per cent. The immediate and follow-up results of this operation are satisfactory to all.

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Discussion

JOHN W. KIRKLIN, M.D. (Rochester, Minn.): It is an honor for me to open the discussion of the paper by Dr. Potts, who is a pioneer investigator and a brilliant surgeon in the field of congenital heart disease.

Tetralogy of Fallot.—The establishment of a shunt between the aorta, or one of its branches, and the pulmonary artery has been the treatment of choice for tetralogy of Fallot at the Mayo Clinic since its introduction by Blalock and its modification by Potts. In occasional cases of the tetralogy, however, for one or another reason, we have chosen instead to make a direct attack on the obstruction to pulmonary blood flow. This should not obscure the fact that at present we continue to employ the shunt operation in most of our cases. This situation may change when completely reliable means of working in the heart, in a bloodless field, are available to us.

It behooves us, then, to review the goals of the surgical treatment of patients with the tetralogy. The *ideal* goal would be a revamping of the intracardiac situation so that neither a right-to-left shunt nor obstruction to the flow of blood from the right ventricle to the pulmonary trunk would exist any longer. Until that anatomically ideal situation can be produced with a low operative mortality, we must be content with other goals. Symptomatic improvements, although real, are difficult to quantitate. We seek also certain physiologic changes as the means of producing the desired symptomatic improvement. These changes can be measured accurately. We seek to elevate the oxygen saturation of arterial blood during rest. We seek to prevent the profound fall in arterial oxygen saturation that characteristically occurs in these patients on exercise. Finally, we strive for reduction in the polycythemia as a response to these improvements.

In a high percentage of cases, the shunt operation produces these desired changes. For example in one case the oxygen saturation of arterial blood as determined by the ear oximeter was 76 per cent before operation and 91 per cent seven days after operation when the patient was at rest; it was 47 per cent during hard work (crying) before operation and 86 per cent seven days after operation. In the same case with the patient breathing 100 per cent oxygen the oxygen saturation of arterial blood was 78 per cent before operation and 86 per cent seven days after operation. In our opinion, any other operation besides the shunt operation which does not produce an anatomically normal heart must result in measurable physiologic changes comparable to those produced by a well-executed shunt in order to be considered equal or superior to aorticopulmonic anastomosis. Furthermore, the operation must be accomplished with the low mortality that now prevails for the shunt procedure.

Pulmonic Stenosis With Intact Ventricular Septum.—For cyanotic patients with pulmonic stenosis and intact ventricular septum, we seek an increase in arterial oxygen saturation and a decrease in right ventricular pressure as the result of operations. In cases of pulmonic stenosis with intact ventricular and atrial septa, we seek reduction in right ventricular pressure. Unfortunately, there is a paucity of information relating to the accomplishment of this reduction in pressure by the usual operation of pulmonic valvotomy as described by Dr. Potts. The information available suggests that although many patients improve considerably after the operation, some obtain only mild reduction of right ventricular pressure. This has stimulated some workers to a more radical attack on the pulmonic valve. Our experience with precise recordings of pressure changes at operation has suggested that such an attack will probably not succeed in bringing the right ventricular pressure to near normal in all cases. Our data indicate that in a significant percentage of cases of pulmonic stenosis with intact ventricular septum there is obstruction not only at the stenotic pulmonic valve but also in a narrowing in the outflow tract of the right ventricle.

Thus we have little enthusiasm for a more radical attack on the valve in such cases. Further improvement in the treatment of certain of these patients may be made by an additional attack, under direct vision, on the narrowed outflow tract of the right ventricle.

In closing these brief remarks, I should like to pay tribute to the fine address given by Dr. Potts and to thank this group for the privilege of opening the discussion on his paper.

CARDIAC SEPTAL DEFECTS (Discussion)

(Continued from Page 191)

TABLE II.

Flow L./min.		L-R Shunt L./min.	Mean Press. Mm. Hg		Pulm. Resist. Units	RV "Work" Kg. M. per Hr.	WRS Axis Degrees Plus
Syst.	Pulm.		PA	RV			
5.4	16.4	11.0	21.0	15.6	1.6	361.4	102.1
1.4	5.8	5.9	9.0	5.7	1.1	215.0	13.7

TABLE III.

Type of Pressure Used		Kg.-m./hr.		Gm.-cm./beat	
		Mean	S.D.	Mean	S.D.
Pulm. mean	Normal*	58	18	1387	517
	I A S D	269	152	5541	3307
RV mean	I A S D	205	120	4191	2524
RV Syst. mean	Normal**	105	—	2362	—
	I A S D	361	215	7550	4997

*From Prec, et al.
**From Handelsman, et al.
IASD Interatrial septal defect.

pressure work. The following is a summary of nineteen cases (Table II).

A comparison was also made of the pressure work of the right ventricle in normal subjects and in patients with interatrial septal defects (Table III).

From the preceding information it would appear that patients with interatrial septal defects, with complaints that are referable to their heart, are indeed in incipient or actual trouble. It would appear that there is no question that surgical correction of this anomaly would be in order. The things that appear to hinder us in the surgical correction of interatrial septal defects are the following: (1) diagnosis of the presence of an atrioventricularis communis. (2) the ability to rule out anomalous pulmonary venous drainage into the right atrium, and (3) the ability to deal with bundle branch blocks which are common in interatrial septal defects.

I should like, lastly, to mention some personal communication that I have received from surgeons interested in this field. Dr. Robert Glover, of Philadelphia, writes that in five cases that he personally had operated on, the big problem that seemed to confront him was the presence of an atrioventricularis communis. In another two, the defect seemed to be extremely large with either no rim of tissue between the mitral and tricuspid valves or with such a tiny half-moon that any repair would prejudice normal valvular function. A fifth case was closed by a method of invaginating the right atrial wall against the defect. Dr. Charles P. Bailey writes that in twenty-four cases in which repair of the septum secundum was attempted, by his technique of atrio-septo-pexy, twenty-two were greatly improved. Postoperative catheterization has been done in ten patients. In all instances, the shunt was greatly reduced and in eight it was completely abolished. There were an additional fourteen cases of persistent ostium primum. The operative mortality in these cases was 57 per cent. All the survivors, however, have shown very definite clinical improvement. Dr. Robert E. Gross writes that he is still continuing to use the "Well" technique that he has published previously. His enthusiasm for this form of surgery continues very high, provided the individual has not yet passed into a state where there is a secondary change and increasing obstruction to the pulmonary vascular bed—which seems to him bad for any type of therapy.

REGIONAL ENTERITIS

Its Surgical Management

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THE AUTHOR will attempt to define and evaluate five procedures used and recommended in the surgical management of regional ileitis, more properly regional enteritis, because any segment of bowel may be involved. Some go so far as to advocate that regional ileitis, certain granulomas and ulcerative colitis have the same or similar etiology. Neither etiology, symptoms, diagnosis nor medical management will be reviewed.

The five procedures listed below will be discussed: (1) conservation, (2) vagotomy, (3) ileocolostomy, (4) ileo-transverse-colostomy with exclusion, and (5) resection.

1. *Conservatism.*—An occasional laparotomy is performed under a mistaken diagnosis of acute appendicitis, or an acute ileitis is found unexpectedly. The bowel may be so acutely inflamed that radical surgery would create an unjustifiable hazard. It is generally agreed, under the circumstances, that the operation should be terminated at once, with or without removal of the appendix. Many believe that an appendectomy increases the risk or the likelihood of fistula formation, while others state that fistula results from the disease in the ileum and that the appendix should be removed. Repeated reports are available to establish the fact that some patients so treated improve on postoperative management and require no further surgery. Certainly, the finding of regional enteritis does not alone constitute adequate reason for radical surgery.

2. *Vagotomy.*—Vagotomy has been done for regional enteritis on the basis of favorable results following its use in ulcerative colitis. Thorek recently reported 80.9 per cent favorable results in twenty-one patients treated for idiopathic ulcerative colitis or regional enteritis by vagotomy. He preferred the transabdominal vagatomy because it gave opportunity to visualize the pathology. Graham states these results constitute additional evidence of the importance of psychosomatic factors in the etiology of the disease. One patient, twenty-four years of age, treated at our Veterans Hospital, in whom a laparotomy was terminated because of severe extensive inflammation, continued to have thirty to forty stools daily and his course continued unfavorable. This patient was treated by transthoracic vagotomy, but he obtained little improvement and one year later a laparotomy was repeated. The bowel showed marked improvement with two remaining skip areas and an abdominal vagotomy was performed. His course consisted of several remissions with numerous stools and malnutrition. Death ensued and autopsy examination revealed severe ileocolitis with ulceration and bleeding. A second patient extremely ill, treated by exclusion, ileo-transverse-colostomy and abdominal vagotomy, is doing fairly well with recurrence. In these cases, vagotomy has been of questionable value. It should probably be reserved for far advanced disease.

3. *Ileocolostomy.* — Ileocolostomy shunts the bowel stream by the diseased loops. It serves well as the first operation of a staged resection, necessitated either by the unfavorable condition of the patient or the far advanced state of the disease, or in the presence of fistula or abscess. As a definitive procedure, it has produced such poor results, that alone it should best be avoided.

4. *Ileo-Transverse-Colostomy with Exclusion.* —Ileo-transverse-colostomy, with exclusion, has produced very favorable results, especially in the presence of extensive bowel involvement, fistulae and/or abscesses. The small bowel is divided twenty-four inches proximal to the highest area of involvement, the proximal end is anastomosed to the transverse colon and the distal end turned in and dropped back. If partial obstruction is present in the lower segment and a blowout of the inverted end is likely, the distal end may be drawn out through a stab wound and a mucous fistula created. Ileo-transverse-colostomy with exclusion also serves well as a first stage before resection. As the disease subsides, the remaining

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abscesses, fistulas, and diseased bowel may be removed with greater safety. Garlock reports the Mount Sinai mortality to be low with the exclusion procedure, and the results excellent. They (Bancroft) reported sixty-five cases treated by this method, with no operative deaths and only 13.8 per cent recurrence rate. Frobese states their experience confirms that of Garlock, and further, that they have had occasion to reoperate several of these cases and were surprised to find that all of the gross evidence of disease in the side-tracked loop had disappeared. In 1945 Garlock et al reported fifty-seven cases treated by this method without mortality, with six recurrences, an incidence of 10.5 per cent. However, in 1951, a subsequent follow-up showed an additional five recurrences among forty-seven cases, ten having been lost from observation, a recurrence rate of 23.4 per cent. He is convinced that exclusion surgery is followed by fewer recurrences than is resection.

Bockus, in discussing this paper, challenges Garlock's method of arriving at his percentages and states, "If we take the 103 cases of Dr. Garlock which have been followed up, forty-one are found to be either dead or have recurrences. To me, that means a bad result in approximately 40 per cent of the cases. If we review the entire group of 137 cases which he reported, we find that seventy-five of them are either dead or have recurrences or have not been followed up, which means 55 per cent have had bad or unknown results." Bockus further states: "Our group in Philadelphia happens to be the group (mentioned by Garlock) which has an authentic recurrence rate following operation (usually resection) which is well over 50 per cent in this entire group of diseases which includes regional enteritis and enterocolitis."

The experience of surgeons generally, is that the exclusion operation is of great value.

5. *Resection.*—Resection may be performed as a single stage with primary anastomosis or as a staged procedure after: (1) ileocolostomy, or (2) ileocolostomy with exclusion. Because the disease may start primarily in the bowel, resection has the great advantage of removing the involved segment and probably reducing absorption of toxins. On the other hand, the regional nodes are usually so extensively involved that complete removal of the disease is impossible. Still, exponents of resection contend that remaining disease often subsides or remains asymptomatic. Garlock's original statistics showed the mortality of resection (13.3 per cent) to be much higher than that for exclusion (0 per cent). With the accumulation of greater knowledge of the disease, better preoperative preparation of the patient, and the liberal use of antibiotics, we have had no surgical mortality in our own limited experience. At the Minneapolis Veterans Hospital the same experience has been obtained in the past three or four years since medical care was taken over by the University of Minnesota. Opinion in our locality definitely favors one-stage resection with primary anastomosis. Cattell states that from their experience a recurrence rate of 30 per cent may be expected. They prefer resection, and, with extensive involvement, perform staged operations. They have performed staged resections of diseased bowel too extensive to be safely done at one operation. Staging permits the patient to adjust his body metabolism without malnutrition. Six of his patients so treated have sixty inches or less of jejunum anastomosed to varying lengths of colon. Cattell has found, as have others, that resection of bowel without excising all of the grossly apparent disease, may be followed by relief of symptoms. Preoperative preparation is very important as malnutrition and the ravages of the disease may be corrected. Adequate quantities of blood, vitamins and food, together with suitable antibiotics (there is none specific), have greatly lessend mortality and morbidity. Equally important factors are the advances made in anesthesia and postoperative care.

Mortality and recurrences: Garlock early reported a mortality of 13.3 per cent after resection but none after exclusion with ileocolostomy. His recurrence rate was better in the latter. Several years later he states that in the exclusion operation, with no mortality, their over-all recurrence rate was 22.8 per cent. In cases treated by resection, their mortality rate was 14 per cent, with an over-all recurrence rate of 46 per cent. Cattell favors resection procedures, which they have done 160 times with only three deaths. They expect a recurrence rate of about thirty per cent. In a recent report from the Lahey Clinic by Kiefer, the last review of their statistics reveals a recurrence rate of 34 per cent. Recurrence has demanded reoperation as many as four times. Of forty-two patients who had recurrence, thirteen recovered without a second operation. Twenty-eight others were submitted to a second operation and eleven patients to a third operation and two to a fourth operation. These were desperate cases which taxed the skill of all concerned and in whom there was a salvage of twenty-seven patients.

Many physicians have repeatedly observed recurrences which have been almost asymptomatic, and which displayed a definite tendency to subside. We have at present one patient, who had a large segment of ileum and the large bowel to the mid transverse colon resected by the Lahey Mikulicz method ten years ago, who has a recurrence. He maintains his weight, carries on a gainful occupation, and plays golf and is being treated conservatively. Others who are known to have had incomplete removal of their lesions get along without great inconvenience.

Indications for surgery are not clear cut nor universally accepted by internists or surgeons. Certainly an x-ray diagnosis or an incidental finding of regional enteritis does not warrant radical surgery. Those who have had most experience with the disease and its treatment feel that surgery at present, when indicated, holds the greatest promise for relief. Both Garlock and Bockus indicate that their attitude toward treatment is, that they prefer not to operate until compelled to do so because surgery often proves to be only palliative. Indications for surgery of recurrent disease are similar to those for primary intervention. Garlock adds that all forms of treatment in a disease like ileitis, without known cause, must be empiric. Therefore, if surgery is palliative, their results must be considered extremely good. Frobese states that he is in agreement with the internists, namely, that only those cases deemed complicated or completely intractable to rigorous medical treatment, should come to operation.

Definitely, those patients who do not respond to medical treatment, who lose weight and bleed, or are unable to maintain a good state of nutrition, should be treated by operation. Similarly, those patients who develop abscesses and/or fistulas will require surgical treatment. Intestinal obstruction, either partial or complete, usually demands some type of surgical relief.

Summary

Although surgery offers the most successful method of treatment, it does entail a very definite mortality and a high rate of recurrence. It must be employed in carefully selected cases until the etiology of regional enteritis and better methods of treatment are discovered. For the present, surgery must be considered as palliative—certainly not curative.

HAZARDS OF THE AGED

Attention has been directed by press, radio, and television to the frightful toll of disaster deaths resulting from highway travel, airplane and train accidents, explosions, fires, and the like. . . .

It should not surprise us, therefore, to learn from the *Statistical Bulletin of the Metropolitan Life Insurance Company*† that accidents are a major threat to the life of the aged "outranking every other cause of death except the cardiovascular diseases and cancer. Of the 100,000 deaths from accidents in the United States annually, about 25,000 occur among people at ages sixty-five and over." These elderly people contribute one-fourth of all the victims of fatal accidental injury, although they comprise only 8 per cent of the total population. The death rate, says the *Bulletin*, in the above group is three times the rate at ages forty-five to sixty-four and seven times that at ages one to fourteen.

Three-fourths of all the fatal accidents among the elders were of two types, falls and motor vehicle injuries. Motor vehicle injuries were most prevalent for men and falls for women.

In terms of actual death rates, the total accident mortality among males was not far from twice that for females. Moreover, the toll from motor vehicle accidents was virtually three times as great for men as for women at ages sixty-five to seventy-four. The wide sex difference in the mortality from motor vehicle accidents reflects mainly the higher proportion of time spent by men on streets and highways. In each sex the majority of such fatalities were among pedestrians, indicating the difficulty that older people have in coping with modern traffic conditions. . . .

Accidents are much more amenable to control than any of the other major causes of death among the aged. The record for the past two decades shows that the mortality from accidents at the later ages has declined more rapidly than that from disease. Further progress could be made if elders learned to adjust their habits in accordance with their changing physical condition and if their environment, particularly in the home, were made as free from hazard as possible.

There is certainly a responsibility here stated and laid at the physician's doorstep. He can impress upon the elderly their limitations and upon their families the importance of eliminating all possible hazards in the home.—Editorial, *New York State Journal of Medicine*, February 1, 1954.

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†October, 1952, p. 5.

BOWEL OBSTRUCTIONS OCCURRING DURING THE POSTOPERATIVE PERIOD

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THERE is no field of surgery that is beset with more difficulties than is the differential diagnosis of the various types of bowel obstruction that occur during the postoperative period. Nowhere are the paths more uncharted. Often more than one condition is present, each accentuating the other. At times an accurate diagnosis is impossible; and, after exhausting every clinical and laboratory aid, one has to rely on his own intuition. In no other situation does "seeing the patient frequently" pay off so well, as it is often necessary to examine a patient many times before making up one's mind as to what is happening.

Diagnosis during the postoperative period is difficult because of several factors. First, we have a recent tender laparotomy wound. Second, there is always a normal postoperative ileus. Third, there are always normal chemical and electrolyte disturbances which have been initiated by the stress and shock of the operation. Fourth, there is a natural aversion on the part of the surgeon to re-open an abdomen which has just recently been explored. This is very justifiable in view of the high mortality associated with such procedure in the past. In 1950 Tito Korner reviewed 132 re-operations for various causes during the postoperative period of which 42 per cent were necessitated by intestinal obstruction. Of the 132 cases re-operated upon, 94 died, an over-all mortality of 70 per cent.

When confronted with a possible intestinal obstruction during the postoperative period, there are a few questions that are peculiarly pertinent to the problem, and if possible, we should always try to get satisfactory answers. First, is this a case of ileus, or is it a mechanical obstruction, Second, if there is a mechanical obstruction, is it simple or is it strangulated? Third, is the obstruction due to an inflammatory mass? The answers that we get to these questions will do much toward deciding whether or not it will be necessary to re-open the patient or whether conservative treatment is safer and will be adequate to relieve the situation. For if one re-operates an ileus, he adds fuel to the fire and usually makes his patient worse. If he re-operates a patient with an inflammatory mass and breaks through nature's barrier, he may cause a spreading peritonitis.

Any type of intestinal obstruction may occur during the postoperative period, and it is not my intent to cover the entire subject. What I am going to do is to outline five cases that presented some particularly difficult diagnostic problem during the postoperative period.

Case Reports

Case 1.—A man, sixty-five years of age, had been complaining of gas pains for about two weeks. X-rays were taken by the family physician and a constricting lesion, probably carcinoma of the transverse colon, was found. He was prepared for surgery. Neomycin was given for thirty-six hours prior to operation. He was operated on the 24th of September, 1952, at which time a contracted lesion of the transverse colon was found. On pathological examination this was found to be not a carcinoma, but a segmental area of ulceration approximately 3 cm. in length and extending completely around the bowel. His bowels moved normally on the fourth postoperative day, and he passed a moderate amount of stool and flatus following a small enema. On the sixth postoperative day, the patient complained of severe gas pains, developed abdominal distension, and a moderate amount of bloody serous drainage was noted on the dressings. Patient was x-rayed and large distended loops of small bowel were found. Cramping pains persisted for about three days, at which time patient began to have several loose stools and the cramps subsided. Patient went home on the twelfth postoperative day. The only plausible explanation for the x-ray findings and for the pains of which the patient complained from the sixth to ninth postoperative day was that when I examined him in the office I found that he had developed a moderate size ventral hernia. Apparently on the day that the bloody drainage appeared, a loop of small bowel had herniated into the wound causing a temporary complete obstruction. As this hernia enlarged, the obstruction relented and the mechanical obstruction was relieved. At no time was any bulging noted in wound while patient was lying in bed in the hospital.

Case 2.—A woman, aged forty, was first seen on the eighth postoperative day. She had been operated upon by a gynecologist for an ovarian cyst. There had been nothing unusual about the surgery except that during the course of removal of the cyst it had ruptured and a large amount of dark hemorrhagic material was released into the peritoneal cavity. Postoperative convalescence was normal until the fifth day, at which time she began to complain of gas pains and vomiting. These colicky pains

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persisted for about three days getting progressively more intense and she began to have severe projectile vomiting. When examined, the patient had evidence of acute mechanical small bowel obstruction with active bowel sounds and distention. X-ray showed marked distention of upper jejunum. As soon as her fluids and electrolytes were replaced, the patient was operated upon. The point of interest in this case was the extremely firm plastic adhesions that started to cause obstruction signs on the fifth postoperative day. In the central portion of the small bowel it was completely and solidly agglutinated together for a distance of 2-3 feet. The bowel above and below this point was normal, making one feel that probably the fluid from the ruptured cysts had been extremely irritating and was the etiological factor in causing these firm plastic adhesions and resulting marked bowel obstruction.

Case 3.—This patient, a woman, aged seventy-five, I saw on her first postoperative day. A bad risk cardiac patient, she had been operated upon the previous day by another surgeon for a strangulated right femoral hernia. By the time the hernia sac was opened, the bowel had already slipped back into the peritoneal cavity and he was unable to inspect it. The next day he was worried about the condition of the bowel and asked me to follow the case along with him. Daily leukocyte and differential counts were obtained. The leukocytes remained persistently high, around 20,000. The abdominal distention and tenderness remained and there were distended loops of intestines seen by x-ray examination. In view of the x-ray and local findings and especially because of the persistent high leukocyte count, we decided to re-operate on the fourth postoperative day. A necrotic loop of bowel about 3 inches long was found friable, soft, and ready to rupture. This loop of bowel was resected, and an end-to-end anastomosis was carried out.

The recognition of a gangrenous loop of bowel inside the abdomen is probably one of the most difficult problems with which we have to deal. I think it is the recognition of this difficulty that has led the trend away from the conservative or non-operative treatment of mechanical obstruction. Rare is the surgeon who has not at one time or another missed a strangulated bowel—always to his regret.

The recognition of a strangulated loop of bowel has been based on criteria upon which there has been rather general agreement among surgeons. These are as follows:

1. Signs of peritoneal irritation
    (a) tenseness
    (b) muscle rigidity
    (c) rebound tenderness
2. Fever
3. Leukocytosis
4. Rapid pulse
5. Presence of mass

On the basis of these criteria, it was felt that one could be fairly certain of the presence of strangulation obstruction.

It is interesting and a bit disconcerting to read a recent article by Walter Becker. He made a study of 412 cases of acute adhesive obstruction attempting to determine whether the presence or absence of the above-named findings is sufficient to differentiate a strangulated from a simple obstruction. The results of his study showed the following: Leukocytosis was present in 50 per cent of the cases of strangulated obstruction and in 44.4 per cent of cases of simple obstruction. Of patients who died with strangulated obstruction, 60.8 per cent had a normal leukocyte count. Fever was present in 22.7 patients with strangulated obstruction, but also 16 per cent of patients with simple obstruction had a fever of 100 or more. Tachycardia with pulse of 100 was present in 22.8 per cent cases of strangulated obstruction but was also present in 14.8 per cent cases of simple obstruction. Tenderness and findings of peritoneal irritation were present in 81.8 per cent cases of strangulation obstruction but was also present in 72.2 per cent cases of simple obstruction. He did find, however, that it was only the occasional case that did not exhibit at least one of the "signs of strangulation." The conclusion reached was that the presence or absence of tenderness, fever, tachycardia, leukocytosis or palpable mass is not sufficient to differentiate simple from strangulation obstruction. He concluded that obstruction due to old adhesions should be surgically cared for as soon as blood volume, fluid and electrolyte balance are corrected. On the other hand obstruction due to recent adhesions should usually respond to suction treatment by gastrointestinal tube but he warned that conservative therapy must be used with great caution, and unless the evidence of recovery was unequivocal, an operation is in order. He showed that in cases where suction decompression has been abused, the mortality rate was 38.4 per cent. The moral to be learned is that regardless of the clinical and laboratory findings, a strangulated loop of bowel must al-

TABLE I.

Post-operative day	6th	7th	8th	9th	10th	12th
Symptoms and signs	Apprehensive Vomiting Distended Rapid pulse Liquid stools	Depressed Listless Passing flatus Hiccoughing Liquid stools	Exhausted Listless Cyanotic Perspiring Cold and clammy B.P. 80/70	Alert Distended Copious liquid stools	Alert Vomiting Distended Liquid stools	Eating No emesis
Urine	200 cc	100 cc	700 cc	1300 cc	1000 cc	700 cc
Blood chlorides	580 Mg.	478 Mg.	484 Mg.	384 Mg.	522 Mg.	580 Mg.
Blood urea			43.7 Mg.	46.4 Mg.	29 Mg.	19.4 Mg.
Blood CO_2	73.9 vol. %	71 vol. %	82.5 vol. %	73 vol. %	70 vol. %	61 vol. %
Intake						
I.V.	2000 cc	3500 cc	4000 cc	3250 cc	2500 cc	500 cc
Mouth	4380 cc	1200 cc	4590 cc	1740 cc	2495 cc	1950 cc
Fluid Loss						
Emesis suction	4400 cc	4000 cc	4900 cc	1400 cc	2200 cc	0
Stool	100 cc	0	100 cc	100 cc	500 cc	1500 cc
Salt	2.5 Gm.	8.5 Gm.	8.5 Gm.	20.5 Gm.	8.5 Gm.	0
KCl I.V.	27 M.E.	54 M.E.	27 M.E.			

ways be considered as a possibility in any mechanical obstruction.

Case 4.—A woman, aged forty, mother of seven children, was first seen on her sixth postoperative day. She had been operated upon for an acute supperative but not ruptured appendix on November 10, 1951. Her postoperative course was characterized almost from the first by inability to alimentate. This started almost at once after surgery and persisted up to the time that I first saw her on the sixth postoperative day. During this period she had very little pain, had frequent emesis, numerous liquid bowel movements and had been passing flatus practically every day. When I saw her on the sixth postoperative day, she was listless, markedly distended, hiccoughing, and with rapid pulse. During her postoperative period she had been receiving about 2000 c.c. of fluid daily, half of this in saline and half in distilled water. Nasal suction was inserted on the third postoperative day. She did not tolerate it well, and it had been discontinued. She had a steadily falling urinary output from 900 c.c. on the third day to 100 c.c. on the seventh postoperative day. Radiographs taken on the sixth and eighth postoperative days showed what was interpreted by the roentgenologist on both occasions as being a mechanical, small bowel obstruction. Needless to say, this opinion worried us considerably.

A nasal suction was again inserted and approximately 2000 c.c. of gastric contents were aspirated. This gave the patient much relief, but distention of the small bowel persisted. Table I gives a résumé of her course until she was recovered on the twelfth day without further operative interference. The picture here was one of an acute distention of the stomach with ileus of small bowel. The loss of fluids and electrolytes resulted in a severe degree of hypokalemic alkalosis. Both the ileus and the alkalosis cleared after fluid and potassium loss were corrected.

Paralytic ileus is seen in pratically every postoperative laparatomy to some degree and as such is considered normal. When present to an excessive degree, it is due to a stimulation of an overactive sympathetic nervous system. Distententention interferes with normal absorptive action of the bowel, so that gas which normally would be absorbed tends to accumulate and aggravate the distention already started. The ileus here was accentuated by the hypokalemic alkalosis which was present to a marked degree.

Clinically ileus is characterized by marked distention of both the large and small bowel usually with a rapid onset soon after surgery. Intestinal colic is usually absent and there are no bowel sounds. The treatment of choice is a gastrointestinal tube, the best of which we have found to be either the Wild or the Grafton-Smith tube. Because either of these has only a single suction opening protected on the one side by the mercury sac and on the other by an air balloon, these tubes function better than the Miller-Abbott tube which has numerous openings. The use of potassium is beneficial in improving ileus. Often a patient will have a good movement as soon as he is given his first intravenous injection of potassium chloride.

Case 5.—This patient ran a similar clinical course, yet her pathologic history was entirely different. She was first seen on her ninth postoperative day. She was operated upon July 23, 1952, by a gynecologist for an unruptured ectopic pregnancy. Her left tube had been removed for a similar condition eight years previously, so at this time in addition to removing her right tube, a hysterectomy was performed. On her third postoperative day, she began to vomit and her abdomen became distended. Nasal suction was inserted, and she continued to lose from 1,400 to 3,000 c.c. daily by nasal suction. Her abdomen continued to be distended but every day she was passing small amounts of flatus. She had been receiving on the average 2000 c.c. of intravenous fluids daily, half of which had been in saline. Flat plate x-ray examination of the abdomen was taken on the eighth postopera-

TABLE II.

Post-operative day	9th	10th	11th	12th	13th	14th	15th
Symptoms and signs	Thirsty Restless Depressed Incoherent Distended	Disoriented Noisy Involuntary Distended Passing flatus, stool	Disoriented Incoherent In restraint Vomiting Distended	Coherent Cooperative Distended	Oriented Quiet Passing flatus	Distended Passing flatus	Re-operated
Urine	900 cc	775 cc	800 cc	1900 cc	1750 cc	2300 cc	
Blood chloride	424 Mg.	502 Mg.	482 Mg.	540 Mg.	437 Mg.	560 Mg.	
Blood urea	78 Mg.	68.4 Mg.	40 Mg.	13.3 Mg.	12.5 Mg.		
Blood CO_2	77 vol. %	78 vol. %	97 vol. %	65 vol. %	67 vol. %	44 vol. %	
Blood potassium	2.75 M.E.—L	3.75 M.E.—L					
Intake							
I.V.	4000 cc	2000 cc	2500 cc	4000 cc	3500 cc	2000 cc	
Mouth	1300 cc	1000 cc	800 cc	1200 cc	800 cc	700 cc	
Fluid loss							
Emesis suction	1050 cc	500 cc	925 cc	1460 cc	1400 cc	2200 cc	
Salt intake	20 Gm.	4 Gm.	8.5 Gm.	4 Gm.	12.75 Gm.	12.75 Gm.	
KCl. I.V.	40 M.E.	60 M.E.	100 M.E.	100 M.E.	80 M.E.	80 M.E.	

tive day and was interpreted as follows: "Film of abdomen shows considerable distention of several segments of small bowel in the upper mid portion of abdomen, slight distention of the stomach, some gas throughout the colon. The appearance could be consistent with an ileus, however the possibility of a strangulation type of small bowel obstruction is to be considered." When I first saw this patient she was incoherent, restless, disoriented and moderately distended. Occasional gas sounds could be heard. The patient was very thin and presented a pot-belly type of distention in the lower abdomen. Table II gives a résumé of her clinical course and laboratory findings.

A radiograph taken on the fourteenth day showed a definite increase of gas in the colon. Presence of gas in the small bowel remained about the same, and the conclusion was a "partial mechanical obstruction not definitely excluded." The patient by this time had entirely recovered from her hypokalemic alkalosis. She was clear mentally, co-ordinated and alert. By this time I was convinced that she had a partial mechanical bowel obstruction. She was re-operated upon the fifteenth postoperative day. The small bowel was greatly distended to about 4 feet from the ileocecal valve. Gastrointestinal tube had stopped at that point. From there on to the cecum, the bowel was matted together by very firm adhesions agglutinating the bowel to itself and to the pelvis. The loops were separated and the obstruction relieved. Postoperative convalescence was normal.

If we have any reason to be happy about this case, it is the fact we postponed surgery until her potassium deficiency and alkalosis were corrected. Had she been reoperated during this phase, I am sure that a fatality would have ensued.

Discussion

These two cases illustrate how hypo-potassemia can readily develop in cases when one could expect a normal convalescence. One patient with an unruptured acute appendix and the other, with an unruptured ectopic pregnancy, had been receiving average amounts of parenteral fluid daily, but both were losing more electrolytes and fluids than they were receiving. Suddenly both were in marked toxemia from hypokalemic alkalosis. This is the most severe type of potassium deficiency. To produce this condition, three things are usually necessary: (1) inadequate intake of potassium; (2) loss of chlorides from the gastrointestinal tract, and (3) the increased secretion of adrenocorticotropic hormones resulting from the stress of surgery which hastens the rate of potassium excretion. When these three factors are present, there often results a profound illness with fever, anorexia and nausea, distention, muscular weakness, mental depression, confusion and delirium, irregular pulse and fall in blood pressure—the picture of hypokalemic alkalosis.

Abdominal distention which may be thought to be due to a neurogenic ileus may be due entirely to a potassium deficiency that may clear up quickly after the potassium deficiency is corrected. Also a delayed emptying of the stomach postoperatively may be cleared up quickly with the administration of potassium.

The body has no mechanism for storing potassium. Any excess of its normal needs is excreted in the urine. Any disturbance of the acid base balance—either alkalosis, which occurs when there is a loss of large amounts of gastric juice, or acidosis induced by losses of pancreatic, biliary or intestinal secretion will result in increased urinary excretion of potassium. Gastric juice contains the highest concentration of potassium of all the body secretions and may contain as much as 40 milliequivalents per litre which is two or three times the concentration of potassium in serum. In this way it is easy to see how continued vomiting or loss by nasal suction will rapidly induce a condition of potassium deficiency.

If a patient loses three or four litres of gastric juice daily, he will lose 80 to 100 milliequivalents of potassium, or approximately six to eight grams daily.

Cardiac disturbances are present frequently in potassium deficiency. Cardiac arrythmias, fall in diastolic blood pressure, and cardiac dilatation have been observed. Cardiac arrest during surgery has been noted in potassium deficiency. Wherever possible surgery should be postponed until potassium deficiency has been cleared.

Electrocardiograph findings are definite in character, but are a poor indicator of the exact potassium level. The changes that should be looked for are:

1. Decreased T wave voltage going on to a flattening or inversion of the T wave
2. Prolongation of the QT interval
3. Depression of the RST segment
4. Prolongation of the PR interval

Conversely, the development of a state of hyperpotassemia may be followed and diagnosed quickly by means of the electrocardiogram during rapid administration of potassium salt. Intravenous potassium can be given until the T wave becomes high peaked.

Since hypokalemic alkalosis in the surgical patient is most commonly produced by chloride loss together with potassium deficiency, the simplest rational treatment is the administration of potassium chloride. The chloride stays outside the cells and repairs the alkalosis, and the potassium moves inside the cell and helps repair the cellular potassium deficiency. This condition does not respond to sodium chloride and ammonium chloride if a co-existing potassium deficiency is present and is not cleared up. Hypokalemic alkalosis will rarely be seen if a surgical patient is managed by what is known as the "three-day rule." By this is meant that after three days of zero potassium intake, potassium should be provided by the mouth or vein.

Clinically, the administration of potassium chloride is usually followed by prompt clinical improvement. It takes on the average three days to restore serum potassium to normal, and it should be continued for another four days to prevent recurrence of potassium deficiency. Three grams or 40 milliequivalents, given intravenously with glucose, in distilled water or saline should be given three to five times daily depending on the needs of the patient. It must be administered very cautiously in a patient whose urinary output is low, as was the case of the fourth case I presented. Daily blood potasium levels should be obtained. Fluids containing potassium should be given slowly, no faster than 120 to 180 drops a minute.

Prophylactic use of potassium should be used on (1) patients who loose large amounts of gastrointestinal secretion by suction or fistula; (2) patients who receive only parenteral alimentation for a period longer than three days; (3) patients undergoing extensive surgery.

In all these patients, 2 litres each containing 2 grams of KCl should be given daily. In addition, another 3 grams should be given for each 1,000 cc. of gastro-intestinal fluids removed by suction or lost by fistula during a twenty-four-hour period.

In conclusion, I would like to emphasize that there is no place in the field of diagnosis where being wrong is so costly and where the rewards of being right pay off so handsomely as they do in the problems of intestinal obstruction that occur during the postoperative period.

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DRUG THERAPY IN HYPERTENSION

A survey has been made of fifteen medical centers working on the problem of hypertension to assess the over-all opinion of the various drugs capable of specifically lowering the blood pressure. All fifteen centers had used hexamethonium, eight ergot derivatives, thirteen veratrum viride derivatives, and thirteen had used 1-hydrazinophthalazine. All fifteen observers reported administering the drugs to both hospitalized and ambulatory patients. Seven of the physicians thought that there was insufficient information to warrant widespread use of these drugs in the treatment of hypertensive patients at the present time. Two more advocated use of this type of therapy only in patients with malignant hypertension, and then only with proper precautions.—Moyer, J. H.: A survey on drug therapy in hypertension, Med. Records & Ann., 47:512 (May) 1953.

HYPERSPLENISM

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HYPERSPLENISM is an abnormally increased activity in one or more of the spleen's functions. It manifests itself by diminishing the numbers of one or more of the three cell types of the blood. The condition may be inherited (familial hemolytic anemia); it may arise out of a primary blood dyscrasia, a generalized infection, or metastatic neoplasm to the spleen. Spontaneous cases are frequent in that hypersplenic episodes occur which are unrelated to any known cause. Any instance in which the spleen enlarges may cause hypersplenism. It is important, however, to differentiate between splenomegaly and hypersplenism—the former being only an anatomical term, the latter being functional. A normal pregnancy or insignificant trauma or minor infections may precipitate severe exacerbations in vulnerable individuals.[21]

Pathogenesis

To better understand the pathogenesis of this syndrome we must look at the anatomy of the spleen. The capsule is made up mainly of smooth muscle fibers which penetrate to the depths of the organ. This enables a marked contraction of the spleen with exercise, massive bleeding, anger or the injection of adrenaline.[18,21] The spleen in a relaxed living body is three to five times heavier than the spleen at autopsy, for there is marked contraction of the organ at death's door. The blood courses slowly through the splenic pulp (so slowly at times that the blood does not appear to be moving) and finally ends in the venous sinuses.[18] Throughout its course the individual cell (be it erythrocyte, leukocyte or platelet) must stand a rigid inspection for countless reticulo-endothelial cells lining the vascular channels. And woe be it for the damaged or senile cell, for there is a selective phagocytic action by the reticulo-endothelial cells on the defective cell. It is immediately apparent that the spleen can influence levels of the blood elements by vascular storage and phagocytosis. In addition, the spleen has been estimated to contain one fourth to one third of the lymphoid and endothelial tissues of the body and these cells are thought to be one of the chief producers of antibodies in various immunological defense mechanisms.[21,27] Up to this point there is rather complete agreement among hematologists. Unfortunately, however, most of these men go their separate ways in considering the pathogenesis of the syndrome. A few feel that a perverted storage and phagocytic function is the cause of hypersplenism;[8,9,10] more men think that abnormal humoral factors causing a suppressive effect on the bone marrow and destructive effect in the peripheral circulation is the cause;[7,11,14,25] recently most hematologists are of the opinion that there is probably a combination of these factors in the production of hypersplenism.[12,18,22,24,27]

It is interesting to note that the normal spleen can be removed with impunity, leaving no evident disability.[8,22] On the other hand, a splenectomy in certain hypersplenic states is mandatory to preserve the life of the patient. Here then, we have a true medical paradox, an organ for which the body has no apparent need when normal but with severe and even death-dealing effects when perverted.

Diagnosis

The history is important in the diagnosis of hypersplenism but since it varies with the different syndromes it will be dealt with later. A constant physical finding is splenomegaly. If one cannot palpate the spleen but there is a great suspicion of hypersplenism, it would be well to get a scout film of the abdomen to determine the size of the spleen since one has great difficulty in palpating the spleen until it weighs 500 grams. The one notable exception to splenomegaly in this condition is idiopathic thrombocytopenic purpura, and although the spleen is not clinically enlarged, the organ at the time of surgery frequently weighs two to three times the normal spleen (approximately 150 grams). A peripheral cytopenia is present, the cell type or types affected varying with the syndrome with which one is confronted. The bone marrow invariably shows compensatory hyperplasia of those cells which are deficient in the blood. Whether there is normal maturation or

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maturation arrest varies according to the prejudices of different authors and the technical skill of the man looking at the bone marrow smear. The adrenaline test is a subject of much controversy among hematologists, and here again, opinion is equally divided as to the value of the test. If, after injecting enough adrenaline to cause an increase in the pulse rate (usually 0.5 to 1.0 cc. 1:1000 solution), one finds an increase in the cellular elements which are deficient in the particular syndrome one is dealing with, the proponents of the test claim that this is proof of splenic hypersequestration.[8,9,10,26] The opponents[7,24] of the test counter by saying that approximately 50 per cent of patients who have unquestioned hypersplenism will not have a significant rise in the depleted cells; moreover, many patients who have long had their spleen removed surgically will have a positive test. It should be said that the adrenaline test is frequently used in the diagnostic work-up of a suspected case of hypersplenism, and when positive it indicates, but does not necessarily prove, splenic hyperactivity. Various tests Coombs, trypsinized red blood cells, Weiner's, et cetera) which demonstrate incomplete antibodies, either in the patient's serum or adsorbed on the patient's red blood cells, are helpful confirmatory tests. It is important to realize that although the test was originally devised to detect acquired hemolytic anemias, a small but significant number of positive titres are obtained in many other hypersplenic syndromes which do not have a hemolytic component. The titres obtained in tests demonstrating univalent antibodies do not appear to be correlated with the activity of the disease.[25] Splenic puncture has been advocated recently by Calkins[4] not for making a diagnosis of primary hypersplenism (a very difficult if not impossible pathological diagnosis) but for finding the cause of acquired hypersplenism seen in conditions such as obscure malignant lymphomas, infections and certain metabolic disturbances or for ruling out cases of myeloid metaplasia. The only contraindications to splenic puncture are a platelet count less than 50,000 or impaired bleeding, clotting, and prothrombin time. Sixty cases were biopsied without event. One merely infiltrates with novacaine down to the spleen and, with a 20-gauge needle and a 5 cc. syringe, aspirate a small amount while the patient is in deep inspiration.

TABLE I. DIFFERENTIAL DIAGNOSIS OF PRIMARY AND SECONDARY THROMBOCYTOPENIAS

	Etiology	Familial	Splenomegaly	Sex	Age	Lymphadenopathy
Primary H., T.	?	occ.	0	F 2:1	young	0
Secondary H., T.	Hypersensitivity to chemicals, vegetables, animal products, physical agents, blood dyscrasias, anemias, infections, any disease associated with splenomegaly	0	present	both	any	occ.

Classification

The following is a list of hypersplenic syndromes which are well recognized:

(a) Primary hypersplenism, thrombocytopenic (idiopathic thrombocytopenic purpura or Werlhof's disease).
(b) Secondary hypersplenism, thrombocytopenic (acquired or secondary thrombocytopenic purpura).
(c) Primary hypersplenism, hemolytic (familial hemolytic anemia).
(d) Secondary hypersplenism, hemolytic (acquired hemolytic anemia).
(e) Primary hypersplenism, neutropenic.
(f) Secondary hypersplenism, neutropenic.
(g) Primary hypersplenism, pancytopenic.
(h) Secondary hypersplenism, pancytopenic.

It is beyond the scope of this paper to describe in detail the above listed syndromes; however, the salient features and the differential diagnoses will be discussed. It should also be emphasized that one rarely sees a "pure" neutropenia, a "pure" thrombocytopenia or a "pure" anemia.[8] Although the predominant syndrome may be hemolytic anemia, for instance, if one looks carefully enough there will usually be a cytopenia in one or more of the other cell types. Curiously enough a hypersplenic syndrome with a diminution in one cell type may completely reverse itself—the previously diminished cell type will come back to normal, only to have a precipitous drop in one or both of the other cellular elements. This phenomenon occurs occasionally after splenectomy.[8]

Thrombocytopenic Syndrome

The thrombocytopenic syndrome is characterized by spontaneous bleeding anywhere in the

TABLE II. DIFFERENTIAL DIAGNOSIS OF PRIMARY AND SECONDARY HEMOLYTIC ANEMIAS

	Etiology	Familial	Coomb's	Age	Results from Splenectomy
Primary H., H	?	always	pos. 32%	children	98% cures
Secondary H., H.	Chemicals, infections, carcinoma, leukemia, lymphomas, any condition asso. with splenomegaly	0	pos. 83%	Adults	30-40% cures

body. The onset and course are variable. Typical laboratory data are: low platelet count; increased bleeding time; positive cuff test; no, or poor, clot retraction; decreased prothrombin consumption (increased prothrombin conversion time).[19] Occasionally one may find an anemia secondary to the bleeding. Bone marrow shows adequate numbers of megakaryocytes, but platelet production is greatly diminished according to most authors.

In treating the primary or idiopathic thrombocytopenias, age is an important factor. In an adult, surgery is considered to be an emergency procedure in some clinics because of imminent cerebral hemorrhage.[7,8] In children, a period of observation for three to six months is indicated if hemorrhage is not severe, for several cases of spontaneous recovery have been reported.[1] One is also more prone to do surgery on females early because of massive bleeding during menses, and more frequent and severe recurrences in females. Those cases which have bone marrow eosinophilia and good response to ACTH or cortisone are considered to have a favorable prognosis. Therapy, preoperatively, is blood transfusions if indicated, cortisone, and recently, plaelet-rich polycythemic blood with siliconized apparatus has been advocated.[19]

In treating the secondary thrombocytopenias, it is mandatory to find the cause of the underlying or primary disease process and treat that if possible (i.e. in lymphatic leukemia irradiate the spleen). Blood transfusions and ACTH are used in some cases. If treatment of the primary disease is not possible and the bleeding is a threat to the patient's life, surgery is necessary. The mere fact that surgery does not affect a cure in every case, and that the disease is self limited in certain cases, mean that there are other factors we know nothing about in this disease. Five year "cures" for the primary form are 70 per cent, for the secondary form 54 per cent.[19] Approximately one third of the failures became cures after re-exploration revealed accessory spleens. It is sometimes useful to inject thorotrast for visualization of aberrant splenic tissue.

Hemolytic Syndrome

The hemolytic syndrome is characterized by weakness and jaundice with no characteristic onset or course. Splenomegaly is invariably found on physical or roentgenologic examination. A normocytic, normochromic anemia, bilirubinemia, reticulocytosis, increased fecal and urine urobilinogen, spherocytosis, increased fragility of red blood cells and erythroid hyperplasia of the bone marrow are present. It should be stressed that anemia develops only if the destructive process in the peripheral circulation is greater than the regenerative process in the bone marrow. To illustrate this point, many hemolytic anemias have M-E layers of 50 to 80 per cent in contrast to the 5 to 8 per cent M-E layer in the normal marrow.

In the primary hemolytic syndrome (familial hemolytic anemia) surgery provides the best results (cures obtained in over 98 per cent of cases; misdiagnosis is thought to be the reason for the 2 per cent failures). However, since many cases are found accidentally late in life without any manifestations of severe anemia, one should not suggest splenectomy indiscriminately. Surgery should be reserved for children, young adults or severe hemolytic anemias in the older age group.[5,17] In secondary hypersplenism, hemolytic (acquired hemolytic anemia) results from surgery are much less favorable and every effort should be made to treat the underlying disorder before one resorts to surgery. Whether it be the primary or secondary hemolytic syndrome, blood transfusions are to be avoided during hemolytic crises and before surgery.[8,9,10] In addition, surgery should be delayed until a hemolytic crisis has passed.

Neutropenic Syndrome

In the neutropenic syndrome one is usually confronted with an insidious onset of malaise and tendency to repeated infections which are difficult to clear. The course is usually chronic although numerous fulminating cases have been reported. The physical examination will usually demonstrate one or more foci of infection. Splenomegaly, neutropenia, and normal or granulocytic hyperplasia of the bone marrow (with or without maturation arrest) are invariably present. There may or may not be an absolute decrease in the lymphocytes. The fact that one usually sees old segmented granulocytes and an absence of young neutrophils favors the hormonal theory in the pathogenesis of this syndrome.[27] One has difficulty in differentiating the primary and secondary forms of neutropenia unless there is an obvious underlying disorder of the spleen. If one fails to find the etiology, splenectomy is the therapy of choice. In the primary neutropenias surgery affords an 85 to 90 per cent cure rate. In secondary hypersplenism, neutropenia, surgery will lead to cures in 60 per cent of the patients. Prior to and immediate after surgery antibiotic therapy is indicated prophylactically even if no focus of infection is present.

Pancytopenic Syndrome

The pancytopenic syndrome is so variable in either the primary or secondary form that no attempt will be made to describe it. Suffice to say that at some time in the course of the disease, symptoms referable to the depletion of one or more cellular elements of the blood will manifest themselves. One may have only a thrombocytopenia for a time. This may correct itself spontaneously only to have a hemolytic crisis or a neutropenia or possibly a combination of all three processes. Again, if one can find and treat an underlying disorder of the spleen, one should do so, but more frequently this is not possible. Splenectomy gives good results in a majority of cases but late recurrences (up to five years) are not infrequent. The acute hemoclastic crisis in hypersplenism may present itself as hemorrhage, hemolytic anemia, sudden sepsis either alone or in combination with one another. Most speculation has been confined to the onset of hemolytic crisis in primary hypersplenism, hemolytic (familial hemolytic anemia).[21] Dameshek and Bloom feel that this is due not only to excessive peripheral destruction of red blood cells, but also due to a maturation arrest of the erythroid series in the bone marrow. Owren believes that crisis arises from a temporary aplasia of the bone marrow (from infection, toxins, et cetera) and the reticulocytosis seen is actually the recovery phase in which the bone marrow is attempting to compensate for a previously diminished production of red blood cells. Doan and Wiseman think that excessive destruction in the spleen could explain the crisis. Troland and Lee have demonstrated a precipitous drop in platelets with purpura when splenic extracts from a case of idiopathic thrombocytopenic purpura were injected into experimental animals. Harrington also demonstrated a precipitous platelet drop in normal persons when serum from cases of idiopathic thrombocytopenic purpura were injected[15].

The term "relative hypersplenism" has been coined to explain those cases of cytopenia from permanent bone marrow damage (toxins, drugs, heavy metals) where cellular recompensation has occurred with the splenectomy. The postulated mechanism is that the spleen had not decreased its normal destructive effect to balance an impaired bone marrow cellular production. Whatever the case might be, splenectomy with hypoplastic bone marrows has definitely alleviated hematological symptoms in several well-documented instances.[5,22,27]

Within a few hours after removal of the spleen, the neutrophils rise (as in any surgical procedure).[1] At one week there is another peak in splenic neutropenia and thereafter there is a gradual diminution to normal or slightly elevated levels. Several months postoperative there is an absolute lymphocytosis with only a relative granulopenia. Four days usually elapse before platelets attain normal blood levels and another eight days pass before maximum levels are reached. Platelet counts have been recorded as high as four million.[1] Postoperative thrombotic phenomena are quite rare, however. If the platelet count is greater than one million, prophylactic anticoagulants are employed.[1,4] The platelet count gradually diminishes to normal levels. Although it ordinarily takes four days for the platelet count to become normal there are case reports in Werlhof's disease in crisis that impresses one with the variability of response or that other unexplained factors play a major role in the spontaneous bleeding. Within minutes after clamping the splenic pedicle there is a seemingly miraculous cessation of oozing in the skin surfaces and subcutaneous tissues.[1] It is also interesting to note that in some cases, although the platelet count remains down in the spontaneous bleeding range of less than 50,000, all bleeding tendencies subside.[19] Conversely, there are cases where the platelet count rises to normal, yet spontaneous bleeding continues to occur.[5] The red blood count and hemoglobin rise slowly after a splenectomy reaching, or approaching, normal values only after two to four weeks have elapsed.[27] In the post-splenectomy state, one regularly notes Howell-Jolly bodies, reticulocytosis, normoblastosis, and target cells, which, because of their flattened form, are more resistant to hypotonic saline.

Although much knowledge has been gained from splenectomy the response is so variable that we are still unable to ascertain whether the activity of the spleen is primarily destruction of cells or inhibition of the bone marrow.[5,27] Regardless of this problem, we are still faced with the fact that the spleen *per se* is not the only regulator of the cellular elements of the blood—it is more likely that the entire reticulo-endothelial system takes part in this syndrome of "hypersplenism" and for this reason, many of the leading hematologists would rather substitute the term "hyper-reticulo endotheliosis."[17,24] Kracke has aptly summarized the proper approach to a suspected case of hypersplenism.[18]

(1) Is the spleen destroying cells more rapidly than the bone marrow is capable of producing?

(2) Is the bone marrow capable of producing a normal number of cells?

If the answer is yes to both questions, then one must decide whether it is safer to leave the spleen in or to risk surgery (1 to 5 per cent mortality rate) in removing the spleen.

References

1. Beard, M. F.: Blood dyscrasias in adults; hypersplenism. Kentucky. M. J. 46:84 (March) 1948.
2. Blackburn, C. R. B.: Indications for splenectomy: discussion of some mechanisms involved in splenopathies. M. J. Australia, 2:641 (Oct. 28) 1950.
3. Burtner, O. W.: Management of thrombocytopenic purpura; a condensed review. J. Florida M. A., 39: 330 (Nov.) 1952.
4. Calkins, E. (editor): Hypersplenism (Mass. Gen. Hosp. case). Am. Pract. and Digest Treat., 3:657 (Aug.) 1952.
5. Calov, W. L.: Indications for splenectomy. M. J. Australia, 2:644 (Oct. 28) 1950.
6. Coombs, R. A.; Mourant, A. E., and Race, R. R.: New test for detection of weak and "incomplete" Rh agglutinins. Brit. J. Exper. Path., 26:255 (Aug.) 1945.
7. Dameshek, W.: Spleen and Hypersplenism. New York: Grune, Stratton and Co., 1948.
8. Doan, C. A.: Hypersplenism. Bull. New York Acad. Med., 25:625 (Oct.) 1949.
9. Doan, C. A., and Wright, C. S.: Further studies of hypersplenic mechanism in human disease syndromes. Proc. Central Soc. Clin. Research, 20:8, 1947.
10. Doan, C. A., and others: Some cyto-immunologic aspects of hypersplenic syndromes. Tr. A. Am. Physicians, 63:172, 1950.
11. Dubois, E. L.: Acquired hemolytic anemia as the presenting syndrome of lupus erythematosus disseminatus. Am. J. Med., 12:197 (Feb.) 1952.
12. Editorial: Pathogenetic mechanisms in idiopathic thrombocytopenic purpura. Ann. Int. Med., 39:379 (Aug.) 1953.
13. Edwards, M. H.; Wagner, J. A., and Krause, L. A. M.: Sarcoidosis with thrombocytopenia: report of a case. Ann. Int. Med., 37:803 (Oct.) 1952.
14. Evans, R. S., and Duane, R. T.: Acquired hemolytic anemia. Blood, 4:1196 (Nov.) 1949.
15. Harrington, W. J.; Minnich, V.; Hollingsworth, J. W., and Moore, C. V.: Demonstration of thrombocytopenic factor in blood of patients with thrombocytopenic purpura. J. Lab. and Clin. Med., 38:1 (July) 1951.
16. Johnson, V. A.: Pathology of hypersplenism. J. Florida M. A., 39:341 (Nov.) 1952.
17. Kaplan, S. R.: Hemolytic anemia. J. Florida M. A., 39:327 (Nov.) 1952.
18. Kracke, R., and Riser, W. H.: Problem of hypersplenism, J.A.M.A., 141:1132 (Dec. 17) 1949.
19. Lozner, E. L.: Differential diagnosis, pathogenesis and treatment of thrombocytopenic purpura. Am. J. Med., 114:459 (April) 1953.
20. Marks, L. J.; Weingarten B., and Gerst, G. R.: Carcinoma of the tail of the pancreas associated with bleeding gastric varices and hypersplenism. Ann. Int. Med., 37:1077 (Nov.) 1952.
21. Rankin, E.: Hypersplenism. J. Kentucky M. A., 49:192 (May) 1951.
22. Robson, H. N.: Medical aspects of splenectomy (Honyman Gillespie lecture). Edinburgh M. J., 56:381 (Sept.) 1949.
23. Russell, H. K.: Hypersplenism; some aspects of diagnosis and treatment. Ann. W. Med. and Surg., 6:290 (May) 1952.
24. Schwartz, S. O.: Symposium on hypersplenism. J. Florida M. A. 39:327-50 (Nov.) 1952.
25. Wright, C. S.; Doan, C. A.; Dodd, V. A., and Thomas, J. D.: Hemophilia: current theories and successful medical managements in traumatic and surgical crises. J. Lab. and Clin. Med., 33:708 (June) 1948.
26. Wright, C. S.; Doan, C. A.; Bouroncle, B. A., and Zollinger, R. M.: Direct splenic arterial and venous blood studies in hypersplenic syndromes before and after epinephrine. Blood, 6:195 (March) 1951.
27. Youngman, R. A.: Hypersplenism. Nebraska M. J., 36:297 (Sept.) 1951.

TREATMENT IN CONGESTIVE HEART FAILURE

The clinical signs and symptoms of congestive heart failure result from two causes. There are symptoms caused by altered pressure-flow relationships in the cardiovascular system, and there are symptoms resulting from abnormal fluid and electrolyte metabolism, affected by several mechanisms. Therefore, therapy should be directed toward restoring a better balance between cardiac output and metabolic requirements, promotion of sodium and fluid loss, and maintenance of the equilibrium between the intake and excretion of salt.—Hanenson, I. B., et al: Pathogenesis and treatment of congestive heart failure. M. Clin. North America, 37: 643 (May) 1953.

Case Report

ARSENIC POISONING RESULTING FROM TREATMENT OF CARDIAC ASTHMA IN AN ALLERGIC PATIENT

J. S. BLUMENTHAL, M.D., F.A.C.P.
Minneapolis, Minnesota

A male paper maker, aged fifty-two, was hospitalized for severe generalized abdominal cramp-like pains, weakness, frequent vomiting and diarrhea, and an itching skin rash and asthma. These symptoms, but for the asthma and skin rash, were of twelve days' duration with a gradual onset. The pain had no relation to meals, exertion or any known factor. The diarrhea was profuse, watery, occurring four to five times daily irrespective of food eaten and was resistant to routine therapy including antibiotics.

The patient is married and has one child. The history of asthma dates back fifteen years. The symptoms first had occurred only in spring and fall and were associated with a stuffy nose. Gradually, these symptoms had become perennial and were aggravated bv dust, paint, sea food, dairy products, alcohol, cooking odors, and severe emotional episodes. He had an allergic work-up twelve years ago, but had little difficulty when he stayed away from known etiologic factors. In July, 1952, he had a "cold" with slight fever, weakness, chest pain, and cough for which he was hospitalized for two weeks and from which he did not seem to recover. Since then his "asthma" has become much worse so that he wheezed a great deal and could not work, as any exertion aggravated his wheezing.

In September, 1952, he went to an allergist who gave him skin tests and told him to stay on a new elimination regime of contactants, foods, and inhalants. He was also given injections for desenitization, but there was no improvement of note. He then was told by a friend, to go to a clinic in Mississippi where he rested and was placed on medication with some relief of his asthma, but on his return home there developed a gradual and increasing onset of his present complaints. The past history is not remarkable, except for a skin rash on the lower extremities which occurred on excessive use of soap or wool; and hives with the use of sulpha drugs three years ago. There is a history of known high blood pressure for five years, but with no referrable symptoms.

The patient was orthopneic when I first saw him in consultation on February 18, 1953, and in evident distress. He was wheezing, coughing, and a little hoarse, and expectorating a stringy thick sputum. There was puffiness about the eyelids and face, 1 plus edema of the ankles and increased pigmentation in the axillae. There was scaling in these areas as well as on the lower extremities with some keratosis of the palms and soles. The conjunctivae were congested. The nasal membranes were boggy and pale with a bluish tinge. The heart was markedly enlarged to the left with A_2 greater than P_2. Its rate was regular with a systolic murmur at the base. Wheezy, inspiratory and expiratory rales were heard in both bases. The blood pressure was 170/110. The abdomen was soft with tenderness in the right upper quadrant. The liver margin was down 4 cm.

Because of these findings and the history of medication for asthma at the clinic in Mississippi, the diagnoses entertained were (1) arsenic poisoning with hepatitis, gastroenteritis, dermatitis, melanosis, (2) cardiac decompensation, (3) allergic asthma and rhinitis, (4) hypertension heart disease, and (5) coronary disease.

The laboratory findings of interest were: a trace of albumin-uria, stools negative, x-ray flat plate of the abdomen, barium meal, and barium enema studies were negative. Blood cholesterol, and prothrombin time were normal. Thymol turbidity was 6.80. Sulphabromophthalein sodium test revealed 10 per cent retention in one hour. The cephalin cholesterol test was 2 plus. The electrocardiogram had a flat T_1 and ventricular extra systoles (Figs. 1 and 2). Chest plates taken February 19, 1953, showed a heart measuring 18.1 cm. or 60.3 per cent of the total thoracic width of 30 cm. with bilateral pleural effusion. Nails and hair and urine were submitted for analysis for arsenic with the following findings:

Report on chemical analysis of samples of nails, hair and urine in case of A. P. submitted on February 24 and 25, 1953.

Laboratory report—The nails contained 2358 milligrams of arsenic calculated as Arsenic Trioxide per 100 grams of nails. This is equal to 2.358 per cent of Arsenic Trioxide in the nails.

Laboratory report—The hair contained 0.55 milligrams of Arsenic calculated as Arsenic Trioxide per 100 grams of hair.

Laboratory report—The urine contained 0.10 milligrams of arsenic calculated as Arsenic Trioxide per 100 ml. of urine.

The patient was digitalized and put on a salt-free diet with dramatic and prompt improvement in his orthopnea, ankle edema and asthma and more gradual but persistent alleviation of other symptoms. No B.A.L.[1] was used. A 6-foot radiograph of the heart taken on February 25, 1953, showed a heart 15.3 cm. in diameter or 51 per cent of total thoracic width and on March 10, 1953, it was 14.2 cm. or 47.3 per cent.

Here, then, we have a patient who is allergic and developed cardiac decompensation. Further, indiscriminate, unsupervised treatment with arsenic complicated the picture. At the clinic in Biloxi, Mississippi, he stayed five days and then was told to go home. He was given Doxychol X for six months—one tablet p.c. and Dicalossa three times daily for twelve months. Most important, however, is the red liquid which he was told to take three times daily until all cleared up, "probably six weeks," after which it was to be reduced to twice daily for six months and then once daily for eighteen months. If, however, the symptoms recurred, the dose was again three times a day until clear of asthma and two weeks longer.

The patient was advised that any prescription given by the family doctor may be taken at the same time with no hesitation. Further, in upper respiratory infections,

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From the Section of Allergy, Department of Internal Medicine, University of Minnesota.

Read before Minnesota Society of Internal Medicine at University of Minnesota, Minneapolis, May 23, 1953.

they (I quote from the clinic literature) "have found chloromycetin particularly helpful since it is a synthetic antibiotic and has no adverse reactions of consequence." They "strongly" advise that this red medicine must not be discontinued and that an occasional skin eruption or moderate swelling is not dangerous and may be reported, but no reduction should be made in dosage. In order to keep adequate supervision, they state "you must write to us for more medicine (usually four to six bottles) and it is essential that you give us the following information so that we may make a change in your formula if it seems advisable to do so:

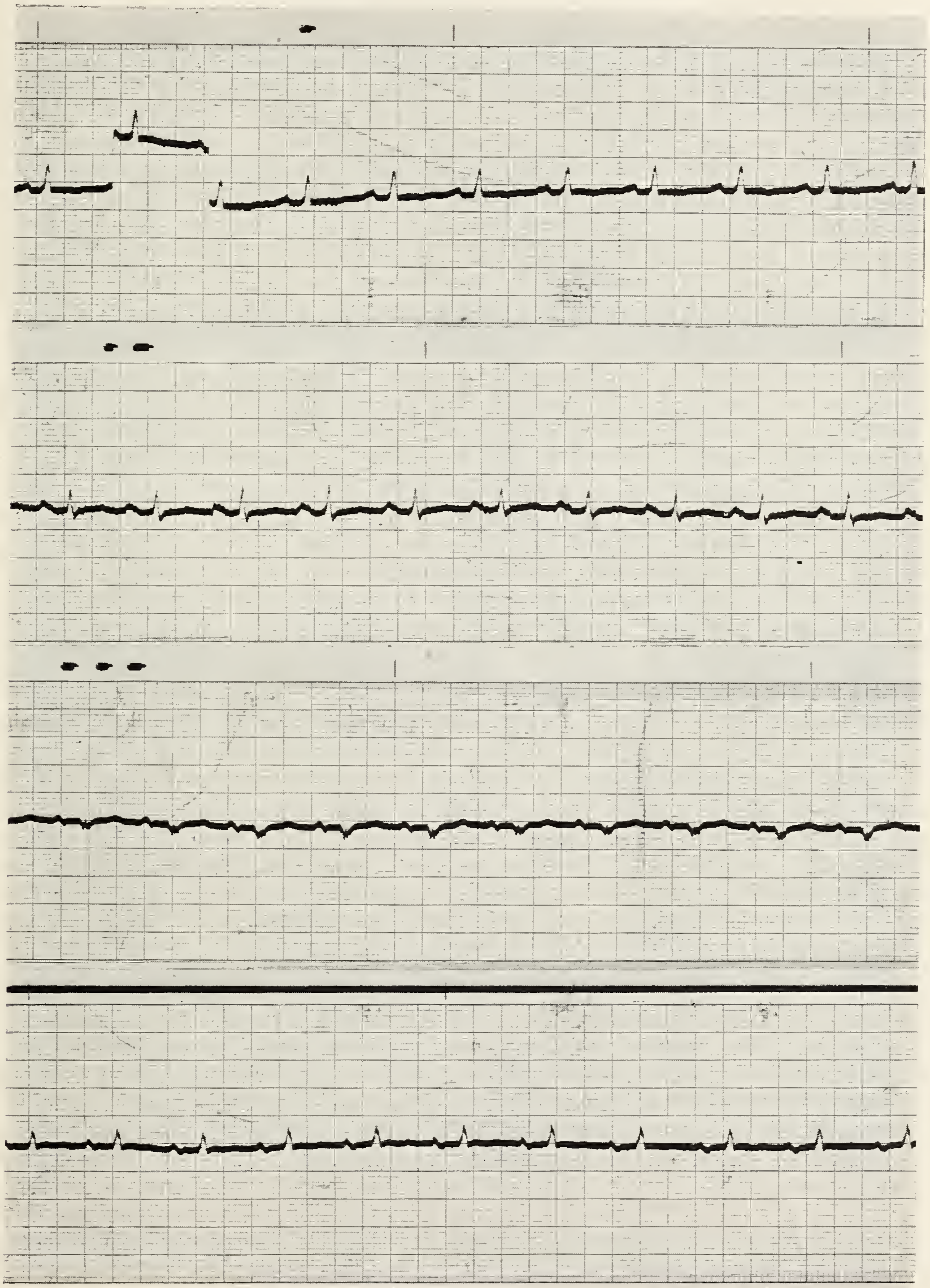

Fig. 1.

1. How often are you taking red liquid medicine?
2. Are you having any asthmatic attacks as evidenced by wheezing, excessive coughing or a 'tight chest'?
3. Are you having any other allergic manifestations, i.e., hay fever, eczema, hives?

4. Are you getting along better since on this treatment? If not, tell us the details.
5. State your weight (if known) and let us know about bowel regularity, conditions of skin, appetite, fever if present, swelling, sleep, and emotional upsets."

They assure the patient that the red coloring matter is an inert substance approved by the Federal Food and Drug Administration and the fading of color does not affect the efficiency of the medicine. Nothing of what the solvent really contains is mentioned. They refill the medicine for $2.50 per bottle postpaid.

This red medicine contains, according to a report of this clinic,[2] 8 c.c. of Fowler's solution (solution of Potassium Arsenite) in 6 oz. or 0.44 mg. of arsenic trioxide per c.c. As our patient had taken his arsenic for twenty-four days, we estimated that he had received approximately 127 mg. of arsenic trioxide. While this clinic states that in 10,000 cases of use they have had no difficulty, this is hard to follow in view of the lack of adequate follow-up. While arsenic was a known therapeutic agent in ancient Greece and Rome, and organic arsenicals have been of major importance until the discovery of penicillin in the treatment of syphilis and other protozoan diseases, other uses of arsenic in modern medicine are not many. It is a general protoplasmic poison, slow acting and may interfere with cell oxidation. Soluble salts of arsenic are absorbed from all

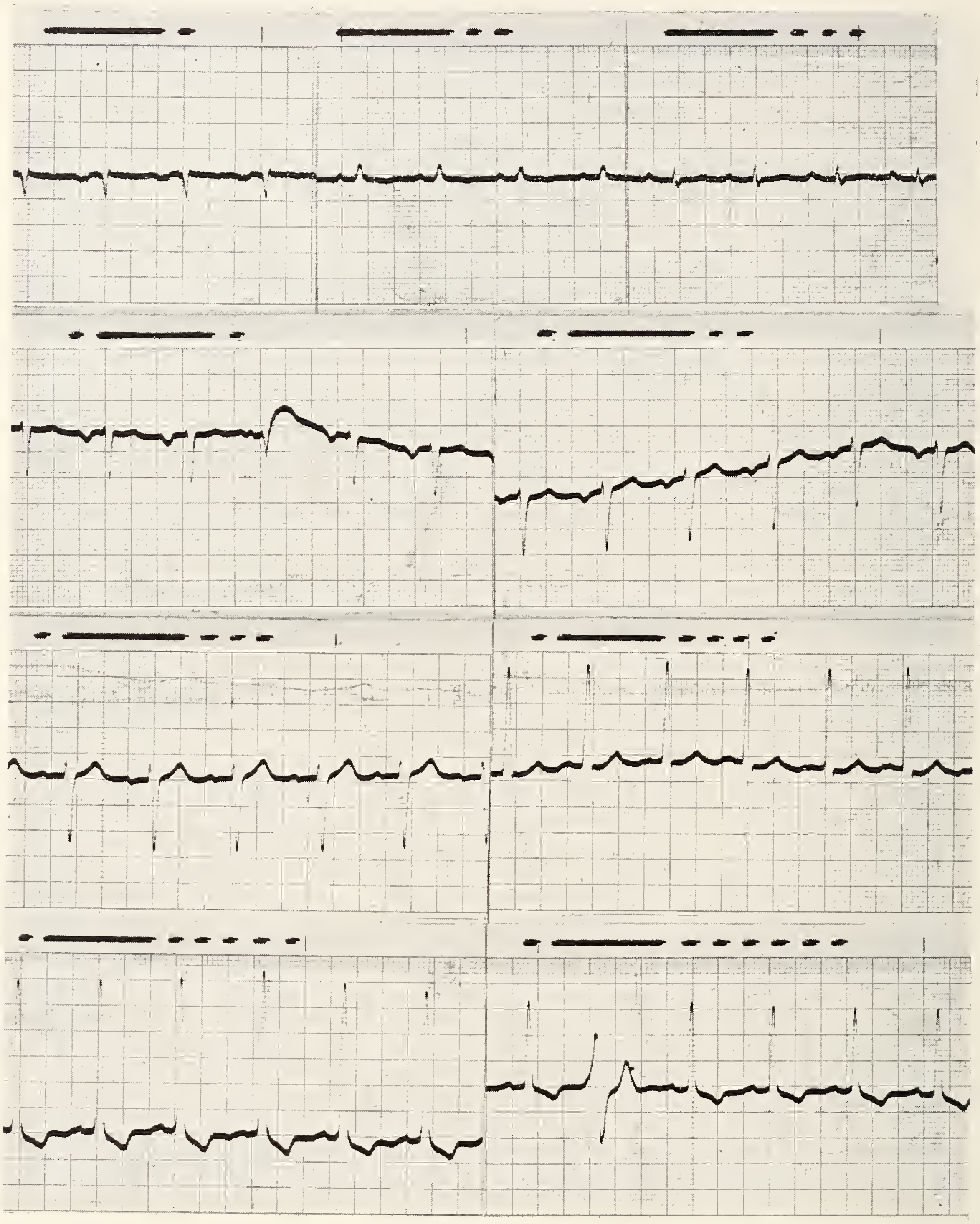

Fig. 2.

(Continued on Page 242)

President's Letter

A FRESH APPROACH

Occasionally a refreshingly amusing note is added to our sometimes too-somber existence. One such item was brought to my attention recently. It is called "Campaign IGHUGS."

We all have been almost saturated with alphabet names over the years, but this one is so well taken as to attract attention and demand explanation, and certainly some form of action. This one stands for "I'm Gonna Howl 'bout Unnecessary Government Spending."

The program itself, while fronted with a delightful title, is actually very serious-minded. It is a non-partisan program designed to create a vivid awareness of the shocking size of today's taxes: 32 cents out of every dollar earned. The purpose of the campaign is quite simple: to drive against all unnecessary Government spending and thereby help to deflate the enormous tax load.

The campaign is one of several which is planned by the Farm-City Conference Board. This organization is an informal group of public relations people representing business firms, large and small industries, farm organizations, trade associations and similar groups. Their primary objectives are to find ways to improve relationships between urban and rural people, and to develop means by which business and agriculture can work together on projects in the public interest. These objectives, though broad, are extremely worthy, and exemplify the voluntary American way of getting something done.

Nationwide organizations have joined the IGHUGS campaign. Among them is the American Medical Association, the General Federation of Women's Clubs, the National Grange and companies such as International Harvester, General Electric, and the Quaker Oats Company.

The group emphasizes that the IGHUGS campaign is not directed against all taxes. Some taxes will always be necessary, the group feels, but the campaign is directed against waste and inefficiency in Government which makes taxes higher than they have to be.

The campaign is patterned after last year's IGHAT campaign—translated to "I'm Gonna Holler About Taxes." Both campaigns are built on the idea that the voice of the people can accomplish miracles—even to the abolishment of huge amounts of wasteful spending by local, state and federal government.

It seems that the clever and engaging titles of this campaign, like last year's, will make the project catch on rapidly, and it is hoped that the IGHUGS project will be equally as successful as IGHAT was.

It is always stimulating to see American citizens organized for group action, and it is especially delightful to see them work so well toward a worthy goal using such a typically American entertaining title which is still serious and sensible. Their success depends much on the degree of support they can enlist from all other voluntary organizations with similar goals, and the medical profession could easily be a moving part of the IGHUGS campaign.

J. Chage.

President, Minnesota State Medical Association

Editorial

Arthur H. Wells, M.D., *Editor;* Henry G. Moehring, M.D., and John F. Briggs, M.D.

SUBSIDIZING COST OF ILLNESS

PRESIDENT Eisenhower's health program relating to a Government Reinsurance Corporation appears to be to the best interest of the people if it does effectively promote the present trend toward a coverage of the cost of illness through private and non-profit voluntary health insurance agencies. A few questions arise: If this plan is not a subsidy to health as claimed, what can it do that private enterprise cannot do as well, if not better? Does his basic principle of "free choice of physician" find its way deep into the make-up of the insurance companies which are to be promoted by this plan? Once established, could this Reinsurance Corporation be expanded into a compulsory health insurance scheme for the nation by another administration? Why has he avoided the Bolton plan of making medical expenses deductible from income taxes?

A tax deduction subsidy of this type could be carried out without bureaucratic overhead. Those suffering most from the cost of illness would be the principal benefactors. The amount of subsidy could be varied from a fraction to complete coverage of the costs of medical care and could be varied for different income brackets and for times of changing financial stress. The loss of revenue resulting from even a small coverage of the Nation's medical bill by this method may seem inadvisable at this time to our present government. If this is the case, we physicians should see to it that the details of a tax deduction type of subsidy are worked out for future consideration, should a less economically inclined administration run our government.

MATERNAL MORTALITY FACTORS

THE REPORT of the second year's state-wide study of the Minnesota Maternal Mortality Committee appeared in the February issue of Minnesota Medicine. The published results call for commendation of the practitioners of the state for their almost universal co-operation in supplying the data necessary to prepare case reports for study and evaluation by the committee. Without such co-operation from them, this survey could not be carried out, for as Gordon[†] has expressed it, "In our search for the controllable factors of maternal death we cannot depend upon the statistician alone, but must continue to examine not only the certificate of death but actual case reports."

Study of these case reports has resulted in important observations as to what must be done to reduce maternal mortality further in the state. For example, in reviewing deaths from hemorrhage, which is now the greatest single cause of maternal mortality, it is a matter of concern that blood replacement is still often too little and too late; this in spite of the practical experience with blood transfusions during two world wars and despite the increasing availability of blood. Nor can one condone the surprising incidence of accouchement forcé, a dangerous obstetrical procedure thought to have been generally discarded, as a contributing factor to maternal death from hemorrhage in the second year's report.

An observation may be made also concerning the choice of anesthetic agents used during delivery. The dangers of spinal anesthesia to the pregnant or parturient patient need to be re-emphasized so that other safer anesthetics, including local anesthesia, may be given proper consideration.

The maternal death rate in the state has improved so much that it is lower now than what was considered speculatively to be the irreducible minimum a few years ago. This low rate indicates that, on the whole, physicians throughout the state are giving excellent obstetric care. But it should not lull them into a state of false security so that their vigilance in preventing obstetric disaster lags or their consciousness of responsibility wanes.

There is need for improvement in such basic requirements for good obstetric care as careful and recorded history-taking, general physical and pelvic examinations, internal pelvic mensuration, and basic laboratory examinations. More frequent and adequate consultations will lower the mortality rate, if only by reducing the 40 per cent of unindicated operative deliveries noted in the reported two years of the survey. Efforts to obtain autopsies, even partial autopsies to permit tissue examination, will help to confirm the cause of death in many cases and will discover, at times, the cause of death in obscure cases.

[†] Gordon, C. A.: The maternal mortality remainder. Am. J. Obst. & Gynec., 61:1132, 1951.

The physicians of Minnesota well may be proud of their low maternal mortality rate, due specifically to obstetric complications, of 0.45 per 1000 live births in a twelve-month period in 1950 and 1951. Their pride in giving good obstetric care should encourage them to improve it further. In addition to their own efforts, it is hoped that they will give continued support to the committee of their own medical association which is carrying on the year-to-year impartial study of factors involved in maternal death with particular emphasis being placed on the preventable factors.

James J. Swendson, M.D.

SCIENTIFIC ARTICLES FOR PUBLICATION

THERE are several hundred scientific meetings held each year in the State of Minnesota. A large number of these represent the meetings of the various District and County Medical Societies, Hospital Staff groups, Northern and Southern Minnesota Medical Associations, and specialty groups. At these meetings, there are many fine scientific papers and case studies presented.

The Editors of Minnesota Medicine would welcome more of these papers and case reports for possible publication. The greater the amount of material from which to select articles for publication, the better our journal will be. We would like to have more material from the groups mentioned above, and we would like to stimulate more medical writing. There must be quite a number of physicians throughout our state who have developed special interests in various diseases or phases of medicine. You who have these interests may have real ability in medical writing, and we would encourage you to contribute to Minnesota Medicine. The editors will review your contributions and use those of unusual scientific interest.

Letters to the secretaries of the various organizations are being sent, and in them will be suggestions which may aid in obtaining papers of scientific interest. We would like to have more material from general practitioners and from smaller hospital groups. You who belong to these groups have a tremendous source of supply, and we would encourage you to send more medical writings to Minnesota Medicine.

G. J. Mouritsen

MEDICAL EDUCATION ON TAPE

A NEW kind of medical education could easily become the most important form of postgraduate training within the next decade. The California Medical Association has formed a corporation, the Audio Digest Foundation, 417 S. Hill Street, Los Angeles. This organization is busily producing sound recording tapes of literature reviews in various fields of medicine, a complete medical lecture library, on-the-spot records of medical conventions, and master lecture tapes from the nation's seventy-nine medical schools. These and other recordings in several languages are being sold or rented to physicians on a nonprofit basis.

The armed forces have approved a pilot plan using this medium in both the United States and overseas medical-military installations.

This investment in medicine may become a necessity to individual physicians or medical groups in smaller communities and could raise havoc with the present mass educational programs in the larger medical centers. Would you prefer hearing Paul Dudley White of Boston talk on "Rheumatic Heart Disease," or Joe Doke of your city?

EXERTION AND ANGINA

Precordial pain is variously and often confusingly described by the patient. In making a diagnosis, a physician requires detailed information as to the character, distribution, and factors related to the pain, in the absence of objective signs. Exertional pain may be described as a dull ache, a burning, or as an intense pain sensation. It is usually substernal and may affect either arm or both. Such pain is attributable to some exertion that adds to the workload on the heart. The amount of exertion that causes discomfort to the patient is modified by many factors that sometimes seem extraneous, such as the weather, the season, time of day, whether the activity is pleasurable, and the patient's excitement or lack of it. Differentiation of heart pain is important to the diagnosis and to the subsequent instruction, care, and protection of the patient.—Bond, G. S.: Differentiation of precordial pain. Postgrad. Med., 14:233 (Sept.) 1953.

The Dean's Page

FAMILY DOCTORS' DAYS

Last spring we initiated a series of Family Doctors' Days at the University Hospitals. Members of the Medical School Faculty had long sought a means whereby they could meet more or less regularly with physicians in active practice in the state in order to discuss problems of mutual interest. The Family Doctors' Day idea seemed admirably suited for this purpose.

To date Family Doctors' Days have been presented by the following departments or divisions: Medicine, Surgery, Psychiatry, Physical Medicine and Rehabilitation, Obstetrics and Gynecology. and Radiology. Attendance has been variable, but in general the response has been very favorable. Many physicians who have attended one or more sessions have indicated that they enjoyed meeting with the faculty in this informal way. Faculty members in turn have been warmly enthusiastic.

After almost a year's experience, our ideas about the Family Doctors' Days have crystallized somewhat. We are most anxious to continue them, and we plan to present one each month throughout the year with the possible exception of July and August. Although in most instances the host department or division will present a program dealing with some topic of general medical interest, we do not look upon these sessions as *primarily* educational in nature. Rather, we look upon them as welcome opportunities for meeting the men who practice in our state and for exchanging viewpoints, ideas, and aspirations with them. We do not believe that the Family Doctors' Days in any way supplant the Continuation Medical Education program. Naturally we also hope that no physician will feel that, because of this program, he is less welcome at the many other Medical School activities such as special lectures, conferences, seminars, and ward rounds, all of which are always open to interested doctors.

Advance announcements of the Family Doctors' Days are now carried in MINNESOTA MEDICINE and other publications seen regularly by Minnesota physicians. In most instances individual announcements are also sent to members of the Minnesota State Medical Association. These announcements frequently accompany announcements of medical continuation courses. Since Family Doctors' Days are usually held just after a continuation course has ended or just before one is to begin, we hope that many physicians will find it convenient and profitable to take in both events during their stay in the Twin Cities.

We are naturally pleased when a particular Family Doctors' Day attracts a large number of physicians. On the other hand, if only fifteen or twenty attend, we by no means consider the session disappointing since the smaller group gives us an even better opportunity to become personally acquainted with those participating.

In short we have been very pleased with the Family Doctors' Days and the way in which they have been received. I would like to extend again a most cordial invitation to all physicians to attend as many Family Doctors' Days as possible. I would also welcome any suggestions as to how these sessions could be improved or made more effective.

Harold S. Diehl, M.D.
Dean of the Medical Sciences

Medical Economics

Edited by the Committee on Medical Economics
of the
Minnesota State Medical Association
George Earl, M.D., Chairman

ECONOMIST PREDICTS GOOD YEAR

Mr. O. F. Litterer, business economist of the Federal Reserve Bank in Minneapolis, predicted a leveling of the present temporary recession which would develop into a good business year.

Speaking at the annual County Officers' meeting of the Minnesota State Medical Association in St. Paul on February 13, Mr. Litterer gave several reasons for refuting the pessimistic attitude on business conditions expressed by some. One of the important factors in this area is the farm income situation. "In spite of the problems of surpluses," Mr. Litterer said, "the U. S. Department of Agriculture has characterized the agricultural outlook for 1954 as regaining stability."

Also, he pointed out, prices of farm products have been stabilized, so have farmers' costs. Consequently, farmers' income in 1954 should be close to the 1953 level. Because farmers' debts are low in relation to their total assets, farmers are still in a strong financial position.

Speaking of the recent increase in unemployment in this area, Mr. Litterer explained that much of it is due to seasonal variations; many industries always reduce their labor forces during the winter. In addition, he said, a few industries not of a seasonal nature have reduced the number of their employes due to accumulation of heavy inventories, and they must stop production and put inventory products on the market.

In the housing situation, the contracts awarded last fall in this district were running well ahead of the previous year. The market is holding up much better than expected.

Stabilizers Noted

Because of the absence of many economic stabilizers in the economy during the last depression, the economic structure was very seriously upset. However, Mr. Litterer emphasized, "stabilizers have been built into the economy since the Great Depression of the early thirties." These stabilizers arise out of the large federal budget, the nature of our tax system, the social security program, and farm price support programs, he continued, but he pointed out that he was not passing judgment on the wisdom of these government programs.

He enumerated the following stabilizers in the monetary and credit system:

1. Bank deposits are insured. Bank failures in the 20's and 30's, not bolstered by federal deposit insurance, added greatly to the severity of the depression.

2. Commercial banks are in excellent condition.

3. Federal Reserve banks have been given additional powers to cope with emergencies. They may extend credit to member banks by accepting any collateral which is satisfactory to Federal Reserve officials.

4. Changes have been made in the field of mortgage lending. The typical mortgage loan now has a maturity of 15 to 25 years, as compared with the five to seven years typical in the twenties.

THREE-DIMENSIONAL PR, GOAL FOR 1954

Three dimensions of medical public relations have recently been described by the Public Relations department of the American Medical Association. They are policy, program and promotion.

According to the PR department, to add "three-D" to medical public relations, every physician must become increasingly aware of the importance of public relations—not only in medical society activities, but in his individual practice.

Three goals have been set up to accomplish the best three-D public relations:

"Goal One—Concentrate on spreading the public relations concept more widely throughout the medical profession.

"Goal Two—Develop a stronger, more unified profession.

1. Plan society programs for new members.
2. Revitalize all medical societies to increase society participation.
3. Present a united front on medical issues, supporting the stand of organized medicine on campaigns of national scope.
4. Increase society and individual community participation; sponsor good citizenship and get-out-the-vote campaigns. Remember—1954 is an important congressional election year.
5. Work with medical students, interns and residents, aiding Student AMA chapters, making available material on socio-economic subjects and public relations, and sponsoring PR courses.
6. Reduce the evils of professional jealousy and unwarranted criticism of one M.D. by another. Such actions harm all doctors in the eyes of the public.

"Goal Three—Utilize every PR outlet to tell medicine's positive story.

1. Present radio and TV programs, using AMA transcriptions or films or developing local shows.
2. Sponsor health forums in co-operation with newspapers.
3. Continue to develop good press co-operation and when possible suggest ideas for local features on medical subjects.
4. Reactivate your society's speakers bureau.
5. Prepare leaflets describing local medical facilities for newcomers.
6. Place medical exhibits at fairs and public gatherings.
7. Capitalize on the PR potential of the Woman's Auxiliary in carrying through society projects.

"Goal Four—Mend PR fences to eliminate complaints about medicine.

1. Set up mediation committees with 'teeth' to actually discipline those few unethical MDs who damage the reputation of the whole profession. Publicize these committees.
2. Make sure your community has a working, well-publicized night and emergency telephone call system.
3. Encourage establishment of medical audits and tissue committees in hospitals to combat widespread publicity regarding ghost surgery and fee splitting.
4. Promote voluntary health insurance and develop closer liaison with such insurance programs; reduce physician abuses of such plans.
5. Offer through your society a guarantee of medical care for all, regardless of ability to pay, and be prepared to make good on that guarantee when necessary.
6. Set up training programs or work with organized medical assistants' groups to teach the importance of medical PR.
7. Encourage greater use of AMA plaques to stimulate fee discussions in the doctor's office and thus reduce fee misunderstandings."

NEW PENSION BOOKLET

The Bureau of Medical Economic Research of the American Medical Association has just issued a new booklet entitled "Prepaid Pensions for All." The booklet is a result of recent questions raised by physicians on the differences between the Jenkins (Reed)-Keogh bills before Congress and other proposals to force self-employed physicians and others under the Old Age and Survivors Insurance tax and benefit provisions of the Social Security Act.

Frank Dickinson, Ph.D., director of the bureau, provides factual material on present prepaid pension plans and the proposed Jenkins-Keogh bills which would allow the self-employed to arrange their own pensions. At the present time, there are two Congressional acts providing retirement income for millions of persons. These are Title II of the Social Security Act of 1935 (Old Age and Survivors Insurance), and Section 165(a) of the 1942 Federal Internal Revenue Code.

How It Works

Explaining briefly how the OASI retirement fund works, the pamphlet states:

> "Employes in covered employment contribute a small percentage of their earnings up to $300 a month (at present, 2 per cent) to the OASI Trust Fund; this amount is matched by the employer. On the same earnings, covered self-employed workers contribute an amount equal to three-fourths the combined tax of the employer and employe."

And, according to Dr. Dickinson, this type of plan is not yet strictly prepaid. He states: "Beneficiaries *now* on the OASI rolls have prepaid (with their employers) only one twenty-fourth of the costs of the benefits they are receiving—scarcely enough to cover the administrative costs. (The source of the 1 to 24 ratio is the report of the Curtis Subcommittee on Social Security of the House Committee on Ways and Means.)"

In Section 165(a) of the Federal Internal Revenue Code, the employer pays for the pension in a noncontributory plan and both the employer and employe pay in the contributory plan. These are completely prepaid.

However, the pamphlet asks this question, "Who bears the burden of employer contributions to OASI and/or Section 165(a) plans?" And the pamphlet answers its own question:

> "The contributions, as a part of employers' costs, are shifted to the consumer in the form of higher prices. The consumer paying for these benefits may be one of the 11 million self-employed who do not benefit under either category or one of the 30 million pensionless employed who do not benefit from a tax-exempt pension plan."

A More Equitable Plan

The pamphlet states that the Jenkins-Keogh, and identical bills, are the answer to making this pension problem more equitable. It also points out that these bills, H.R. 10 and 11, do not amend the Social Security Act, but would amend the internal revenue law. "Hence it should not be confused with proposed changes in the Social Security Act," the pamphlet emphasizes.

How It Would Work

H.R. 10 and 11 would work thusly: Self-employed and pensionless employed persons would be permitted to set aside, tax-free, a limited portion of their earned incomes into individual retirement trust funds; taxes will be paid upon the withdrawal (usually as pensions) of these funds. The result would be tax deferment similar to that on employers' contributions to tax-exempt plans approved by the Bureau of Internal Revenue under Section 165(a). Ten per cent of earned income (but not more than $7,500) may be excluded annually from taxation under this plan. Furthermore, a lifetime limit of $150,000 would, in effect, prevent financing a pension of more than $1,000 a month starting at age seventy. The accumulated money may be withdrawn from the restricted trust fund in a lump sum or in installments, and all withdrawals are taxable income.

In summary, the pamphlet states: "Repeal of Section 165(a) as a method of obtaining equity for the self-employed and the pensionless employed would be a cruel and nonprogressive step to destroy this rapidly growing plan for old-age protection. The only reasonable solution is to provide the self-employed and pensionless employed the same tax-deferment advantages and privileges as are now enjoyed by those covered by pension plans under Section 165(a)."

> "H. R. 10 and 11, the 'Individual Retirement Act,' will provide the means to restore equity; *prepaid* financial security for old age would then be possible for all Americans. A generation after this pending legislation is enacted, much of the need for non-prepaid pensions for an aging population will have been met by prepaid pensions. This is what OASI was supposed to do, but has not. Perhaps it never could.
>
> "For it may be inherently impossible for a government operated program to achieve prepaid pensions for all. The wiser program may be for the government to provide the maximum encouragement for private pension plans, both individual and group."

DOCTOR BILLS MADE LESS PAINFUL

There is a doctor in Portland, Oregon, who has initiated a clever technique to smooth out the rough kinks in his relationships with his patients. More than a year ago this general practitioner began sending a warm, chatty letter with the bills he mailed to his patients. The idea clicked immediately. He now mimeographs and mails more than 400 "newsletters" each month, not only to patients who owe him money but also to many who do not.

The text of the beginning of the first letter is as follows: "Dear Friends: The thought has occurred to me that opening a stack of bills around the first of the month can be pretty unpleasant. . . . This, then, is my attempt to make one of the most unpleasant parts of medical practice a little less so.

"My plan is to enclose a letter like this with each bill. In these letters I'll try to keep you informed about medical matters that I think you'll be interested in. . . ."

The doctor's letters discuss everything under the sun: immunization, polio, obesity, emergency operations, appointments, parking at the doctor's office, diet, toddlers, big kids and adults, baby and child care, how to stop worrying, hygiene, colds and nose drops, chickenpox, measles and politics.

This type of public relations is one of the most valuable of all for the medical profession. It handles people like they wish to be handled—with a human touch. Its value is especially important because it is practicing good public relations at the right level: the doctor's office.

Minneapolis Surgical Society

Meeting of November 5, 1953

The President, Robert J. Tenner, M.D.

GASTRIC SARCOMA

HAROLD G. BENJAMIN, M.D.
Minneapolis, Minnesota

EWING defines sarcoma as a malignant tumor composed of cells of the connective tissue type. We are inclined to think of this tumor as being a rare lesion. However, a survey of recent literature indicates that sarcomas may comprise 3 per cent of all malignant gastric neoplasms, and therefore must be considered in the differential diagnosis of tumors arising in the stomach. We shall never know how many sarcomas have been returned from the operating room with the mistaken diagnosis of "inoperable carcinoma."

The duration of life following the discovery of a gastric sarcoma may be quite long, even though a complete cure is not obtained. Their response to treatment is favorable and the importance of their recognition should be emphasized.

A uniform classification for sarcomas has not yet been adopted and this fact leads to some confusion in the discussion of these tumors. Perhaps the most satisfactory classification is a modification of Ewing's.

I. *Spindle Cell Sarcoma*
  (a) Leiomyosarcoma
  (b) Fibrosarcoma
  (c) Neurofibrosarcoma

II. *Lymphosarcoma*
  (a) Reticulum cell sarcoma
  (b) Malignant lymphocytoma (small round cell sarcoma, lymphocytic type lymphosarcoma).

Lymphosarcomas account for approximately 60 per cent of all gastric sarcomas.

From the surgeon's point of view, gastric sarcoma is a localized tumor and should be resected. The average survival period following surgery for gastric sarcoma is much longer than that following surgery for gastric carcinoma. During the years 1945 to 1952, there were ninety-three cases of gastric maligancy admitted to Northwestern Hospital in Minneapolis. Of these, eighty-eight were carcinoma, and five were sarcoma.

I would like to report in some detail a case of sarcoma which came under my care. Like nearly all other sarcomas reported, the diagnosis in this case was not made pre-operatively, but there are certain features of the history and examination which sometimes make this diagnosis possible.

Case Report

This patient was a woman, aged fifty-four, who was first seen in the early part of June, 1949. At that time, she stated she had noticed a vague epigastric pain with occasional nausea and vomiting during the previous year. For one week prior to admission, the pains had become quite severe, and she had noticed the stools were black. She was nauseated most of the time and could retain only milk. On one occasion, she vomited a little blood. Food did not relieve the pain.

Physical examination revealed a white woman 5 feet 5½ inches tall, weighing 134 pounds. She was thin, nervous, and appeared to be weak and rather chronically ill. Her blood pressure was 116 systolic and 74 diastolic. There was no abnormality of the head, neck, or extremities. Her pulse was 76 per minute and regular. The heart and lungs were normal. There was a slight tenderness to deep palpation in the epigastrium, but no masses were palpable and the liver and spleen were not enlarged. The rectal and pelvic examinations were negative.

Laboratory: X-ray examination at that time revealed an area of infiltration on the lesser curvature of the stomach suggestive of an inflammatory lesion rather than a malignancy. Gastric analysis showed free acid 73°, total acid 85°. Urinalysis showed a specific gravity of 1.014, albumin 0, sugar 0, and microscopic negative. Her hemoglobin was 13.5 grams, with a white blood cell count of 5,400, and a normal differential. The Wassermann test was negative.

Treatment: She was placed on a modified Sippy diet, rest, and antacids. Radiographs taken on June 30, 1949, showed a small ulcer crater on the lesser curvature of the stomach about 2 inches above the pylorus with a moderate amount of induration about it. At that time the diagnosis was gastric ulcer.

Medical management failed to result in any substantial reduction in pain or discomfort. The patient eventually consented to a subtotal gastric resection and she was operated upon on October 27, 1949. At the time of the

GASTRIC MALIGNANCY
Northwestern Hospital, Minneapolis
1945—1952

Age	*Carcinoma*	*Sarcoma*
0–20	0	0
21–30	0	0
31–40	2	1
41–50	4	1
51–60	18	1
61–70	31	1
71–80	27	0
81–90	6	1
	88	5

operation, a small area of induration 2 cms. in diameter was found on the lesser curvature of the stomach 2 inches above the pylorus. The gross appearance seemed to confirm the diagnosis of peptic ulcer. Approximately two-thirds of the distal end of the stomach was resected, using a Hoffmeister modification of the Polya type of repair and a retro-colic anastomosis to the jejunum. One small lymph gland was found along the lesser curvature of the stomach at the time of its removal.

Pathology: The macroscopic specimen showed the distal portion of a stomach, measuring 12.5 cm. along the lesser curvature and 17 cm. along the greater curvature. At a point about 4 cm. above the distal end of the specimen on the lesser curvature there was the edge of a tan, granular patch 3.5 x 3 cm. The mucosa was slightly thickened and indurated in this area. It had a grey-yellow color and appeared slightly granular. Near the center of this patch there was a sharply limited ulcer, 7 x 6 mm. and 2 mm. deep. The wall of the stomach was of average thickness and consistency.

Sections from the pyloric portion of the stomach show that the mucosa was moderately infiltrated by lymphocytes and plasma cells. Sections made at the junction of the tan granular area and the apparently normal antral mucosa, showed an increasingly intense lymphocytic infiltrate. The ulcer has a necrotic floor with many neutrophiles. Sections from the center of the tan patch show the mucosa to be replaced by medium-sized lymphoid cells with a few mitoses and a moderate number of eosinophiles. The mesenteric node showed only benign hyperplasia.

The diagnosis was early lymphosarcoma of the stomach with ulceration.

The patient's postoperative course was uneventful. She has continued to be in good health except for a susceptibility to colds and viral infections. Fluoroscopic and film examinations made two years after operation, on September 26, 1951, showed the esophagus to be normal. A partial gastric resection was noted with a normally functioning anastomosis. There was no evidence of an ulcer or other abnormality.

Because of the small size of the lesion and the fact that it was an early lymphosarcoma with no evidence of metastasis or local extension, no radiation was given postoperatively. It is now four years since the operation and the patient remains in good general health.

Ewing ventured the opinion that lymphosarcoma may begin as an inflammatory process. It is interesting to note that since he made this observation, nothing has been suggested to relate peptic ulcers and sarcomas. It is generally believed that ulceration of the lesion is due to necrosis within the tumor itself.

The clinical and laboratory findings should be studied carefully since the possibility of arriving at a diagnosis of sarcoma of the stomach before operation resides in these features:

1. Epigastric pain is nearly always the major complaint.
2. Hemorrhage is frequent; more often melena than hematemesis.
3. The general condition of the patient is often comparatively good since the lesion usually localizes away from the gastric orifices and obstruction does not develop early.
4. The average age is forty-five, while for cancer it is fifty-five or sixty.
5. The x-ray findings of a filling defect with smooth margins, or of diffuse types with rigidty of the walls and infiltration, plus the presence of giant rugae; or of the presence of two or more defects in the mucosa; are all suggestive of gastric sarcoma.
6. It may be possible to procure a specimen for biopsy by esophagoscopic examination, but frequently visualization alone cannot differentiate the lesions.
7. The absence of free acid in the stomach is significant in its relationship to the presence of sarcoma and carcinoma alike.

Sarcoma occurs in a younger average age group than does carcinoma. In a series of sarcomas reported Pack, the average age was forty-five years. In Broder's series, the average age was fifty years; and in Walter's series, the average age was 43.7 years. The youngest case of gastric sarcoma seen at the Mayo Clinic was ten years and the oldest was sixty-eight. In this series of gastric sarcomas, the extremes were thirty-seven to eighty-three years of age.

The average duration of symptoms in forty-one cases of gastric sarcoma seen at the Lahey Clinic between 1929 and 1949 was ten months, with extremes of from three weeks to four years. These cases were all of the sarcomas seen in 1,171 operations for malignant tumors of the stomach during that interval.

It is generally believed that sarcomas metastasize by way of the blood stream with the exception of lymphosarcomas, which may spread by direct extension or by the lymphatics.

In most cases reported, the tumor could not be diagnosed by the gross specimen as to whether it was carcinoma or sarcoma. It is usually necessary to examine the tissue microscopically in order to establish the diagnosis. This accounts for the difficulty in x-ray or endoscopic diagnosis.

Grossly, the lesion may be divided into four types as suggested by Pack and McNeer:

1. Single bulky polypoid growths well demarcated from the stomach.
2. Solitary or multiple ulcers with surrounding infiltration.
3. Multiple nodular tumors of the stomach.
4. Diffuse thickening of the wall of the stomach.

The lesion in this discussion falls under Group 2.

These tumors may grow very rapidly or at other times exceedingly slowly. The lymphosarcomas generally pursue a more rapid growth. They arise in the submucous layers of the stomach and infiltrate the surrounding layers. Probably their proximity to the submucous plexus of nerves and their early infiltration into the muscle layers of the stomach accounts for the prominent and early symptom of pain.

At operation, the lymphosarcomatous mass, if large, is usually firm and may be necrotic, friable, nodular and rubbery. If secondary infection sets in, chills and fever may be a symptom. Metastases to the regional lymph nodes are frequent.

Treatment

Therapy falls under the two main headings of surgery and radiation. Subtotal gastrectomy is accepted as the treatment of choice for all but the most advanced cases. Probably more frozen sections should be done at the time of operation, where grossly malignant lesions of the stomach are encountered. This is particularly true when a sarcoma is suspected. If the lesion proves to be a sarcoma, the surgeon should make every effort to resect the stomach. Some lesions appearing to be an inoperable carcinoma, have been found to be sarcomas quite amenable to resection.

McSwain and Beal (Ann. Surg., 1944) state that "roentgenotherapy should be given in every case of lymphosarcoma of the gastrointestinal tract unless the operator and the pathologist are certain that the lesion has been completely eradicated." A combination of the two is most frequently effective. In the case reported, it was felt that all of the lesion was removed because of its early stage and absence of demonstrable metastases.

Sarcomas are far more sensitive to deep x-ray radiation than carcinomas. In diffusely infiltrative, non-ulcerated types, as in the aged and debilitated, or in already metastasized lesions, the case may more rightly be considered one for radiation therapy than for surgical intervention. Postoperative radiation should be started as soon as the wound is healed.

The surgeon who encounters a large infiltrating mass and is inclined not to resect, must consider the possibility that the lesion may be a sarcoma. As long as this tumor cannot be excluded, the patient should be given a chance for the more favorable prognosis which a resected and radiated sarcoma carries.

Conclusion

In conclusion, then, it has been my purpose to emphasize that sarcomas must be considered in the differential diagnosis of gastric tumors. A uniform terminology for their description is needed. Their consistent symptoms of severe pain, and hemorrhage, in a relatively slightly cachectic individual in the younger age group, plus the suggestive x-ray and gastroscopic appearance, should make a pre-operative diagnosis more common. Gastric resection plus x-ray radiation offers a very good chance of long palliation and occasionally a cure.

The case report of another gastric lymphosarcoma has been added to the literature on this subject.

Bibliography

1. Bassler, A., et al: Distinctions between gastric sarcoma and carcinoma. J.A.M.A., 138:489-494 (Oct. 16) 1948.
2. Berman, Harry: Lymphosarcoma of the stomach. Med. Record, 160:30-32, 1947.
3. Cash, I. I., et al: Reticulum cell sarcoma of the stomach. Gastroenterol., 6:40-49 (Jan.) 1946.
4. Dixon, C. F., et al: Differential diagnosis of sarcoma of the stomach. S. Clin. North America, 29:1109-1113, 1949.
5. Greathead, E.: Leiomyosarcoma of the stomach. Brit. M. J., 2:422-423 (Aug. 20) 1949.
6. Kersner, D.: Leiomyosarcoma of the stomach with hepatic metastasis; radical resection with surgical recovery. Gastroenterol., 18:456-462 (July) 1951.

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History of Medicine In Minnesota

MEDICAL HISTORY OF THE COUNTY OF YELLOW MEDICINE

MILDRED B. LEE
Granite Falls, Minnesota

(Continued from January, 1954, issue)

Alonzo T. Munro was born at New Auburn, Minnesota, on September 12, 1875, the third son in the family. His parents were Scotch, and had come to the United States soon after their marriage. He received his early education in the schools of New Auburn and Glencoe, Minnesota, then entered the University of Minnesota for his medical training. He graduated with his degree in medicine in 1897, and went to Hanley Falls to begin his practice.

Dr. Munro practiced at Hanley Falls and Wood Lake for three years. In 1901 he married Miss Maud A. Force of Madison, a teacher who had been on the staff of the Hanley Falls schools, and also had taught in Echo. In the following year he left Yellow Medicine County for Kalispell, Montana, where he took the position of assistant physician of the Great Northern Railroad, for the division at Kalispell. After a few years there he went to New York for special training in the diseases of eye, ear, nose and throat, and after qualifying as a specialist in this field he returned to Kalispell where he resumed his practice, and continued to serve the railroad in that capacity.

The region around Kalispell, Montana, was sparsely settled and greatly in need of doctors when Dr. Munro located there. He was one of the pioneer doctors of that area. He was a member of the American Medical Association, and at one time was on the Montana State Board of Medical Examiners. He was also a member of the Montana State Medical Association, and was instrumental in organizing the Flathead Medical Society in 1904. He was a member of the Montana Soldiers' Home Board, and a charter member of the state's Fifty Year Club, which honored those who had served the medical profession for that length of time. For many years he was on the staff of the Kalispell General Hospital, and also served as County Health officer.

During the period of his residence in Yellow Medicine County Dr. Munro was a candidate for coroner of Yellow Medicine County, and in the *Canby News* he was described as follows:

> "Dr. Munro is of Scotch origin, and possesses all the manly, upright qualities of that sturdy race. He was born on a farm near New Auburn, Sibley County, Minnesota, Sept. 12, 1875, attended the public schools until 16 years of age, when he entered Stevens Seminary, Glencoe, from which he graduated three years later. He then took up the study of medicine at the University of Minnesota, graduating in 1897, since which time he has practiced the healing art continuously, the last six months at Hanley Falls. If you cast your ballot for Dr. Munro you have voted for an honest man, a clean man, a capable man and a man who refuses to be a hypocrite 'e'en tho the heavens fall.' He should be elected almost unanimously."

Dr. Munro continued in active practice until his death on December 18, 1951, of congestive heart failure.

He was survived by his wife, Maud A. Munro, and three daughters: Mrs. Phil U. Kington, Mrs. Maurice S. Fisher, both of Kalispell, and Mrs. O. A. Bessey of Galveston, Texas.

Dr. Munro was licensed in Minnesota (No. 824) on June 10, 1897. The state medical records indicate that Dr. Munro was in Wood Lake in 1903 and 1905, but the date of his removal from the county is given by his family as 1902, at which time he was practicing in Wood Lake.

Dr. George W. Nichols graduated from the Vermont Academy of Medicine (Castleton Medical College) in 1861, and in the summer of 1871 came to Minnesota where he located in Rochester. His first office there was in his home on the corner of Fourth and Main Streets. Later he bought a residence on Prospect and Fourth Streets and opened an office on Broadway.

While in Olmsted County Dr. Nichols became a member of the Olmsted County Medical Society and of the Minnesota State Medical Society. In 1874 he became coroner of that county and also county physician. According to the Olmsted County Medical History Dr. Nichols was remembered as "a nice man, a good doctor, an all around good fellow," and had an extensive practice. He was married in 1864, and had a daughter, Edith, and one son.

Dr. Nichols also was a member of the city board of health of Rochester, and of the Independent Order of Odd Fellows, and Ashlar Lodge No. 23 of the Ancient Order of United Workmen.

In 1879 Dr. Nichols bought a farm in South Dakota, then sold his Rochester home and removed to Wahpeton, South Dakota. In 1883 he moved from Wahpeton to Minneapolis where his daughter, seven years old, died. A few months later he moved to Belle Plaine, and in December, 1883 to Saint Paul. From 1887 to 1891 he was in Merriam Park; from 1892 to 1895 in Wood Lake, Yellow Medicine County.

Dr. Nichols' first professional notice appeared in the *Granite Falls Tribune* of August 4, 1891, but it is apparent that he had then been established for some time in Wood Lake. The first mention of him is in the July 14 issue: "Dr. Nichols invited us to visit a patient with him, and we can vouch for the rapid transit to and from." From this it may be inferred that he was still equipped with the spirited horse and fine buggy described in the Olmsted County History as:

> "A Dexter side-spring phaeton with a roomy top; a suitable box beneath the seat will accommodate the side curtains and the useful rubber lap blanket. On the dash, directly in the center is firmly fixed a physician's lamp with a powerful reflector of an improved design, calculated as a light unto the doctor as he meanders over the countless mudholes. The carriage is well built, and judging from the short whirl we took in it behind the doctor's fiery horse, Dan, it is as easy riding a vehicle as we have seen in many a day."

Dr. Nichols had received his state license, No. 646 (r) in 1883, and was a member of the state Medical Society. He may also have been a member of the Camp Release Branch of the State Medical Society while in Yellow Medicine County, but all early records of this organization have been lost and such membership cannot be established.

Dr. Nichols had an office in Granite Falls over Nelson and Johnson's Drug Store, and since he practiced in both Wood Lake and Granite Falls he spent a part of the week at the Sullivan House, one of the early hotels of Granite Falls. The family at Wood Lake is mentioned only when "Dr. Nichols' little girl" had measles.

In 1896 Dr. Nichols left Yellow Medicine County to locate in Mille Lacs County. From there he went to Hinckley, Pine County. Still later he moved to California, where he died in September, 1908.

Dr. Martin A. Nelson graduated from the Physio-Medical College of Indiana, Indianapolis, Indiana, in 1894, and was licensed in Minnesota April 6, 1894, License No. 410. He seems to have located in Clarkfield shortly after that date. Medical directories place him in Clarkfield in 1894 and 1896; in 1898 he was in Fertile, Iowa, where he remained for several years.

In 1909 he was in Minneapolis, and did not register with the State Board until 1928.

Dr. Tord Nielsen (also known as Ford Nielsen) was born in Norway on April 25, 1857. He received his early education in that country, then entered the Royal University of Norway (also called the University of King Frederick) and was graduated with a medical degree in 1881. He then came to the United States and was licensed to practice medicine in Minnesota October 11, 1883 (License No. 11). In 1885 he was licensed in North Dakota, and may have practiced in that state for a number of years. Medical records place him in Minneapolis in 1895, but this seems to be an error, for Dr. Nielsen came to Canby in 1894, from Minneapolis.

Dr. Nielsen's first professional card appears in the *Canby News* of March 30, 1894; "Dr. Tord Nielsen, Graduate of Royal University of Norway, Office over the Citizen's State Bank of Canby."

Dr. Nielsen's reputation had preceded him to Canby, for a local man had been successfully treated by him for tuberculosis. It appears that Dr. Nielsen had built up some reputation for himself in Minneapolis because of his success in treating this disease, for the Canby man, who had tuberculosis in an advanced stage, heard of him quite by accident, from a stranger in a railroad station. The treatment prescribed by Dr. Nielsen for this disease is said to have been both severe and dangerous and was called the "lymph treatment," based upon methods and medicines used in Norway. It brought on a period of serious illness which was followed, in this case, by complete recovery and the patient* lived in good health to great age.

Dr. Nielsen was very well educated and considered a good doctor. His success with the treatment of tuberculosis did not, however, continue; for the reason (it was said) that the necessary medicines could not be secured. Although Dr. Nielsen's prospects for building up a practice were very good, it may be that this failure weighed upon him, for he became moody and began to drink to excess, so that his practice and reputation suffered.

Dr. Nielsen's family consisted of his wife, whose former home had been at Fergus Falls, Minnesota, and two children. The boy, Thorulf, came with them to Canby, and there was a girl born in the summer of 1894. After about a year in Canby Mrs. Nielsen returned to her former home with the children, and Dr. Nielsen went to Minot, North Dakota. In 1903 he was, according to medical records, in Kenmare, North Dakota, and also in 1905. He was later reported in Grafton and Edmore, North Dakota. In 1909 his address was not known.

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*The Yellow Medicine County man referred to was a brother of Mrs. Ole O. Ostensoe, of Canby (widow of Ole O. Ostensoe, attorney, deceased) who related this incident in considerably more detail. She was an intimate friend of both Dr. Nielsen and his wife.

Nicolay Hilmar Scheldrup (Schjelderup) was born in Tovik, Norway, on September 1, 1873, the son of Andrew Martin and Hanna (Hanvig) Schjelderup. The following contemporary biography is taken from a *History of Central and Northern Minnesota* published in 1904:

> "Dr. N. H. Schelderup, who is an exceedingly well finished and widely educated physician of Granite Falls, Minnesota, belongs to the younger generation of physicians who approach the profession not only with bright and gifted minds but keenly polished and thoroughly educated with the best schooling the times afford. Dr. Schjeldrup was born in Tovik, Norway, in 1872 and his father who was a man of much prominence in his day held a government position in Norway.
>
> Dr. Schelderup was the oldest of a family of children and was reared to manhood in Norway, where he graduated from the University of Christiania, one of the great and famous schools of the old world. This was in 1891, and he immediately came to the United States. Some years were occupied by him in both general and medical study, much of the time being under the special direction of Dr. Fenger, a great operative surgeon of this country. The young student for medical knowledge graduated after a thorough preparation in 1897 and at once began practice in Granite Falls. After two years of professional work he took a postgraduate course at the University of Berlin. Dr. Schjelderup became interested with Dr. F. H. Hacking in 1902, and the two associated themselves together professionally under the firm name of Schjelderup and Hacking. They have a well appointed suite of offices over the Nelson Drug Store.
>
> Dr. Schjeldrup is a Republican and was elected coroner, in 1902, in which he is serving his first term. He was married in 1897 to Miss Silvia Odegaard. She was born in Chicago, comes of Norwegian blood, and is the mother of two children, a boy and a girl. She is a bright and charming lady and is very popular as is her husband in the social circles of the city.
>
> Dr. Schjelderup is Vice President of the Western State Bank and one of the principal stockholders in the institution."

Dr. Scheldrup's professional card began to appear in the local newspapers in the early summer of 1897. He appealed to the foreign born by announcing himself as "Skandinavisk Laege" in all his professional notices. His first office was in a building "opposite Hotchkiss' store" on Prentice Street.

In appearance Dr. Scheldrup was a slender young man of medium height and smoothfaced, so that he appeared younger than his years. In later life he wore a goatee in deference to the custom of the time. His hair was reddish, his eyes blue, his personality well adapted to the community and the profession which he served.

When Dr. Scheldrup came to Granite Falls the twin daughters of the pioneer merchant, R. H. Hotchkiss, were often on the street in their wheel chairs. The twins had never walked, and their parents had taken the children to many physicians only to be told that they were paralyzed from the waist down and there was no help for them. Their case was a challenge to a young doctor with ambition and not too much to do, and Dr. Scheldrup undertook to make a study of it, with the understanding that if he could not help the little girls there would be no charge for his services. He found that the hip joints were immovable, and after much study and consultation with specialists in Chicago he tried out a new technique for relieving the condition. After the required surgery and treatment the girls began to walk, first with crutches and then with canes, and eventually made a complete recovery. It was no small accomplishment for a new arrival in the field and Dr. Scheldrup's professional reputation in the community was definitely made.

In September, 1897, Dr. Scheldrup was married to Miss Sylvia M. Odegaard of Chicago, at the residence of J. F. Ireland in Granite Falls. Announcement of this wedding in the *Granite Falls Tribune* was followed by the note that Dr. Scheldrup "is a promising young physician who came here about six months ago and in that time secured a large practice and has many friends."

In the January 18, 1898, issue of the *Granite Falls Tribune* Dr. Scheldrup is quoted as saying the greatest need of the town was for a hospital and sanatorium. He set out to supply the need for a hospital himself by purchasing the old Henry Hill homestead in East Granite Falls, and furnishing it with the necessary equipment. A registered nurse, Miss Marie Agee of Chicago, was placed in charge. The hospital was operated by Dr. Scheldrup privately for the use of his own patients, but other doctors were free to use it also.

By 1900, the needs of the community required a larger hospital, and Dr. Scheldrup sold it to Mr. and Mrs. Walter W. Stoppe (Miss Marie Agee) who occupied it as a residence until it was destroyed by fire. A new hospital was established in the Pinney homestead, a large, two-story building which is still in use as a dwelling on Ninth Avenue, just off Prentice street. Here there was room for additional beds and for a laboratory and operating room.

On July 12, 1898, Dr. Scheldrup's first child, a son, named Albert H. Scheldrup, was born. Dr. Scheldrup's practice was steadily increasing, and the family occupied a large home at 2155 Prentice Street (now known as the Olds residence). The four horses necessary to get the doctor around the country were housed in a barn on the river bank back of the house. The driver who was also a necessary adjunct to the doctor's menage was established in a small room off the kitchen where he could be reached conveniently at any time.

The doctor was interested in civic projects and in the general welfare and progress of the community. He found time to take part in promoting local celebrations and other projects.

Dr. Scheldrup's second child, a son, Amos Stephan, was born August 20, 1901, and in 1904 another child, a girl, Sylvia Louise, was born on August 14.

In 1902 Dr. Frank H. Hacking, then practicing at Wood Lake, Minnesota, was taken into partnership, and this arrangement continued until 1906, when Dr. Scheldrup was invited to take over Dr. F. A. Dunsmoor's surgical practice during his absence. Dr. Dunsmoor was also acting as professor in clinical surgery at the University of Minnesota, and this position was also to be taken over by Dr. Scheldrup during the time Dr. Dunsmoor and his family were away on an extended tour of the European countries. This invitation was accepted, and Dr. Scheldrup terminated his connections in Granite Falls to take over the responsibilities of a wider field.

At this time his domestic affairs were also in crisis; Mrs. Scheldrup commenced an action for divorce and then returned to Chicago with her three children.

Upon his return from Europe Dr. Dunsmoor proposed that Dr. Scheldrup continue to be associated with him, and this proposal resulted in a pleasant and harmonious partnership which lasted for six years. But Dr. Scheldrup began to see the tendency of the medical profession to become highly specialized, not only in internal medicine but also in surgery, and he thought the time had come to follow the trend of the profession and establish a clinic. As an older man Dr. Dunsmoor did not feel inclined to accept this idea, so Dr. Scheldrup carried out his plan and established a clinic with which he was actively associated for many years at 1111 Nicollet Avenue, Minneapolis. When the clinic was formed Dr. Hacking was invited to join it, and accepted. Later through the efforts of Dr. Scheldrup, Dr. Hacking was appointed as the chief tubercular expert at the Thomas Tubercular Hospital, affiliated with and owned by the Fairview Hospital.

About the same time Dr. Scheldrup and a group of friends organized the Fairview Hospital, built it, and made it one of the outstanding hospitals in the state.

Throughout his career Dr. Scheldrup kept abreast of medical developments and steadily sought to perfect the techniques, methods, and services of his profession. Until 1932 he was a surgeon at Fairview hospital and engaged in all the activities necessary to promoting the interests of the hospital and keeping up with the demands of his profession. But the pace which he had set for himself told upon his health; a heart ailment curtailed activities and forced him to retire from active participation in 1932.

Since that time Dr. Scheldrup has lived at 185 S.E. Eighth Street, Miami, Florida, during the winter months, and at his summer home at Rainy Lake in northern Minnesota.

During World War I, Dr. Scheldrup served as Captain, Medical Corps, of the U. S. Army, from 1918 to 1919; he was decorated Knight, Order of St. Olaf, first class (Norway); and is a director of Perfection Manufacturing Company. His name appears in International Blue Book and in Who's Who.

His four children, A. S. Scheldrup, Eugene W. Scheldrup, Sylvia Louise, and Robert, are living at this time. Sylvia Louise is the wife of Dr. Harold R. Leland of Minneapolis.

The doctor is a Republican, and a member of the Lutheran Church of which he has been a loyal and generous supporter. Throughout his career he has kept in touch with valued friends in Granite Falls and is given credit for many acts of charity and kindness.

Dr. N. M. Smith, a doctor of the Eclectic School, was located in Granite Falls in 1873. Rose's History of Yellow Medicine County places him as a resident of East Granite Falls; but a map or sketch of the city made in 1874 shows his house in West Granite Falls. Some old residents remember the Dr. Smith house as the one now standing at the intersection of Ninth Avenue with Third Street, and known as the Stoppe residence.

Dr. Smith's professional cards appeared in the *Granite Falls Journal* until about 1878, when he seems to have moved from Granite Falls to locate in Iowa. The few newspaper items about him indicate that he originally came from Iowa, as both his own and his wife's relatives lived in that state. While in Granite Falls two children were born to the N. M. Smiths.

The libel action which Dr. Smith commenced against the editor of the *Granite Rock* is elsewhere reported. The account of the trial in justice court in Minnesota Falls leaves one with the impression that he was not a skilled physician, and certainly the other members of the profession were not in sympathy with him. Two doctors testified for the defense, and nowhere in the *Minnesota Sentinel* edited by Drs. Stratton and Falk is Dr. Smith referred to as Dr. N. M. Smith.

Dr. F. G. Sparling. In 1896 the village of Wood Lake had a population of 100 people, and one physician, Dr. F. G. Sparling.

He is remembered as a tall, slender young man who ministered to the people of the community through epidemics of measles and other diseases. He did not have a family at that time.

Dr. Sparling graduated from Rush Medical College in 1895, and probably began his medical practice in Wood Lake. He practiced about two years in that village.

In 1901 Dr. Sparling was licensed in Nebraska, and in 1904, in Iowa.

He died in Minneapolis on January 5, 1922, of heart disease, at the age of fifty-seven years.

Albert Burbank Sweet was born at Walden, near Montpelier, Vermont, July 14, 1847. He moved west with his parents in 1851. The family traveled on a home-made flatboat going by way of the Great Lakes to Freeport, Illinois, then by covered wagon to Waukon, Iowa, and to Winona County, Minnesota, in 1854. His early education was probably gained in the schools of that region.

At the age of sixteen he answered the call for volunteers to serve in the Civil War, serving with Company F, of the Fourth Minnesota Volunteer Infantry. He was wounded while accompanying General Sherman on his march to the sea.

After the war Dr. Sweet taught school in Brown County, Minnesota, and during the blizzard of 1873 was marooned for two days in the school house with his pupils. He was credited with saving their lives. He then took up the study of pharmacy and became a graduate pharmacist. He went into business as a druggist in Marshall, Lyon County, where, in 1880, he married Fannie Hortense Simpson. In 1884 the Sweets moved to the village of Hanley Falls, then called Hanley, where he established the first drug store in that village. The "City Drug Store" advertised in the *Granite Falls Journal* and prospered; in 1886 a cousin, also a doctor and a druggist, F. B. Sweet, was taken in as a partner.

During this period Dr. Sweet began the study of medicine at the University of Minnesota, while still retaining his interest in the drug business. He graduated from the Medical Departmnt of the State University in 1884, and was licensed to practice in Minnesota on October 27, 1885 (No. 1117)—Basic Science Certificate No. 3178. He was named surgeon for the Minneapolis and St. Louis Railway at Hanley.

Dr. Sweet was progressive and interested in the development of the new settlement. He is reported as trying to interest Saint Paul people in establishing a bank in Hanley; then he enlarged his drug store, advertised as a "manufacturing chemist" and also practiced medicine. Dr. Sweet was very successful as a druggist; as a young practitioner in Hanley Falls he was not so successful.

Dr. Sweet left Hanley about 1888 or 1889 for Kasota, and later practiced in St. Peter for a time. In 1891 he moved to Minneapolis where he practiced medicine exclusively, and in 1892, settled in Hopkins, the first physician to locate in that place. In 1922 he retired from active practice, but continued his activities in the G.A.R. and V.F.W. until his death. Mrs. Sweet died in 1937.

Dr. Sweet died on July 6, 1943, at the Veterans Hospital, after an illness of one day. He was then ninety-five years old. He was a member of the Minnesota State Medical Association, of the G.A.R. and V.F.W. as above noted. His death was due to cerebral thrombosis and arteriosclerosis. He was buried at Lakewood Cemetery with military honors.

(To be Continued)

✦ Reports and Announcements ✦

AMERICAN GERIATRICS SOCIETY

The eleventh Annual Meeting of the American Geriatrics Society will be held at the Fairmont Hotel, San Francisco, preceding the meeting of the American Medical Association. The scientific sessions of the meeting will begin Thursday afternoon, June 17, and continue through Saturday morning, June 19.

Hotel reservations should be made through the San Francisco Convention and Visitors Bureau, 200 Civic Auditorium, San Francisco 2, California. Members should reserve accommodations immediately, stating date and time of arrival and departure, because the hotels expect to be filled to capacity.

The Annual Business Meeting will be held in the Fairmont Hotel, June 17, 9 a.m. The room where this meeting will be held will be announced later. All Scientific Sessions will be held in the Nob Hill Room of the Fairmont, and the annual dinner is scheduled for the Terrace Room on Friday evening, June 18.

The meeting will be open to all members of the American Geriatrics Society and to physicians and other scientists who are interested in the field of geriatrics. The program will cover many aspects of geriatric medicine, and there will be several panel discussions on such subjects as recent developments in cardiology, methods of determining operability in older patients and the relation of industry to geriatrics. Outstanding clinicians and investigators will participate.

Dr. Laurance W. Kinsell, Highland Alameda County Hospital, 2701 Fourteenth Avenue, Oakland 6, California, is in charge of local arrangements for the meeting.

AMERICAN GOITER ASSOCIATION

The American Goiter Association will hold the 1954 meeting at the Somerset Hotel, Boston, Massachusetts, April 29, 30 and May 1, 1954. The program will consist of papers and discussions on the physiology and diseases of the thyroid gland. For further information write John C. McClintock, M.D., Corresponding and Recording Secretary, 149½ Washington Avenue, Albany, New York.

THE CANCER INSTITUTE AT MIAMI, CANCER RESEARCH AND CYTOLOGY CENTER

Dr. Joseph S. Stewart, chairman of the third annual Seminar and Conference of Cancer Cytology to be conducted by the Cancer Institute at Miami, announces that the conference will be held April 21-24, 1954, inclusive.

This year the conference will bring together several leading authorities on cancer from this country and abroad. The last day will be devoted to a special session for medical practitioners, who will visit the Cancer Institute to see demonstrations on the taking and preparation of cytodiagnostic tests for cancer of many types, with special sessions devoted to cancer of the uterus, breast, prostate, lung, and stomach. The latest advances in cancer diagnosis using new cytological methods and blood testing procedures will be presented.

Members of Dr. Stewart's Committee include: Dr. Virgil H. Moon, honorary chairman; Dr. Richard Fleming; Dr. Ralph W. Jack, and Dr. Milton Coplan. The program will be conducted under the direction of Dr. J. Ernest Ayre.

CHEST DISEASE SYMPOSIUM FOR GENERAL PRACTITIONERS IN SARANAC LAKE THIS SUMMER

The third annual Symposium on Tuberculosis and Other Chronic Pulmonary Diseases for General Practitioners will be held in Saranac Lake, New York, July 12 through 16, 1954. It is approved by the American Academy of General Practice for twenty-six hours of formal credit for its members.

The Symposium is sponsored by the American Trudeau Society, the Saranac Lake Medical Society and the Adirondack Counties Chapter of the New York State Academy of General Practice. The registration fee is $40 for A.A.G.P. members and $50 for nonmembers.

The scope of this year's Symposium has been broadened to cover chest diseases other than tuberculosis. Included in the course will be discussions of the diagnosis and treatment of nontuberculous pneumonias, pulmonary cancer, lung abscess, fungus diseases, bronchiectasis, sarcoid, cystic disease, emphysema, and the pneumoconioses.

The speakers and panel members at the Symposium will include thirty-four physicians, surgeons, and scientists from Saranac Lake and surrounding areas. Guest lecturers include Dr. D. Ewan Cameron, professor of psychiatry, McGill University, Montreal, and Dr. Donald Miller, assistant professor of thoracic surgery, University of Vermont Medical School, Burlington, Vermont.

Many physicians who have attended previous symposia have brought their families to Saranac Lake. So that families might have use of the car to enjoy the many recreation and vacation facilities of the Adirondack Mountains, free bus transportation was provided for physicians from Saranac Lake to the various meeting places. This practice will be followed again this year.

These Symposia are the result of many requests, during the last few years, from the General Practitioners for a postgraduate course on chronic pulmonary diseases designed for them and presented over a period short enough so that they might readily attend. The 1954 Symposium has been planned to meet those needs and to cover all important aspects of these diseases from the General Practitioner's point of view.

Many of the sessions are informal panel discussions with ample opportunities for questions from the audience.

The Symposium will be held in various sanatoria and laboratories in the Saranac Lake area. Morning sessions will be from 8:30-12:30 and afternoon sessions from 2:00-4:00 (Monday, Wednesday, and Thursday). There will be elective sessions on Tuesday and Friday afternoon. Physicians desiring to make patient rounds will have that opportunity each afternoon at 4:00. On Monday, July 12, there will be a dinner for physicians attending the course, their families, and faculty.

Complete information concerning this program can be obtained by writing: Richard P. Bellaire, M.D., Chest Disease Symposium, P.O. Box 2, Saranac Lake, New York.

PAN-PACIFIC SURGICAL ASSOCIATION SIXTH CONGRESS

Doctors are cordially invited to attend the Sixth Congress of the Pan-Pacific Surgical Association to be held in Honolulu, October 7-8, 1954, and are urged to make arrangements as soon as possible if they wish to be assured of adequate facilities.

An outstanding scientific program with over 100 leading surgeons, including sessions in all divisions of surgery and related fields, promises to be of interest to all members of the profession. An extensive social program is being developed for the doctors' families.

The Association office has been appointed as travel agent for those attending the Congress and it is important that all hotel and travel reservations be made through the Honolulu headquarters of the Pan-Pacific Surgical Association.

For further information, please write to F. J.. Pinkerton, M.D., Director General, PAN-PACIFIC SURGICAL ASSOCIATION, Suite Seven, Young Building, Honolulu, Hawaii.

INDUSTRIAL HEALTH CONFERENCE

Significant industrial health problems, ranging from increasing incidence of lung cancer to industrial absenteeism, will be discussed in a five-day session at the annual meeting of the Industrial Medical Association, April 26 to 30, at the Sherman Hotel, Chicago.

This large meeting will be attended by more than 2,000 delegates to the 1954 Industrial Health Conference, which also includes gatherings of the American Association of Industrial Nurses, Incorporated, the American Association of Industrial Dentists, American Industrial Hygiene Association and American Conference of Governmental Industrial Hygienists.

After registration on April 26, the meeting of the Industrial Medical Association will open with clinics on April 27. That afternoon, the opening session will feature papers on health in industry, plus a panel discussion.

April 28, the IMA will meet jointly with the American Industrial Hygiene Association to consider "The Responsibility of the Profession in the Health Education of the Employee."

That afternoon, in a joint session with the nurses and dentists, the Industrial Medical Association will discuss causes of absenteeism.

April 29, the Surgical Section will center its discussion around major surgery of the extremities and the relationship of the Industrial Medical Department and Civil Defense. Meeting simultaneously, the Medical Section will hear papers on "Lung Cancer" and "Cardiacs in Industry."

Plant tours will climax the meeting on Friday.

Theme of the 1954 Industrial Health Conference is "Protecting our greatest industrial wealth—employee health." The conference has been described as a "post-graduate course, covering the entire field of industrial health and hygiene, occupational ailments and traumatic surgery."

SCHOLARSHIPS IN PLASTIC AND RECONSTRUCTIVE SURGERY

The Foundation of the American Society of Plastic and Reconstructive Surgery announces the regulations of the Fifth Annual Scholarship Contest:

1. The contest in *Junior* Classification is restricted to residents in training and to plastic surgeons who have been in practice *no longer than five years.*
2. Two main prizes are offered, each consisting of a *three-months' plastic surgery scholarship* with full maintenance in a number of selected leading services, in the United States and abroad.
3. The subject matter of the essay must be the result of some *original* research (either clinical or experimental) in the field of plastic and reconstructive surgery. Thesis should be of about 5,000 words, and written in English.
4. Work previously published wholly or in part is not acceptable.
5. Expenses of travel between the services will be allotted.
6. A silver plaque or a certificate of honorable mention is also offered for a winning essay in *Senior* Classification to contestants active in practice of plastic and reconstructive surgery for *more than five years.*
7. Manuscripts will not be accepted by the Award Committee *after July 1, 1954.*
8. The winning essays will be presented in person at a formal program of the Foundation of the A. S. of P. and R. S. to be held in the Fall of 1954.

The 1953 Scholarships were awarded to the following: Robert M. McCormack, M.D., Rochester, N. Y., for an Essay, "Simultaneous Chordee Repair and Urethral Reconstruction for Hypospadias."

James Calnan, F.R.C.S., Oxford, England, for an Essay, "The Error of Gustav Passavant," Sub-title: "A Refutation of the Recognized Velo-Pharyngeal Mechanism in Speech."

For further information, write to the Award Committee, c/o Jacques W. Maliniac, M.D., Chairman, 30 Central Park South, New York, N. Y.

FAMILY DOCTORS' DAYS

University of Minnesota Hospitals

All physicians are cordially invited to attend the following Family Doctors' Days which will be presented under the direction of the department indicated.

March 10—Dermatology
April 7—Pediatrics
May 5—Hospital Laboratories
June 9—Pathology

Activities begin with luncheon at 12:00 noon with the staff of the department in the Hospital Dining Room and will continue throughout the afternoon.

For further information, write the head of the department concerned or the Director, Department of Continuation Medical Education, 3330 Powell Hall, University of Minnesota.

REGIONAL MEETING OF THE NATIONAL GASTROENTEROLOGICAL ASSOCIATION

A regional meeting of the Central Region of the National Gastroenterological Association will be held in Milwaukee, Wisconsin, March 28. 1954. The scientific sessions will be held at the Hotel Schroeder at 2:00 p.m., following the semi-annual meeting of the Association's National Council.

Participating in the meeting will be Dr. J. Arnold Bargen, Rochester, Minn.; Dr. H. B. Benjamin, Milwaukee, Wisc.; Dr. Carl W. Eberbach, Milwaukee, Wisc.; Dr. James A. Ferguson, Grand Rapids, Mich.; Dr. Robert McCarty, Milwaukee, Wisc.; Dr. Karver L. Puestow, Milwaukee, Wisc.; Dr. James Smith, Milwaukee, Wisc.; Dr. Gerhard Strauss, Milwaukee, Wisc.; Dr. Philip Thorek, Chicago, Ill. and Dr. Marvin Wagner, Milwaukee, Wisc.

Dr. Sigurd W. Johnsen, Passaic, N. J., president, and Dr. Lynn A. Ferguson, Grand Rapids, Mich., president-elect of the National Gastroenterological Association, will preside at the sessions. Dr. Michael W. Shutkin and Dr. Joseph Shaiken, both of Milwaukee, are the chairmen of the program and general arrangements committees, respectively.

The Central Region is comprised of the states of Illinois, Indiana, Iowa, Kansas, Michigan, Minnesota, Missouri, Nebraska, North Dakota, Ohio, South Dakota and Wisconsin.

Members of the medical profession are cordially invited to attend. A copy of the program may be obtained by writing to Dr. Joseph Shaiken, 536 West Wisconsin Ave., Milwaukee 3, Wisc., or to the Secretary, National Gastroenterological Association, 33 West 60th Street, New York 23, N. Y.

CRIPPLED CHILDREN'S SERVICES

The attention of practitioners, throughout the State of Minnesota, is called to the fact that the Crippled Children's Services spring orthopedic field clinics began in February of this year. The following is the schedule of clinics:

Spring Clinic Schedule

St. Cloud*	February 27	Technical High School	Benton Stearns
Cloquet†	March 17 & 18	Civic Center	Carlton Cook So. St. Louis Lake
Faribault†	March 23	Bethlehem Academy	Carver Dakota Goodhue Rice Scott Steele Waseca
Brainerd*	March 27	Franklin Jr. High	Crow Wing Wadena
Hibbing†	April 1 & 2	Village Health Dept.	St. Louis West Range
Worthington*	April 10	Grade School	Cottonwood Jackson Murray Nobles Pipestone Rock
Albert Lea†	April 20	Hawthorn School	Freeborn
Grand Rapids†	April 23 & 24	Court House, County Nurse's Office	Itasca
Austin†	May 4	High School, Nurse's Office	Dodge Mower
Detroit Lakes*	May 8	Rossman School	Becker Clay Mahnomen
International Falls†	May 14	Holler School	Koochiching Lake of the Woods
Thief River Falls*	May 22	High School	Kittson Marshall Pennington Red Lake Roseau
Morris*	June 5	High School	Bigstone Douglas Grant Pope Stevens Traverse

*County Welfare Board Social Service for clinic intake arranged by Field Representative. All *new* patients are requested to bring referrals from their family physicians.

†Social problems arising at clinic will be referred to County Welfare Board on day of clinic. Intake service *not* necessary from County Welfare Board. Gillette patients should not be invited to these clinics. New patients are requested to bring written referrals from their family physicians. *All* patients should have appointments arranged through local Public Health Nurses.

Children may be referred, for consultation purposes, to any of these clinics. A written referral would be appreciated. The clinics are sponsored by Crippled Children Services of the Medical Services Division of the Department of Public Welfare of the State of Minnesota.

RUSSELL M. WILDER FELLOWSHIP

A three-year, $15,000 fellowship, for post-doctorate training in the science of nutrition has been established as the Russell M. Wilder Fellowship in honor of the Rochester physician, Russell M. Wilder, the National Vitamin Foundation of New York reported recently. This foundation's program is designed to stimulate the training of qualified young men and women to pursue research and teaching in the science of nutrition.

Dr. Wilder, who recently retired as director of the National Institute of Arthritis and Metabolic Diseases of the National Institute of Health, is living in Rochester. Head of the Mayo Foundation's department of medicine, 1931-1950, he served during World War II also as chief of the civilian food requirements branch for the United States War Food Administration.

Candidates for the fellowship must apply in writing to the national Vitamin Foundation, 15 East 58th Street, New York 22, New York, to obtain official application forms that must be returned on or before March 15. The successful applicant will begin his work at the beginning of the academic year, September, 1954.

LECTURE ON MEDICINE AND SURGERY

Mr. August C. Krey, distinguished Professor of Medieval History, will give a lecture at the Todd Amphitheater, University of Minnesota Hospitals on Monday, April 5, 1954, at 7:30 P.M. on the subject "One Thousand Years of Medicine and Surgery, 600-1600 A.D."

Mr. Krey's special interest in the history of medicine and surgery suggests definitely that this should be a very delightful occasion. Anyone interested is invited.

DULUTH ACADEMY OF GENERAL PRACTICE

The Duluth Academy of General Practice held its first annual meeting at St. Mary's Hospital, January 28, with the following elected to office: Dr. Herbert O. Hoff, president; Dr. Ralph J. Eckman, vice president; Dr. C. H. Christensen, secretary, and Dr. Henry J. Jeronimus, representative of the Duluth chapter to the state Academy of General Practice.

HENNEPIN COUNTY MEDICAL SOCIETY

Dr. Charles A. Aling is president-elect of the Hennepin County Medical Society to succeed Dr. John Moe, October 1, 1954. Other officers elected by mail ballot and announced at the meeting held February 1 are: Dr. Willis L. Herbert, first vice president; Dr. James A. Blake, second vice president; Dr. Silas C. Andersen and Dr. Leo C. Culligan, board of directors; Dr. Charles E. Merkert and Dr. Dale H. Correa, board of censors; Dr. James F. Shandorf and Dr. Walter H. Ude, board of ethics, and Dr. Roy E. Swanson and Dr. Ude, board of trustees.

RED RIVER VALLEY MEDICAL ASSOCIATION

Dr. Alan M. McKaig, Red Lake Falls, was elected president of the Red River Valley Medical Association at the annual meeting January 8, held at Crookston. Dr. McKaig succeeds Dr. John L. Delmore, Roseau, as president. Other officers elected were Dr. Harold R. Thysell, Crookston, vice-president; Dr. R. O. Sather, Crookston, secretary-treasurer; Dr. B. R. Kinkade, Ada, Board of Censors; Dr. George Sather, Fosston, delegate to the House of Delegates, Minnesota Medical Association, with Dr. O. K. Behr, Crookston, as alternate.

CONTINUATION COURSES

The University of Minnesota will present a continuation course in *Radiology for General Physicians* May 3 to 5, 1954, under the direction of Dr. Leo G. Rigler, professor and head, Department of Radiology. Registrants for the course will spend most of their time in the University Hospital Department of Theology reading films and carrying out procedures in association with members of the faculty of that department. A minimum of didactic material will be presented. Because of the nature of this course, registration will be strictly limited. Early application is therefore suggested. Accommodations will be available in the Center for Continuation Study as usual.

The University of Minnesota will present a continuation course in *Surgery for Specialists* in that field May 6 to 8, 1954, at the Center for Continuaion Study. Emphasis will be placed on recent advances in gastrointestinal and biliary tract surgery. Guest speaker will be Dr. Warren H. Cole, professor and head, Department of Surgery, University of Illinois College of Medicine, Chicago, who will also deliver the annual E. Starr Judd Lecture on the evening of May 6. The course will be presented under the direction of Dr. Owen H. Wangensteen, professor and chairman, Department of Surgery, and the remainder of the faculty will include members of the faculty of the University of Minnesota Medical School and the Mayo Foundation.

The University of Minnesota will present a continuation course in *Electrocardiography for General Physicians* at the Center for Continuation Study May 10 to 15, 1954. Emphasis will be placed on practical experience in the interpretation of electrocardiograms. Each registrant will have an opportunity to read approximately 200 tracings under the supervision of the teaching staff. Separate sessions are being planned for those who consider themselves "beginners" in electrocardiography. The faculty for the course will consist of members of the staff of the University of Minnesota Medical School and the Mayo Foundation.

In Memoriam

SETH EDWIN GILKEY

Dr. Seth Edwin Gilkey died January 19, 1954, at St. Andrews Hospital in Minneapolis. He was sixty-eight years old.

Dr. Gilkey had practiced medicine for nearly twenty-five years at Watson, Minnesota, and in Minneapolis for several years. He was born at Dundas and attended South High school in Minneapolis and Hamline University before going to the University of Minnesota medical school from which he was graduated in 1909.

At the time of his death Dr. Gilkey was associated with the Minnesota Department of Veterans Affairs and lived at 1911 West Ramsey County Road D. He served in the United States Army Medical Corps in World War I and as an examining physician at the Farragut, Idaho, naval base in World War II. He was also medical advisor for an extended period for the Minnesota Division of Social Welfare. He was a member of the Ramsey County Medical Society, the Minnesota State Medical Association and the American Medical Association.

Surviving are his wife, the former Jean Robb of Minneapolis; a son, Seth E. Gilkey, Jr., St. Paul; his mother, Mrs. Eunice L. Gilkey, Minneapolis, and four sisters, Mrs. John Robb, Mrs. Albert Maiers, and Mrs. Anthony Pratschner, all of Minneapolis, and Mrs. Raymond Galbraith, California.

RICHARD WILLIAM MAERTZ

Dr. Richard William Maertz died at the age of thirty-three after a long illness in Faribault, January 13, 1954. He was the son of Dr. W. F. Maertz of Saint Paul and had lived in Faribault since 1950.

Dr. Maertz was born at New Prague and received his preliminary education in the New Prague schools and at St. John's University at Collegeville. He was graduated in medicine from Creighton University at Omaha in 1946 and served his internship at St. Joseph's Hospital in Saint Paul. He practiced first at Goodhue, Minnesota, before moving to Faribault. He was a member of the Rice County Medical Society, the Minnesota State Medical Association and the American Medical Association.

He is survived by his wife, the former Shirley O'Connor and six children: Richard, 8; twins David and Thomas, 7; James, 5; Mary, 3, and Elizabeth Ann, three months. Also surviving are his parents, Dr. and Mrs. Maertz, and three sisters, Mrs. Leon Cain and Mary Ruth Maertz of Saint Paul and Mrs. Robert Turner of Alexandria, Virginia.

ALBERT MILLER

Dr. Albert Miller, emeritus member of the staff of the Mayo Clinic, died at the age of eighty-six at Methodist Hospital in Rochester, February 7, 1954.

Dr. Miller was a pioneer roentgenologist. He joined the Clinic staff in 1913 as assistant to Dr. Russell D. Carman. The two men together published many papers on roentgenology, also the textbook, "The Roentgen Diagnosis of Diseases of the Alimentary Tract" in 1917. In 1923 he became a consultant in the section of roentgenology at the Clinic and in 1923 an instructor in radiology in the Mayo Foundation graduate school of the University of Minnesota. He retired from the Clinic staff in 1944.

Dr. Miller was born in Posey county in Indiana. As a young man he was interested in newspaper work and he became editor for a time of a Princeton, Indiana, newspaper. He was graduated from the University of Indiana in 1888 and from the Marion Sims-Beaumont Medical college, later the St. Louis University medical school, in 1903. He served an internship in the St. Louis City Hospital and practiced there and in East St. Louis until he went to Rochester.

His only survivor is his wife, the former Myrtle M. Richardson, of Rochester.

OLAUS LEWIS PETERSON

Dr. Olaus Lewis Peterson who practiced medicine for forty-five years at Cokato, died January 30, 1954, at the age of eighty-one.

Dr. Peterson was a leading citizen of his community for many years, having served as mayor and as a long-time member of the Board of Education as well as physician and hundreds of patients and friends attended a community celebration in his honor held at Cokato in 1949.

He was born at Collinwood Town near Cokato and received his B.A. degree from Gustavus Adolphus college. He was graduated in medicine from the University of Minnesota in 1905 and served his internship at Bethesda Hospital in St. Paul. He entered practice at Lindstrom in 1906 and practiced thereafter for short periods at Mora and Lafayette before settling permanently in Cokato.

Dr. Peterson was a member of the Wright County Medical Society, the Minnesota State Medical Association and the American Medical Association.

Surviving him are his wife, Mathilda; a son, Dr. Osler Peterson, who is associated with the Rockefeller Foundation and stationed at Chapel Hill, North Carolina; and two grandchildren.

AVERY deHART PRANGEN

Dr. Avery deHart Prangen, senior consultant in ophthalmology at the Mayo Clinic, died after a long illness at St. Mary's Hospital in Rochester January 30, 1954. He was sixty-three years old.

Dr. Prangen was born in Hornell, New York, and

(Continued on Page 240)

Woman's Auxiliary

WANTED: RECRUITS FOR THE HEALING ARTS

The following article was written by Mrs. David J. Halpern, Auxiliary member from Brewster, for *Minnesota Clubwomen,* the publication of the Minnesota Federation of Women's Clubs. This organization is doing an excellent job with a program of recruitment for allied medical careers. It was felt that this article was worthy of reprint here since doctors' wives are interested in the projects of other women's groups who are so vitally interested in medicine and related subjects. Auxiliary members are asked to write Mrs. Stanley Peterson, 510 Rockford Street, Austin, chairman of the Auxiliary's committee on allied medical careers, for informational material.

" 'I'd like to be a doctor,' says your teen-age son. Or. 'I think I'll go into nursing,' muses your high-school daughter. These are well-known professions, respected and honored. But too few young people with an interest in the healing arts know about the opportunities in other fields that are closely allied to medicine and nursing. That is one reason why there is a serious shortage of trained workers in these fields. We as clubwomen can help to meet this need by informing ourselves about these opportunities and telling our young folks about them.

"Many young people are fascinated by hospitals but know little of the variety of talents, skills, and opportunities involved in some of the jobs done there.

"*Medical technology,* for instance . . . The 'med tech' —usually a girl, though some young men do enter this profession—is the doctor's invaluable aide in performing tests for diagnosing hundreds of conditions and in determining the most effective treatments to use. Both men and women find exciting jobs in the field of *x-ray* technology—a rapidly developing field that requires a variety of highly skilled techniques. Aptitude in science and mathematics, plus an interest in medicine, are desirable in the young person looking toward either of these jobs.

"Any girl who likes to study foods and nutrition in relation to their bearing on health would do well to consider becoming a *dietitian.* Many dietitians work in hospitals, where they are responsible for planning and serving special diets prescribed by doctors for their patients. With this skill and training as a basis, the dietitian might go on to become a nutritionist and serve as consultant to many dietitians and other professional people.

"Two careers are of special interest to those who deplore the tragic waste of economic and human resources because thousands of otherwise active people are disabled by accidents or disease. Feats that seem like miracles are being performed with such people today, through the aid of *physical therapy*—treatments using hot and cold water, massage, electric currents, and various mechanical devices. As the patient by these means recovers some of the normal functions of injured or paralyzed muscles, he can improve his co-ordination, learn new skills, and discover a fresh interest in life through *occupational therapy.* Leather work, woodwork, metal work, clay modeling, weaving—these are only a few of the skills taught by the occupational therapist. Both physical and occupational therapy—especially the latter—are frequently of value in restoring sick minds as well as disabled bodies.

"There is also a career as a *medical record librarian.* A medical record is written by the physician and hospital team for every patient entering a hospital. This story of why the patient was admitted, the special examinations made to arrive at a diagnosis, the treatment given and the results of care then becomes the medical record librarian's responsibility. She uses these living accounts daily in working with physicians and hospital staff for better patient care, hospital administration, and medical research. For a boy or girl with an interest in patient care, administration and education who also has a flair for detail, the position of medical record librarian offers unlimited opportunities.

"The job of medical record librarian must not be confused with that of *medical librarian* or *hospital librarian.* These, too, offer interesting careers. Both require basic training in library science, followed by some specialization. The medical librarian works in a medical library—a library that serves chiefly physicians and members of allied professions. The hospital librarian is a specialist in a library that serves chiefly hospital patients.

"Our mental picture of a *nurse* is apt to be of a white-clad woman moving quietly and efficiently about a hospital floor. But we must remember that there are many different careers in the general field of nursing. The need for both professional and practical nurses still far exceeds the number in training, and men as well as women are needed in many branches of nursing. There is need for nurse anesthetists, for public health nurses, for nurse educators, nurses to serve as helpers in medical research and to administer hospitals, schools of nursing, and state and national programs.

"Still another field in which the number of jobs available far exceeds the supply of trained people is that of *health education.* Health educators are recruited from a surprising variety of backgrounds—teaching, nursing, and journalism being the chief. A health educator serves as consultant in educational methods and materials to schools, communities, health departments, or voluntary health agencies.

"All the professions mentioned require considerable education beyond high school. Not quite so exacting in its demands but offering an attractive career for the girl interested in both medicine and business is the job of *medical secretary.* Her work may begin with recording and transcribing reports of diagnoses and prescribed treatments, taking appointments and acting as receptionist, but many medical secretaries have gone far beyond this point. Some of them have even collaborated with doctors in preparing and publishing medical articles and books.

"In all these fields allied to medicine, the young person with ability, initiative, and a genuine liking for the healing arts can find satisfaction, security, and success. Every graduate of an accredited school can be sure of a position, not only in Minnesota but anywhere in the United States. There is a trend today toward higher salaries, shorter hours, and improved working conditions in all these professions. There are loans and scholarships available. For young people with a desire to serve, what could be more attractive than the opportunity to become a member of a team that works together to alleviate suffering, promote health, and save lives?"

RAMSEY AUXILIARY REPORTS PROJECTS

The Woman's Auxiliary to the Ramsey County Medical Society continued its regular activities during recent weeks.

Members staffed booths for the March of Dimes in January, in downtown Saint Paul stores, in Highland Village and in the Midway district. Approximately $300 was collected by the women. Mrs. R. K. Grau served as chairman.

The February board meeting, held on February 15 in the medical library of the Lowry Medical Arts Building, had good attendance. Mrs. Philip Arzt, president, presided, and doctors' wives made cancer dressings before and after the meeting.

The March meeting was held on March 22 at 12:30 p.m. at the International Institute. Foods from foreign countries were served and a short musical program followed. Members were taken on a tour of the Institute while regular classes were in progress. Program chairmen for this meeting were Mrs. L. T. Simons and Mrs. M. D. Hilker.

STEARNS-BENTON AUXILIARY

Early days of the Stearns-Benton County Medical Society Auxiliary were recalled at the November meeting of the group held at the home of the president, Mrs. L. M. Evans, Sauk Rapids.

Scrap books and minute books for the years from 1924 when the auxiliary group was organized, until 1930 were displayed and reminiscences were exchanged. A radio interview with founding members was re-enacted and the cook book which the auxiliary published in 1926 with an initial investment of $4.43 and an ultimate profit of $1,164 was especially remembered. The money went to furnish a room in the St. Cloud Hospital. Mrs. R. J. Cesnick of St. Cloud was program chairman for the event. Assisting hostesses were Mrs. S. J. Raetz of Maple Lake and Mrs. C. S. Donaldson of St. Cloud.

The January meeting of the Stearns-Benton group was held at the home of Mrs. W. B. Richards of St. Cloud with Mrs. Edward M. Anderson, St. Cloud, vice president, acting as chairman in the absence of Mrs. Evans.

Plans for a tea for the purpose of interesting high school girls in nursing as a career to be held soon at the St. Cloud Hospital, were outlined by Mrs. Gilman Goehrs of St. Cloud. A motion picture film called "St. Cloud, France," was shown through the courtesy of the St. Cloud Public Library. Mrs. C. F. Brigham, Jr. and Mrs. James P. O'Keefe, secretary of the auxiliary, and both of St. Cloud, assisted Mrs. Richards at the coffee hour which followed and which served to introduce two new members, Mrs. Robert De Weese and Mrs. L. A. Loes of St. Cloud.

THE MINNESOTA STATE BOARD OF MEDICAL EXAMINERS

230 Lowry Medical Arts Building
Saint Paul 2, Minnesota

E. M. Jones, M.D., Secretary

OLIVIA PHYSICIAN'S LICENSE SUSPENDED FOR FIVE YEARS

In the matter of the revocation of the license of Manford B. Dahle, M.D.

The Minnesota State Board of Medical Examiners on February 13, 1954, suspended for a period of five years the medical license held by Dr. Manford B. Dahle of Olivia, Minnesota. Dr. Dahle, at a previous meeting of the Medical Board, had admitted the truth of the charges contained in a citation for the revocation of his license which was served upon him on October 21, 1953. The citation charged Dr. Dahle with a series of acts of misconduct as a physician. These acts arose at the time or immediately following the use of alcoholic liquors by Dr. Dahle. Following Dr. Dahle's appearance before the Medical Board in November, 1953, and before the case was disposed of, Dr. Dahle was arrested and entered a plea of guilty to a charge of driving while under the influence of intoxicating liquor.

Dr. Dahle was born at Eagle Bend, Minnesota, on March 2, 1912, and received his Doctor of Medicine degree from Rush Medical College on June 10, 1938. He was licensed by examination in Minnesota in the following year and practiced at Glenwood, Minnesota, prior to locating in Olivia in October, 1948.

Of General Interest

Dr. Corazon A. Manayan, a young native of the Philippines, now is a resident physician in obstetrics at St. Mary's hospital in Minneapolis. She received her medical degree from the University of Santo Tomas in Manila. She was a second lieutenant in the underground organization when the Japanese invaded the Philippines in 1942.

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Dr. Hermina Hartig left for an extended trip in the South upon her retirement as chief Minneapolis school physician. She will return about April 1.

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Dr. Karl Vestling, and family, left for Bremerhaven, Germany, and then will leave for Stockholm, Sweden, where he will do research in bio-chemistry at the Nobel Medical institute with Dr. Hugo Theorell of that institution. Dr. Vestling is a graduate of Carleton College in 1934.

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A special alarm fire destroyed the offices of **Dr. John Williams, Dr. Robert K. Grau,** and Dr. Sherman Miller, dentist, all of Saint Paul, on February 5. Damage to the building is about $20,000 and to equipment about $25,000.

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Dr. E. J. Huenekens was awarded the 1954 distinguished service certificate today by the Community Chest and Council of Hennepin County. The Minneapolis pediatrician and civic leader is the twenty-first person to receive the annual award, given in recognition of exemplary community service.

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Dr. Karl S. Klica, medical director of St. Barnabas hospital for the past three years, submitted his resignation to become administrator of Presbyterian hospital in Chicago.

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Dr. Lawrence C. Kolb, member of the staff of the Mayo Clinic, has been appointed director of the New York State Psychiatric Institute. He will also become professor of psychiatry and executive officer of the department of psychiatry at the Columbia university college of physicians and surgeons and director of psychiatry service at Presbyterian hospital in New York.

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Dr. O. L. Norman Nelson was elected chief of staff of St. Barnabas hospital. **Dr. Robert G. Tinkham** was elected vice chairman and **Dr. Harold Buckstein** secretary-treasurer.

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Dr. David F. Struxness, formerly of Alexandria, opened his new office in Glencoe on February 1.

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Dr. Grafton A. Smith, fellow in surgery at the University of Minnesota, has been granted a $5,400 Damon Runyon fund fellowship to study under Dr. Owen H. Wangensteen, chief of surgery.

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Dr. Grant L. Griebe, Brownton, has been elected president of the McLeod County Medical Society. Dr. Griebe succeeds **Dr. Kenneth Peterson,** Hutchinson. Dr. John Smythe, Lester Prairie, was elected secretary-treasurer.

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Dr. Herman Juergens, Belle Plaine, was elected chief of staff of New Prague Community Memorial hospital. **Dr. Elmer Doherty,** New Prague, is vice president and re-elected secretary-treasurer is **Dr. Elizabeth Rieschel,** Jordan.

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Dr. Erikki A. Leppo, Duluth pediatrician, was the guest speaker at the annual meeting of the International Institute. Dr. Leppo assisted in organizing the child health section of the United Nations World Health organization and has been a medical officer with WHO in the Middle and Far East.

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Dr. Irvine McQuarrie, head of the University of Minnesota's pediatrics department, and **Dr. Edith L. Potter,** University medical school graduate and former staff member, are among ten doctors to be named to the 1954 honor roll of medicine by the magazine, *Modern Medicine*. Dr. McQuarrie was honored "for bettering understanding of metabolic disturbances in children," and Dr. Potter's award was for her "outstanding contributions in the field of pediatric pathology." Dr. Potter is the only woman to win the award this year.

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Dr. Donn G. Mosser, instructor in radiology, University of Minnesota, has been awarded one of three American Cancer society fellowships in clinical radiation therapy for study in cancer centers in Great Britain, France and the Scandinavian countries.

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Dr. David Glick, professor of physiological chemistry in the University of Minnesota medical school, will deliver two lectures in March at the Royal Caroline Medico-Surgical Institute of Sweden in Stockholm. In addition, he had been asked to lecture in the Carlsberg laboratory in Copenhagen.

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Elmer S. Paulson, Saint Paul, radiologist and clinical instructor in radiology at the University of Minnesota, spoke to the Cerro Gordo County Medical Society in Mason City, Iowa, February 9, 1954, on "Radio-iodine."

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Dr. Joseph Brennan, Duluth, told of his experiences as a camp physician when he was a prisoner of war of the Germans, his later escape and subsequent work in the American and British underground, at the Friday Club in Cloquet January 15. Dr. Brennan was educated at the University of Malta, Rome, Bologna, London as well as Minnesota. He is a Fellow in the Royal Society of England for his work in tropical medicine and hygiene.

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Dr. Carl J. Potthoff, Minneapolis, regional medical consultant for the National Foundation for Infantile Paralysis, discussed the progress of the foundation's polio prevention program at the Mankato Lions Club meeting January 14. Dr. Potthoff has previously served as associate medical director of the American Red Cross, Washington, D. C., and an area medical officer in the Bureau of Indian Affairs at Albuquerque, New Mexico.

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Dr. James H. Dokken, associated with **Dr. L. J. Hoyer**, at the Windom Clinic since July, 1953, has now become a partner. Dr. Dokken, a graduate of University of Minnesota School of Medicine, had a Fellowship at the Mount Sinai Hospital, Minneapolis, prior to his coming to Windom.

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Dr. James O'Keefe, St. Cloud, has recently moved into a new nine-room clinic with facilities for offices and laboratory, plus a private parking lot to accommodate the patients.

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A seventy-five-year-old painting has been hung in the lobby of the Waseca Memorial Hospital recently, in memory of the late Dr. Clifford T. Wadd, Janesville, who died in an automobile near Franklin, in 1952. The painting, selected by **Dr. and Mrs. S. C. G. Oeljen**, Waseca, is "Tranquility," a pastoral scene by the English artist, George Turner. It was purchased at the Finley galleries in Chicago from funds given in memory of Dr. Wadd.

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Dr. A. M. Nielsen, Northfield, spoke of the time and expense involved in building up the medical divisions of civil defense at a recent meeting of the Business and Professional Women's meeting. Supplies needed would be expensive, Dr. Nielsen said, and also a high percentage of the medicines would deteriorate, be useless after a few months, and need replacement.

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Dr. Margaret C. Anderson, Mankato, gave a talk on the topic, "Recent Advances in Anesthesia as Related to the Small General Hospital," at a meeting of the Tri-County Society January 13. The Tri-County Society includes doctors from Blue Earth, Watonwan, Brown, Sibley, Le Sueur and Nicollet counties.

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Hennepin County had 455 cases of polio with seventeen deaths in 1953 which made it the hardest hit county in Minnesota. Ramsey County had 414 cases with twenty-five deaths during the year.

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Dr. Carl W. Waldron, Hopkins, was appointed head of the Emergency Medical and Public Health Committee of the Minneapolis Civil Defense office January 14. Dr. Waldron succeeds **Dr. Frank J. Hill**, who recently resigned as city health commissioner.

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Dr. Philip A. Olson, Grand Forks, North Dakota, and Ruth Ann Tjossem, Sioux City, Iowa, were married recently at the Mount Olivet Lutheran Church, Minneapolis. Dr. Olson attended the University of North Dakota, was graduated from the University of Illinois School of Medicine, and is completing a residency in radiology at University of Minnesota.

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Duluth Health Department closed its venereal disease clinic recently after thirty-five years of operation. Dr. Mario Fischer, health director, said that the clinic, held twice a week at the Miller Memorial Hospital is no longer necessary. He said, "It is significant in that many years ago the venereal disease picture was thought to be almost insurmountable. It has now become so infrequent here that, having it treated at taxpayers' expense is not justifiable."

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Dr. Mitrofan Smorszczok, a refugee from Europe, now of the Hart Hospital staff at Monticello, spoke at the Braham Area Parent-Teacher Association January 18. A practicing physician in Latvia, he came under Communist rule when that country was divided between Germans and Russians. In a so-called free election of commissar, Dr. Smorszczok found he had to vote for the Communist candidate in order to stay alive. Medical supplies were also difficult to get during his practice there.

* * *

Dr. Hermina Hartig, Minneapolis, had her last day as chief school physician of Minneapolis schools when she turned sixty-five January 13, but she is not ready to "sit down in a rocking chair and knit." "In fact, I'd first have to learn how to knit," she said recently. Born in Wahpeton, North Dakota, Hermina Simonson came to the University of Minnesota to train as a medical missionary. While at the University she met and married Dr. Hugo Hartig. Her husband was killed in an automobile accident in 1926, and, with four children, Dr. Hartig went back to her profession after taking refresher courses. In 1934 she became school physician under the city's health commissioner and in 1944 she was made head of the department.

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Dr. Justus Ohage, Saint Paul, president of the Minnesota State Medical Association, is the newest member of the Minnesota Poll's Advisory Board, succeeding **Dr. O. J. Campbell** of Minneapolis.

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Dr. Walter C. Alvarez, Chicago, spoke at the Minnetonka Parent-Teacher Association, Excelsior, January 12, on the topic, "What Makes Mothers Nervous." Dr. Alvarez, medical columnist, editor, and former Mayo Clinic physician, is a recognized authority on psychosomatic medicine.

Dr. Norman Johnson, Minneapolis, spoke at St. Luke's Episcopal Hospital Church, Hastings, January 20, on the subject "The Religious Approach to Psychosomatic Medicine." Dr. Johnson, son of an Episcopal bishop, has the background to speak authoritatively in the field of medicine and religion.

* * *

Dr. B. J. Grimes, superintendent of the State Hospital at St. Peter, spoke at St. James' Rotarians at a club luncheon January 12, at St. James. His theme, care for the aged, brought out the need for the older folk to stay in their own communities and be near their families and relatives. Referring to those who also need hospitalization, attention was focused on the need for a geriatric unit in connection with the Watonwan Memorial Hospital.

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Dr. Albert J. Chesley, secretary and executive officer of the State Board of Health, stated recently that the board "wants to be sure of the safety and probable results" of the polio vaccine before it is used in Minnesota. A special committee has been named to make a continuing study of the vaccine in order that a decision can be reached before the polio season begins next summer.

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Dr. E. S. Boone, Luverne, has moved in to his new office location which is equipped with examining suite, x-ray machine, et cetera, all newly purchased.

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Dr. J. T. O'Neil, intern at St. Mary's Hospital, Duluth, addressed members of the Miller Hospital School of Nursing, January 12, at the hospital clinic.

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Dr. D. P. Anderson, Austin, president of the Minnesota Division of American Cancer Society, spoke on lung cancer and the effects of smoking at a meeting held by the Austin Chamber of Commerce January 4.

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Five study groups have been set up recently by the Minnesota Commission to study the problems of the aging, with John Poor of the State Department as head of the subcommittee. The following have been chosen as chairmen: **Dr. Helen Knudsen**, State Health Department, to study health, chronic illness and rehabilitation; Robert Cerny, Minneapolis architect, living arrangement; A. Merrill Anderson, director of Minnesota state employment and economic welfare, and Dr. John E. Anderson, director of University of Minnesota Institute of child welfare, education, recreation, counselling and vocation skills.

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Dr. and Mrs. J. H. Leebens, formerly of Lismore, now of Minneapolis, observed their golden wedding anniversary January 17, with open house at their home. Dr. Leebens practiced medicine in the Lismore community from 1907 to 1935.

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Dr. J. A. Rorem, Appleton, was reinstated as health officer of the village at the village council's meeting January 6.

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Dr. Michael Danyluk, until recently associated with Dr. Hamlin Mattson and Dr. K. A. March in pathology and surgery at the Swedish Hospital in Minneapolis, has joined Dr. March in his practice at Cambridge. Dr. Danyluk studied medicine at the University of Munich in Germany. He came to Minneapolis in 1950.

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Dr. and Mrs. R. E. Billings, Franklin, were hosts to the Renville County Medical Association and its Auxiliary at their home recently. A pot-luck supper was served to the twenty-eight guests attending.

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In the first edition of a publication, "Leaders in American Science," recently issued in Nashville, Tennessee, several Mayo Clinic staff men were listed. They are as follows: **Drs. Joseph McDonald, William H. Feldman, Frank C. Mann, J. Grafton Love,** and **J. A. Bargen** of Rochester, and **Benjamin T. Terry,** Tacoma, Washington, formerly of the Clinic.

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Dr. G. F. Engstrom, Belgrade, is temporarily filling the position as physician in charge of medical-surgical emergencies at the Moose Lake State Hospital. Dr. Engstrom has recently completed four months of medical work at the University of Minnesota and University Hospital.

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Dr. James H. Kelly, associated with the Davis Clinic, Wadena, for the past three and a half years, has affiliated with his brother, **Dr. Robert Kelly** in the Itasca Clinic in Nashwauk.

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Dr. W. H. Valentine, Tracy, president of the Lyon County Tuberculosis and Health Association, stated recently that the county-wide tuberculin testing for all ages has received state and nationwide attention. More than $6,800 in county Christmas Seal funds financed the testing program with 13,667 persons having been tested. Of these, 20 per cent had tuberculosis germs, while 30 per cent of the adults are reactors.

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Dr. Harold Williamson, Fairmont, spoke on the need and usefulness of clinics at the Lions Club in Fairmont, January 11. "Group practice means better working conditions and better prepared doctors," he said.

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The Minneapolis War Memorial Blood Bank has increased its services to local and Upper Midwest residents. Last year a total of 20,560 units of blood were handled by the bank making a 12 per cent increase over the year before.

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First Lieutenant John Rutledge, son of **Dr. and Mrs. L. H. Rutledge** of Detroit Lakes, serving in the medical corps of the army, has been given a forty-five day assignment in Raykjavik, Iceland. Lt. Rutledge, stationed at Westover Field, Chicopee Falls, Massachusetts, will serve as flight surgeon on this assignment.

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The medical staff at the Lutheran Hospital at Bemidji were hosts to the pharmacists of Bemidji and surrounding villages January 20. Problems common to the professions, such as control of prescription refills, wider use of standard drugs, measures to minimize self diagnosis and self medication in the presence of serious symptoms and control of drug addiction were discussed. Henry Moen, a pharmacist and executive secretary of the Minnesota Pharmaceutical Association, was the principal speaker. **Dr. Daniel F. McCann** paid tribute to the silent service the pharmacist performs in checking and preparing prescriptions.

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Dr. Lloyd C. Gilman, who has been on leave of absence from his practice at Willmar while doing special work in the field of surgery at the Jackson Clinic, Madison, Wisconsin, returned to his office in January.

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A University of Minnesota Research Director and other heart specialists left January 8, for Italy where they will study the connection between the small incidence of heart disease in that country and the Italian diet. "We noted some peculiarities of blood chemistry there before and now we want to explore further the relation of diet and heart disease," said **Dr. Ancel Keys,** member of the party and director of the University's laboratory of physiological hygiene.

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Mount Sinai Hospital, Minneapolis, has started construction of a $100,000 animal research laboratory near the hospital. There will be a large operating and sterilizer room, isotope laboratory, food preparation area, post-mortem room, preparation and recovery room. There will also be separate quarters for dogs, mice, guinea pigs, rabbits, and other animals used for medical research. The unit will be known as the Jay Phillips Research Laboratory, in honor of the first and present president of the Hospital Association.

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Dr. A. J. Floersch, a graduate of Creighton University Medical School, Omaha, Nebraska, opened his office for practicing medicine at Glencoe, February 1. Dr. Floersch took his internship at St. Mary's Hospital, Minneapolis, served in the armed forces two years, and had a year's surgical residency at Northwestern Hospital, Minneapolis, before coming to Glencoe.

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Dr. J. J. Heimark and **Dr. R. L. Parsons** have dissolved their partnership at Monterey. Dr. Heimark is returning to Fairmont to his former office to practice medicine, and Dr. Parsons will, after a vacation, open an office either in Fairmont or elsewhere. Dr. Vincent Ryding, at present resident surgeon at Northwestern Hospital, Minneapolis, will open his office in Monterey July 1, when his residency is completed.

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Dr. B. J. Cronwell, Austin, left for a week of postgraduate work in electrocardiography. The course was offered by Emory University.

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Dr. Lewis Hanson, Frost, was elected chairman of the Faribault county public health nursing advisory board. **Dr. A. B. Peterson,** Wells, was elected vice chairman and Mrs. Carl Hawkins, secretary-treasurer.

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Dr. Frederick A. Figi, Mayo Clinic, Department of Plastic Surgery, will speak on the subject "Cancer and Chemodectoma of the Middle Ear and Mastoid," at the meeting of the American Medical Association in San Francisco, June 21-25.

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Dr. C. M. Smith, Duluth, was installed as chief of staff of St. Luke's hospital for 1954. The chief elect for 1955 is **Dr. M. F. Fellows. Dr. Ralph J. Eckman** was re-elected secretary. New executive members are **Dr. D. W. Wheeler,** chief of medicine; **Dr. P. N. Bray,** chief of obstetrics; **Dr. William Swedberg,** chief of orthopedics; **Dr. Gordon Strewler,** chief of neuropsychiatry; **Dr. L. L. Merrian,** chief of infirmary service, and **Dr. H. O. Hoff,** chief of general practice.

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Dr. Albert J. Chesley, Saint Paul, **Dr. William Benedict,** and **Dr. Charles Sheard,** Rochester, have been re-elected to three-year terms on the board of directors of the National Society for the Prevention of Blindness.

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Dr. C. H. Sherman, Bayport, was appointed physician and surgeon of the state prison. He succeeds **Dr. E. M. Jones,** Bayport.

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Dr. and Mrs. P. H. Macfarlane were aboard the luxury liner SS Lurine for an extended visit in Hawaii. Dr. Macfarlane is associated with the Mesaba Clinic and the Chisholm Memorial Hospital.

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Dr. Philip Hench attended the regional meeting of the American College of Physicians in Colorado Springs, Colorado. He presented a paper "The Cortisones and Corticotropins in Rheumatoid Arthritis."

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Dr. Henry Reif was elected president of the Stearns-Benton Medical society, to serve in 1955. **Dr. John Kelly,** Cold Spring, will serve as head of the group during 1954. Other officers elected for 1954 were **Dr. William Autrey,** vice president and **Dr. E. M. Anderson,** secretary.

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Dr. D. V. Boardman was elected president of the Winona County Medical Association. **Dr. Pat Rol-**

lins was re-elected treasurer and **Dr. W. O. Finkelnburg** was re-named secretary.

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Dr. Thomas J. Kenyon has been named president of the medical staff of St. Joseph's Hospital. Other new officers are **Dr. J. P. Medelman,** vice president, **Dr. Richard W. Leick,** secretary, and **Dr. Bernard O'Reilley,** treasurer.

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Dr. Kristofer Hagen, who has for some time, been associated with Dr. Theodore Greenfield at Cokato, Minn., has now opened his own office at the Hub Medical Center, 34 W. 66th Street, Richfield, Minnesota. Dr. Hagen returned from India in 1952, after six years' service under the Santal Mission in charge of a mission hospital at Mohulpahari, Santal Parganas, Bihar.

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HOSPITAL NEWS

At a recent election at Asbury Methodist Hospital, Minneapolis, members of the medical staff were elected as follows: Dr. Paul J. Preston, chief-of-staff; Dr. Robert B. Potter, vice president, and Dr. Charles D. Adkins, secretary-treasurer. Members of the advisory board together with the officers are: Dr. R. C. Webb, surgery; Dr. George E. Hudson, maternity; Dr. Wayne S. Hagen, medicine; Dr. Alice K. Brill, pediatrics, and Dr. P. C. Petersen, general practice.

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Dr. Olga Hansen-Litzenberg has been re-elected chief of Eitel Hospital medical staff. Dr. G. T. Schimelpfenig, is assistant chief, and Dr. O. L. Zahrendt is secretary-treasurer.

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The Minneapolis Hospital Council held elections at their annual meeting January 21 with the following officers elected: Dr. Benjamin W. Mandelstam, administrator of Mount Sinai Hospital, president; Kenneth J. Holmquist, superintendent of Minneapolis General Hospital, vice president; Vernon Spry, administrator of Asbury Methodist Hospital, secretary, and Carl N. Platou, administrator of Fairview Hospital, treasurer. As president Dr. Mandelstam succeeds Dr. Karl S. Klicka, administrator of St. Barnabas Hospital.

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Dr. F. G. Chermak, International Falls, has been elected chief of staff of Falls Memorial Hospital recently. Dr. Chermak was formerly associated with the Webber Clinic in Duluth and the Hibbing General Hospital.

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Dr. J. F. Zagaria, formerly of Wausau, Wisconsin, has been named chief anesthesiologist of St. John's Hospital, St. Paul. Mrs. Mary Anna Anderson has been named chief pharmacist and Miss Virginia Reid, head dietitian.

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The medical staff of St. Ansgars Hospital, Moorhead, elected Dr. V. J. Carlson, president; Dr. O. H. Johnson, vice president, and Dr. Carl Simison, secretary-treasurer. The president and secretary-treasurer together with Dr. H. G. Rice and Dr. J. W. Duncan, will compose the executive committee.

Formal organization of the Twin City Hospital Association by the Minneapolis and St. Paul Hospital Councils has been complete. Donald Wood, executive secretary, will be located in the Minnesota Hospital Service Association (Blue Cross) Building, 2610 University Avenue, St. Paul.

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Dr. Bernard I. Saliterman succeeded Dr. Max Seham as chief of staff of Mount Sinai Hospital, Minneapolis, recently. Dr. Meyer Z. Goldner, was elected vice president; Dr. Ephraim B. Cohen, secretary-treasurer. Department chiefs are: Dr. Robert A. Green, medicine; Dr. David Gaviser, surgery, and Dr. Maxwell M. Barr, obstetrics-gynecology. The executive committee is comprised of the officers, department chiefs, and Dr. Sidney Scherling, Dr. Julius Feinstein, and Dr. Max Seham.

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Miller Memorial Hospital, Duluth, held elections for the medical staff January 8. Dr. A. C. Kelly was elected chief-of-staff; Dr. F. T. Becker, vice chief of staff; Dr. John Schmid, secretary-treasurer, and Dr. J. C. Feuling and Dr. J. J. Dwyer, executive committee members.

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Officers of the Abbott Hospital's medical staff is as follows: Dr. John Haugen, president; Dr. O. J. Campbell, vice president; Dr. Walter Hoffman, secretary; Dr. John Tobin, Dr. John Pewters and Dr. John Boehrer, members of the executive committee.

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The Fairmont Hospital board at a meeting recently offered to meet the trustees of Tri-Mont Hospital, hospitals located in Martin County, to work out proposals to submit to the county commissioners that would be satisfactory and helpful to both hospitals. Fairmont Hospital, forty-six beds, is in dire need of enlargement and added facilities, and is seeking an allotment of county funds to meet the extra expenditures. Citizens of the Triumph-Monterey area feel that they should not be asked to support through taxes another hospital when they have their own, built a few years ago at a cost of $150,000.

Dr. Helen Knudsen, State Chief of Section of Hospital Service, and Dr. Robert N. Barr, deputy executive officer of the State Board of Health, spoke to the group concerning these problems.

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Facing the need of enlargement both in space and facilities of the St. Mary's Hospital of Duluth, the administration is getting under way in the fund-raising campaign. G. E. Andreson, Chairman of the St. Mary's Hospital Building Fund campaign, has appointed R. P. Buckley, M.D., chairman of the Medical Division of the campaign. F. T. Becker, M.D., and E. D. Bettenhausen, D.D.S., will assist him as vice chairmen in organizing solicitations among doctors and dentists of Duluth.

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Dr. O. J. Seifert, New Ulm, was elected president of the Loretto Hospital at the annual staff meeting January 19, with Dr. Rolf Hovde, Winthrop, as vice president, and Dr. Howard A. Vogel, secretary. Dr. C. A. Saffert is the retiring president.

At the annual staff meeting of the Union Hospital January 19, the following were elected: Dr. Howard A. Vogel, president; Dr. Albert Fritsche, vice president, and Dr. Rolf Hovde, Winthrop, secretary. Dr. Vogel succeeds Dr. William Black as president.

MINNESOTA BLUE CROSS-BLUE SHIELD

In the year 1953, payments to doctors for services rendered to Blue Shield subscribers totaled $4,247,000. This amount marks a new high in annual amount of money paid to doctors as well as an increased percentage of the amount paid by the subscriber returned to him in the form of medical care benefits. While the dollar volume of claim payments in 1953 has increased 15 per cent over 1952, the amount paid out in 1953 represents approximately 50 per cent of the total amount paid out for claims in the preceding three years. Further analysis shows that the payments in 1953 are 88 per cent greater than the amounts paid in 1950. With the constant increase noted each year, and with the increased benefits in the contract effective January 1, 1954, Blue Shield payments to doctors of medicine for services rendered Blue Shield subscribers should approach the $5,000,000 mark in 1954.

Other interesting facts in the growth of Blue Shield claims payments are the great number of claims received and processed during the year with no increase in the number of personnel employed. Through a four-year period, while the dollar volume of benefits has increased 88 per cent, the number of claims received has increased over 100 per cent. However, the number of persons employed by Blue Shield has increased only 20 per cent since 1950, and for almost two years has remained constant. Where in 1952, an average of about 10,000 claims was processed each month, in 1953 this increased to an average of 11,500 or an increase of 15 per cent. Yet with this increase, no additional personnel was necessary. Again in 1953, as in 1952, the assimilation of an increased work-load reflects progress in internal procedures which permits of an efficient operation and a reduction in the cost of claims processing.

Another interesting element in the ability of the personnel to assume an additional work-load is evident by a reallocation of the correspondence received. While in 1952, six persons were involved in handling the various types of correspondence, with the increased number of claims and growth of Blue Shield, reorganization of work-loads and personnel has avoided the need to increase the number of personnel in this particular unit.

Internal reorganization of the Blue Shield Claims Department also provides more time to disseminate information to members of the medical profession and to improve relations with doctors. More time has been available for providing more information about the Blue Shield contract and the Blue Shield procedures, both of which result in greater efficiency in the doctor's office and in Blue Shield. In this particular field, many of the problems which have formerly existed were eliminated through the professional relations activities in 1953. The program of professional relations is continuing and is being increased as rapidly as possible. It is intended that continued progress will be made in this field and that greater contact can be made with those persons in the doctor's office in order to eliminate many of the problems confronting them.

During the month of December, 10,376 participant subscribers were enrolled in Blue Cross bringing the net enrollment as of December 31, 1953, to 986,885 Blue Cross participant subscribers.

Hospitalization expense incurred for subscribers for the month of December, 1953, reached the all time high point of $1,471,749.53, bringing the total amount paid for the care of subscribers during 1953 to $15,134,121.69. In comparison with the figures of 1952 to date, over $2,587,665.46 more was paid for the care of subscribers during 1953.

At the same time, even though hospital costs were at an all time high, Blue Cross reduced its overhead expense to an all time low of 6.8 per cent of income compared with 7.7 per cent of income during 1952.

Mr. A. M. Calvin, president of Blue Cross, said that operational improvements, as a result of a competent staff, have helped to reduce operational costs. Hospitalization has shown a decided increase with one out of every six Blue Cross participant subscribers hospitalized last year. The co-operation of the public, the hospitals, and the medical profession is needed to determine whether or not the hospitalization of all persons was for necessary hospital care.

One out of every three persons in Minnesota is now enrolled in Blue Cross and this figure exceeds that of the nation as a whole which has every fourth person enrolled.

IN MEMORIAM

Avery deHart Prangen

(Continued from Page 232)

received a B.S. degree in 1913 and an M.D. degree in 1916 at the University of Michigan. He served his internship at the University of Michigan hospital and became an instructor there in 1917. He joined the Mayo Clinic staff as an assistant in ophthalmology in 1918 and continued with the Clinic for thirty-five years until his retirement because of illness in 1952. He served also as associate professor of ophthalmology in the Mayo Foundation.

He was a member of the Minnesota Academy of Ophthalmology and Otolaryngology, the American Academy of Ophthalmology and Otolaryngology, the American Ophthalmological Society, Alpha Kappa Kappa and Sigma Xi. He held membership, also, in the Olmsted-Houston-Fillmore-Dodge County Medical Society, the Minnesota State Medical Association and the American Medical Association. Until his retirement Dr. Prangen had been chairman for four years of the National Interprofessional Committee on Eye Care and he was the first to hold the post. He was a member of the Rochester Kiwanis club and president in 1926.

Surviving are his wife, the former Alice Conley, a daughter, Mrs. David Judd of Eugene, Oregon, and a son, William, of Minneapolis.

BOOK REVIEWS

Books listed here become the property of the Ramsey, Hennepin and St. Louis County Medical Libraries when reviewed. Members, however, are urged to write reviews of any or every recent book which may be of interest to physicians.

THE PEPTIC ULCER INDIVIDUAL. Gerdt Wretmark. 183 pages. Illus. Price 20 Swedish Crowns, paper cover. Copenhagen, Denmark: Ejnar Munksgaard, 1953.

SCHOOL HEALTH SERVICES: A report of the Joint Committee on Health Problems in Education of the National Education Association and the American Medical Association. Charles C. Wilson, M.D., Professor of Education and Public Health, Yale University, Editor. Illus. Price $5.00. Washington: National Education Association of the United States, 1201 Sixteenth Street N.W. or Chicago: American Medical Association, 535 North Dearborn Street, 1953.

This is a comprehensive and highly useful book for anybody who has any responsibility for the care of children, but especially for teachers, nurses and school physicians.

It is happily free from the high-flown educator's shop-talk which occasionally plagues the uninitiated reader. And many for whom the book will be useful are not initiated into these technical mysteries.

Since it is sponsored jointly by the National Education Association and the American Medical Association, it steers a sure course through the sometimes ruffled waters of division of responsibility for the care of children between the school, the parent and the family physician. Also it provides an excellent, up-to-date compendium of information on child health, including a valuable section on all the health problems connected with physical education, school athletics, and school camps.

Each chapter and section was prepared by experts in their fields and the whole has been assembled with a wealth of illustrative material, sample forms, lists of supplies, studies and graphs to serve as a handy manual, as well as a statement of sound principles underlying a school health program.

Many a school administrator has been waiting for a practical book like this, but parents, as well as teachers, could profit by reading it and keeping it handy.

MANAGING YOUR CORONARY. By William A. Brams, M.D., 158 pages. Illus. Price $2.95, cloth. Philadelphia: J. B. Lippincott Co., 1953.

Explaining science to the general reader is a responsibility not always accepted by physicians. Dr. Brams is well equipped to help patients understand the heart, and its most common affliction, and discharges his appointed task. I found little to object to and would recommend it to patients who are interested in their condition. The patient with "the head in the sand" attitude toward his illness would not be benefited by such a discussion.

The critical would not accept the thesis that coronary disease is more common in the "executive class" or that worry and modern life's tension necessarily are causative. The drawings of the heart and its vessels (Figs. 2 and 3) are anything but clear. Figure 4 is not labeled as a microscopic detailed drawing. On page 44, there is a reference to "the ST wave," apparently a new electrocardiographic term. On page 96, it is stated "the commonest complication after an attack of coronary thrombosis is embolism . . ."; this would be unacceptable to many workers in the field.

But these are minor points. The book is for patients and it is informative and readable. The philosophy expressed in the dedication deserves emphasis.

W.B.M.

SYNOPSIS OF PEDIATRICS. By John Zahorsky, M.D., and T. S. Zahorsky, M.D. Sixth edition. 470 pages. Illus. Price $7.50. St. Louis: C. V. Mosby Co., 1953.

This book covers the whole field of pediatrics in condensed form in its 470 pages. It is amazing how much information on children's diseases the authors have been able to include in this handbook of fairly small size. The most important technical procedures in diagnosis and treatment have been intelligently selected and discussed with authority. Especially valuable are many important personal observations and advice on pediatric practice in the patient's home and in the doctor's office. The book is well illustrated with many vivid photographs, tables and graphs.

The authors have tried to keep the book up to date as much as possible. Probably the text could be made still more concise by omitting some obsolete treatment methods (e.g. for syphilis), which now have mainly historical significance, and present only modern methods. The book is printed with small print which might be a handicap for readers with impaired vision. A larger print in future editions would be an asset.

The authors have written this book especially for the use of medical students to give them in a concise form all the important aspects of various pediatric problems. The book is particularly helpful for examinations and quick references; larger pediatric textbooks should be consulted for detailed references.

This book can be recommended also to general practitioners because the authors give excellent advice on many practical questions encountered in pediatric practice and cover all the essential points in the diagnosis and treatment of various diseases of infants and children.

N. E. A. Leppo, M.D.

SURGERY OF THE BILIARY TRACT, PANCREAS AND SPLEEN. Charles B. Puestow, M.D., Ph.D. (Surg.) Clinical Professor of Surgery, College of Medicine and Graduate College, University of Illinois: Attending Surgeon, University of Illinois Research and Educational Hospitals; Chief of Surgical Service, Veterans Administration Hospital, Hines, Illinois; Chief Surgeon, Henrotin Hospital, Chicago. 370 pages. Illus. Price $9.00, cloth. Chicago: Year Book Publishers, 1953.

This handbook of operative surgery is a well-written, easily read and understood book which contains many excellent and properly placed illustrations. Throughout the book, all illustrations have the explanatory notes placed on the page opposite the illustration making for easy access to them.

The book is divided into four main sections:

I. The Liver
II. Extrahepatic Biliary System
III. The Pancreas
IV. The Spleen

Each of these is subdivided into Anatomy and Physiology, Surgical Conditions, Differential Diagnosis and Surgical Techniques. Preoperative and postoperative care, as well as types of anesthesia, are well covered.

This book, as stated in the preface, is not intended for the untrained or inexperienced physician who might wish to perform operations for which he is not adequately prepared, but offers a wealth of material for one who is obtaining such training or experience under adequate supervision. It is also an excellent review and refresher course for a busy older surgeon who may find it difficult to keep up with all the vast amount of literature.

N. E. Tosseland, M.D.

ARSENIC POISONING

(Continued from Page 210)

mucous membranes and are stored in the liver, kidney, walls of the gastrointestinal tract, the spleen and lungs. Deposition in the hair and bones starts within two weeks and stays fixed for years. It is excreted in the urine and feces, within two to eight hours and up to seventy days after repeated administration.[1-3]

While several case reports on the use of this medication in allergy with toxic results have appeared, it is evident that many more must go unrecognized.[4,5,6] This case further well illustrates the medical dictum that all that wheezes, and sneezes and itches is not allergy, not even in the allergic patient; and that potent medication should be prescribed only under constant and rigid supervision.

References

1. Althausen, T. L., and Gunther, L.: Acute arsenic poisoning. J.A.M.A., 92:2002 (June 15) 1929.
2. Gay, F. S., and Gay, E. D.: The Gay treatment of asthma. Mississippi Doctor, 29:142 (Nov.) 1951.
3. Goodman, Louis, and Gilman, Alfred: The Pharmacological Basis of Therapeutics. New York: Macmillan Co., 1941.
4. Harris, Robert O.: Arsenical dermatitis (Correspondence). J.A.M.A., 151:847 (Mch. 7) 1953.
5. Pascher, F., and Wolf, J.: Cutaneous sequalle following treatment of bronchial asthma with inorganic arsenic. J.A.M.A., 148:734 (Mch. 1) 1952.
6. Silver, Alfred S., and Wainman, Philip L.: Chronic arsenic poisoning following use of an asthma remedy. J.A.M.A., 150:585 (Oct. 11) 1952.

585 40th Avenue N.E.
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GASTRIC SARCOMA

(Continued from Page 220)

7. Kushlan, S. D.: Primary lymphosarcoma of the stomach with five-year survival after operation. Clinical x-ray and gastroscopic features. Gastroenterol., 16:250-258 (Sept.) 1950.
8. Laube, P. J.: Leiomyosarcoma of the stomach. Am. J. Surg., 80:249-252 (Aug.) 1950.
9. Lazarus, Ely E.: Lymphosarcoma of the stomach; an autopsy report. Bull. N. Y. Med. College, Flower & 5th Ave. Hospitals, 9:75-80 (Oct.) 1946.
10. Madding, G. F., and Walters, W.: Lymphosarcoma of the stomach. Arch. Surg., 40:120, 1940.
11. Marks, J. H.: Leiomyosarcoma of the stomach with excavation of the center of the tumor. Am. J. Roentgenol., 67:76-79 (Jan.) 1952.
12. Marshall, S. F., et al: Sarcoma of the stomach. Ann. Surg., 131:824-837 (June) 1950.
13. Marvin, C. P., and Walters, Waltman: Leiomyosarcoma of the stomach. Arch. Surg., 57:62-70 (July) 1948.
14. Maas, Gen. Hosp. Case 37272: Leiomyosarcoma of the stomach. New England J. M., 245:30-32 (July 5) 1951.
15. O'Donoghue, J. R., et al: Primary lymphosarcoma of the stomach. Am. J. Surg., 74:174-179 (Aug.) 1947.
16. Pack and McNeer: Sarcoma. Ann. Surg., 1935.
17. Perkel, L. L., et al: Reticulum cell sarcoma of the stomach; report of case in a young woman. Gastroenterol., 15:505-529 (July) 1950.
18. Pontius, Guy V., and Witkowski, Leon J.: Lymphosarcoma of the stomach. Surg. Clin. North America, 32:123-133 (Feb.) 1952.
19. Rabinovitch, Jacob; Grayzel, David; Sawyer, Alfred J., and Pines, Bernard: Sarcoma of the stomach. Am. J. Surg., 80:550-555, 1950.
20. Schindler, E., et al: Leiomyosarcoma of the stomach. Surg., Gynec. & Obst., 82:1-14 (March) 1946.
21. Shepherd, J. A.: Perforation of leiomyosarcoma of the stomach. Brit. J. Surg., 37:479-481 (Apr.) 1950.
22. Sønder, Palle: Sarcoma ventriculi. (Gastric sarcoma.) Ugesk. Laeger, 112:702-705, 1950.
23. Spencer, F. M., et al: Sarcoma of the stomach. Cleveland Clin. Quart., 14:282-295 (Oct.) 1947.
24. Turcot, J.: Sarcoma of the stomach. Canad. M. A. J., 66:252-255 (March) 1952.
25. Wolman, B.: Sarcoma of the stomach simulating splenic tumor; case. Brit. J. Surg., 34:322-323 (Jan.) 1947.

NUMBERS SYSTEM

How many times have you been at a baseball game or a movie when a physician is called away for an emergency? To allow discreet paging of doctors at large gatherings and eliminate any unnecessary embarrassment on the doctor's part, the Medical Society of Milwaukee County has activated a new system. Each member has been assigned a numbered card which is to be used by members of his family, his hospital, the Medical Society Physicians' Service or his office when it is necessary to page him while he is in a public place or at a public gathering.

"Instead of 'Dr. Smith, please call your office,' you will soon hear 'Dr. 464, please call your office,'" writes Dr. Robert S. Irwin, Milwaukee County president in the *Milwaukee Medical Times*. He feels that all society members will welcome the new system.

Minnesota Medicine

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CANCER DETECTION

A Report from the Cancer Detection Center, University of Minnesota

CLAUDE R. HITCHCOCK, M.D., and J. BRADLEY AUST, M.D.
Minneapolis, Minnesota

THE GOAL of the Minnesota Cancer Detection Center has been to develop a workable and accurate method of cancer examination including all internal organs as well as readily accessible external organs. Until a simple serodiagnostic or biologic test is available, such an effort is the only available means of detecting malignant disease in its early stages.

"Cancer Detection" implies the process of finding malignancies before symptoms or signs herald their presence, in addition to the diagnosis of precancerous conditions. Through the application of a well conceived routine plan of examination in doctors' offices, we believe several types of serious malignancy can be identified in advance of their usual signs and symptoms. More important than this, the 24 per cent of our population over the age of forty-five years who harbor a precancerous condition can be alerted for close observation or therapy.

During the five years from March 1, 1948, to February 28, 1953, cancer detection examinations have been performed on 5,352 men and women. Ninety-one cancers have been discovered at the Detection Center, and proved with biopsy or surgery. Treatment has been under the direction of the patient's personal physicians. Furthermore, a total of 1300 lesions considered to be precancerous have been detected and reported; an incidence of twenty-four per one hundred people examined over forty-five years of age. Table I demonstrates the ratio of new patient examinations and the incidence of malignancies and precancerous lesions found.

TABLE I.

	Male	Female
New Patients	2665	2687
Recheck Exams	3005	2986

Total Examinations	11,343
Total Patient's Examined	5,352
Cancer Incidence	17 per 1000 patients examined (1.7%) 7.9 per 1000 examinations performed
Precancerous Lesions	1300 (24%)

In order to facilitate the study and assure maximum cancer case finding a lower age limit of forty-five years was established. Recently the lower age limit for males has been raised to fifty years. The high incidence of carcinoma of the cervix in women in the fifth decade indicated the advisability of maintaining the lower age limit for females at forty-five years.

It is obvious that all people have *some* symptoms during any given interval of time. We have not been able to fulfill the original concept of obtaining a group of completely asymptomatic examinees for cancer detection observation. Therefore, our examinees with malignant disease have been separated into symptomatic and asymptomatic cases in reference to the particular organ or organ system in which the malignancy was found. In the group of ninety-one patients found to have cancer, forty-six patients had no symptoms referable to the organ or organ system in which the malignancy was detected and forty-five

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Sponsored by the Council of the State Medical Society and Supported by a grant from the Minnesota Division of the American Cancer Society.

Dr. Hitchcock is Director, Cancer Detection Center, University of Minnesota, and Dr. Aust is Clinical Cancer Trainee, National Cancer Institute, National Institute of Health.

TABLE II. VALUE OF REPEATED RECHECK EXAMINATIONS IN DETECTING EARLY CANCERS

	Asymptomatic	Symptomatic
New	22	41
Recheck	*24	4

*Note that over half of the lesions that were asymptomatic were found at recheck examination.

TABLE III. CANCERS FOUND IN FIVE-YEAR STUDY AT CANCER DETECTION CENTER

Site	Asymptomatic	Symptomatic
Stomach	6	5
Colon and rectum	13	5
Small bowel	1	0
Lung	0	3
Breast	6	1
Ovaries	2	3
Cervix	5	2
Uterus	0	2
Prostate	3	4
Skin	7	15
Thyroid	1	0
Lymphosarcoma	1	0
Leukemia	1	5
Total	46	45

patients had symptoms referable to the organ or organ system involved. It will be shown later that survival figures are significantly better in the asymptomatic group. Table II demonstrates the value of the recheck examination in detecting asymptomatic cancers.

Table III portrays the cancers found in the first five years of operation, according to site and relationship to symptoms.

Method of Examination

Principles of completeness and economy have been important considerations in developing the pattern of the examination. Laboratory procedures have been reduced to bare essentials, requiring only a minimum amount of technical assistance.

A detailed history form is completed by the patient prior to his visit to the Center. The patient brings a urine and stool specimen (collected after three days on meat free diet) the morning of the examination. A routine urinalysis is performed (urine sugar, albumin, microscopic for cells if urine is positive for albumin). A benzidine test on the urine has been tried, but is not as accurate as microscopic cell study to detect occult urinary bleeding. The stool is examined for occult blood with the Guaiac and Benzidine test. Routine examination of the blood includes hemoglobin determination, white blood cell count, differential count and cell morphology.

Every patient has a Levine tube passed for gastric analysis using one dose of histamine (0.5 mgm.) as stimulant. The standard sodium hydroxide and Toepfer's reagent determination for free HCl has proved most satisfactory. The Quininium ion urinary excretion test has been tried and proved too time consuming in the laboratory for large numbers of determinations daily.

Chest x-rays are taken on each patient. Suspicious lesions or single nodules are further investigated with planigrams of the involved area. Sputum is studied for malignant cells by the Papanicolaou method. If no diagnosis is forthcoming, bronchograms, bronchial washings, and bronchial biopsy (if possible) are recommended.

All patients come to the Center prepared with enemas for a proctosigmoidoscopic examination. The scope is passed to the 25 cm. level, unless peculiar conformity of the bowel (acute angulation) or stricture prevents passage of the instrument. The following conditions are checked for: (1) small adenomas (2-3 millimeters); (2) polyps (sessile or pedunculated); (3) character of the mucosa (bleeds easily, inflamed, small ulcerations); (4) diverticula, diverticulitis; (5) hemorrhoids, fissures, fistulas; (6) frank ulcerating carcinomas or intramural tumors. Excision biopsy and fulguration is recommended for all small adenomas and polyps. During the digital examination of the rectum, the prostate is palpated for hypertrophy or nodules.

All women receive a pelvic examination including bimanual palpation of adnexal areas, and direct visual inspection of the vaginal vault and cervix. Any adnexal mass is considered suspicious in this age group, and pelvic laparotomy advised. Suspicious findings in the cervix includes: (1) rough area on lip of cervix; (2) polyps; (3) blood at the os, not menstruating); (4) atrophic cervical mucosa; (5) erosion of the cervix; (6) cervicitis.

Papanicolaou smears are made of vaginal secretions and scrapings of the cervix. Suspicious areas are biopsied. Positive Papanicolaou smears must be confirmed later by positive biopsy. Suspicious (Class III) smears, in an otherwise normal vagina, indicate need for repeated cytologic examination every six weeks until smears are negative or biopsy proves the presence of a carcinoma.

After the laboratory work has been completed and the proctoscopic and pelvic examinations performed, a clinician examines the patient for the remaining problems:

1. Nose and throat with visual inspection of nasal cavity, oral cavity, and auditory canal. Inspection of the nasal pharynx and larynx with laryngeal mirror. Palpation of the neck and submandibular area for palpable lymph nodes. The thyroid gland is palpated for adenomas or masses.
2. Skin (senile keratoses, and pigmented nevi in areas of irritation are classified as suspicious). Any recently darkening or enlarging nevus is considered suspect for malignant melanoma.
3. Adenopathy is searched for in supraclavicular, axillary, and inguinal areas. The chest x-ray will detect mediastinal adenopathy.
4. Breasts are examined by inspection and palpation in both the sitting and supine position.
5. Abdominal palpation is important for detection of renal masses. Liver, spleen, gall bladder, enlarged lymph nodes, or any other masses are carefully checked.
6. External genitalia of the males are examined to include palpation of testes.
7. Peripheral muscle tissue and skeletal system are examined for deformities or lesions.
8. Brief neurological examination for possible manifestations of cerebral neoplasms.

Criteria for Special X-Ray Studies

While summarizing the results of examination, the clinician determines whether further x-ray studies are needed according to the following plan.

Gastric Intestinal Series X-Rays are Ordered for:

1. Patients with histamine achlorhydria and hypochlorhydria (less than 30° free acid).
2. Patients with occult blood in the stool.
3. Patients with strong family history of gastric carcinoma.
4. Patients with unexplained hemoglobin level below 11 grams.
5. Patients with vague symptoms of gastric pathology which in any sense could be interpreted as being due to gastric carcinoma by the examining physician.
6. Any evidence of undue weight loss.

The air contrast technique is helpful in cases of suspected gastric polyps or small mucosal defects.

Barium Enema Colon X-Rays are Indicated for:

1. Patients with abnormal findings on proctoscopic examination.
2. Patients with occult blood in the stool.
3. Patients with unexplained hemoglobin level below 11 grams.
4. Patients with a family history of large bowel malignancy.
5. Patients who have vague symptoms that could possibly be due to malignancy of the large bowel as determined by the examining physician.
6. Any evidence of undue weight loss (10 pounds or more).

TABLE IV.
COMPARISON OF GASTRIC CANCER PATIENTS

	Asymptomatic	Symptomatic
Total Number	6	5
Achlorhydria	6	5
Occult Blood	1	1
X-Ray Diagnosis (1st Visit)	1	2
X-Ray Diagnosis (Subsequent Visits)	5	3
Scirrhous	1	3
Polypoid	5	1
Ulcerating	0	1
Positive Lymph Nodes	1	4
Alive	3 (1½, 3, 5 yrs.)	1 (1½ yr.)
Deceased	3 (1¼, 1/12, 1 yr.)	4 (1/12, ⅓, ½, 1 yr.)

*Survivals are considered as of December 31, 1953.

In addition to routine barium enema x-rays, all patients found to have polyps on proctoscopic exam are given air contrast barium enema studies.

Intravenous Pyelogram X-Rays are Ordered for Patients with:

1. Blood in the urine.
2. Costovertebral pain or tenderness.
3. Palpable mass in region of kidney.

Results of Five Years' Operation

Esophagus and Stomach.—Eleven gastric carcinomas have been found, seven in males and four in females. All patients had achlorhydria and would have been been picked up by x-ray performed on this basis alone exclusive of other indications for performance of a G.I. series. In only two cases (with symptoms) was the hemoglobin below 11.0 grams. Occult blood was present in the stool of only one asymptomatic patient and one patient with symptoms. Table III demonstrates that in six patients the gastric cancers gave no symptoms, while in five patients symptoms were present.

Polypoid type carcinomas were found in *five* of the *six asymptomatic* patients and scirrhous carcinoma in one patient. In the *five* gastric cancer patients with symptoms, *four* had scirrhous carcinoma, and only one had a polypoid lesion. Table IV demonstrates the results of early diagnosis (asymptomatic patients) compared with late diagnosis (symptomatic patients) in the gastric cancer group. Three of the patients are living and well up to five years in the asymptomatic group. There is only one survivor in the group of patients with symptoms at time of diagnosis.

Early polypoid lesions have a good chance for cure with subtotal gastrectomy. Since virtually 100

TABLE V. RECTAL AND COLON CANCERS

	Asymptomatic	Symptomatic
Total Number	13	5
Diagnosed first visit	11	5
Occult blood	2	3
Hemoglobin below 12.5	0	1
Proctology positive	10	4
Barium enema	3+ (also 7 of 10 procto pos.)	3+ (also 2 of 4 procto pos.)
Ulcerating cancer	2	4
Polypoid cancer	11	1
Lymph nodes positive	2	3
Alive	11 (2⅙, 2½, 4, 3⅓, 4¾, 2¾, 1¾, 3¼, 5¼, 4¾, 4¾ yrs.)	1 (2¾ yrs.)
Deceased	2 (1⅔, 2 years after CVA)	4 (1/12, 7/12, 1⅙, 2½)

*Survivals are considered as of December 31, 1953.

per cent of polyp-bearing stomachs are achlorhydric, the gastric analysis seems to be a satisfactory method for indicating those patients with a likelihood for polyps. Polyps greater than 2 cm. in diameter have a 50 per cent chance of being malignant when found. Of those 1 cm. or less in diameter when detected about 11 per cent are malignant.

For early asymptomatic gastric cancers the most significant criterion indicating need for gastrointestinal series x-rays is achlorhydria. We do not know of a stomach cancer that has been found during this five years among the three-fourths of our patients who have normal gastric acidity.

One examinee presented during the first six months of operation of the Center with total obstruction of the esophagus due to known esophageal carcinoma. He was too ill to undergo the examination and is not counted in the series of cancers picked up in the Center. This was the only esophageal cancer seen during the first five years of function of this Center.

Colon and Rectum.—Eighteen cases of cancer of the rectum and colon have been detected: Ten in males and eight in females. Fourteen of the lesions (77 per cent) were within reach of the sigmoidoscope. Thirteen of these fourteen were in the 9 cm. to 16 cm. level. Three lesions were in the ascending colon or cecum and one was in the descending colon.

Of the eighteen patients, thirteen were free of symptoms for G.I. tract disease and five had symptoms referable to the G.I. tract. Only two of the thirteen asymptomatic patients had occult blood in the stool; three of the patients with symptoms had occult blood. Here again, for early lesions still not causing symptoms, the stool occult blood determination is not a satisfactory single screening device for indicating necessity of x-ray examination.

Routine examination of the rectum and sigmoid through the sigmoidoscope has proved to be the most useful screening device for large bowel lesions. Table V compares the colon and rectal cancers with and without symptoms. Of the group of thirteen symptom-free patients, eleven are living and well up to five years (seven over three years). Only one of the five patients who had symptoms referable to the colon at the time of diagnosis is living and well (two and three-fourths years).

There is no etiologic relationship between diverticulitis and carcinoma of the colon. However, inflammatory strictures due to diverticulitis often cannot be differentiated from constricting adenocarcinoma by x-ray studies. Exploratory laparotomy and resection of the involved area should be performed for potentially obstructing inflammatory lesions, thereby circumventing the risk of unsuccessful differentiation of the benign from the malignant process.

Female Pelvic Organs.—Seven cancers of the cervix have been found and proved. Five women were without symptoms and two had symptoms of pelvic disease when examined. Two women in the asymptomatic group had negative Papanicolaou smears, but suspicious areas noted during inspection of the cervix indicated the need for biopsy. In only one case (asymptomatic) was the cervix entirely normal in appearance and the positive Papanicolaou smear was the specific indication for biopsy which proved the presence of the carcinoma.

A total of 7,940 Papanicolaou studies have been performed. This includes both vaginal and cervical smears. Ninety-eight Class III (suspicious) reports were made, and ten Class IV (malignant cell) diagnoses were made. Seven of the above 108 patients had squamous cell cancer proved and treated. It is possible that an indeterminate number of the patients in whom a suspicious Papanicolaou has been reported, will eventually prove to have cervical carcinoma.

However, it must be emphasized, that visual inspection of the cervix with biopsy of any suspicious areas provides an excellent chance of detecting lesions early; particularly when such examinations are repeated at yearly or six-month intervals.

Endometrial Carcinoma.—Two endometrial carcinomas have been found, and both had symptoms and signs of disease (abnormal spotting). Papanicolaou smears were negative. Diagnosis was established by dilatation and currettage. Both patients are living and well after surgery. No good screening technique is yet available for *early* endometrial carcinoma. Bleeding and discharge from the cervical os, and uterine enlargement remain well-proved danger signs.

Ovarian Carcinoma.—Of forty-seven women who reported back to their physicians with a diagnosis of suspicious adnexal masses, five proved to have carcinoma of the ovary. Four of these patients were asymptomatic and one had symptoms. All were detected by means of bimanual palpation. In every case the surgical specimen revealed early cystadenocarcinoma of the ovary without spread to other tissues. All patients are living and well. Pelvic examination is the only method for detection of these early ovarian tumors. Laparotomy is necessary to establish the nature of a palpable mass.

Prostate.—Thirty-two patients were reported to have prostatic nodules suspicious for carcinoma. Twenty-one had resection or biopsy of the lesion, and seven carcinomas were proved histologically. We have accepted only histologic proof of malignancy, as against clinical impression. Nine patients have been treated for carcinoma of the prostate on clinical evidence only. It is curious that of all human malignancies, the diagnosis of prostatic cancer is commonly accepted on clinical evidence alone, whereas histologic or cytologic proof is demanded for other forms of cancer.

Three of the seven proved cases were asymptomatic, and four had symptoms of prostatic obstruction. Two of the symptom-free patients had bony metastases, as well as two of the patients with symptoms. One out of the four symptomatic patients is dead, and two of the three asymptomatic patients are dead. No satisfactory screening test is available for prostatic cancer. Unfortunately, widespread bony metastases are found in many patients at the time symptoms of prostatic obstruction appear. It is only conjectural to assume that repeated examination will significantly alter the outcome of prostatic cancer.

Skin.—Twenty-two patients have had skin cancer diagnosed at the Center. Fifteen of the lesions were basal cell carcinomas, and six were squamous cell carcinomas. One pigmented nevus proved to be a malignant melanoma. All of these lesions were small and localized; treatment has been successful, and all of the patients are well at this time. Fifteen of the twenty-two patients were aware of the presence of the lesion, but considered it benign and of no consequence.

Senile keratosis of the skin is considered a pre-malignant lesion and 183 patients have been found with keratoses. A total of 240 pigmented nevi, exposed to chronic irritation, have been reported, but only the one malignant melanoma has been demonstrated. No problem attends the diagnosis of skin cancer when clinicians are quick to submit biopsies of all suspicious lesions for histologic evaluation.

Lung.—Only one primary carcinoma of the lung was found in the first five years of function of the Detection Center. This lesion was far advanced and the patient had obvious symptoms of pulmonary malignancy. Two metastatic carcinomas were picked up, one a metastatic melanoma (not our melanoma patient) and the other a metastatic hypernephroma. No case of asymptomatic primary carcinoma of the lung has been detected, but one examinee was misdiagnosed, and will be reported in a following section. This is an unusual circumstance, perhaps owing to the exceptionally good detection of lung lesions in the mobile unit chest survey which has been extensively used in our State. Willingness on the part of our examinees to participate in a cancer detection program indicates a likelihood that they availed themselves of the opportunity for a chest x-ray examination.

There are no pathognomonic symptoms of *early* lung cancer. When symptoms are present during the earliest *phases* of lung cancer, they are the same as those produced by common respiratory diseases. These symptoms often respond to non-specific treatment, and serious delays in accurate diagnosis and treatment are fostered. The highly lethal nature of lung cancer makes it imperative that clinicians exercise a high index of suspicion towards silent lung nodules (coin lesions) and other suspicious pulmonary changes if a significant increase in five-year survivals is to be effected.

Breast.—Ninety-two women had breast lesions considered suspicious for the presence of cancer. Seven (9 per cent) carcinomas have been proved at surgery, and seventy-seven lesions have been established as benign. Eight lesions are not yet fully diagnosed. All of these tumors were unknown to the patients at the time of examination.

Six patients are living and well, and one is dead of cerebral metastases fifteen months after surgery. At the present time periodic physical examination of the breasts is the only proved method of detecting this cancer.

Urinary Tract.—The kidney has been a difficult organ to survey with detection techniques. Routine urinalysis for detection of urinary tract bleeding has not been adequate as an indication of the need for advanced roentgen studies. Most kidney lesions are large enough to be palpated during physical examination before they have invaded the renal pelvis and caused urinary tract bleeding. The Detection Center missed the diagnosis in two patients who subsequently developed adenocarcinoma of the kidney. In the absence of an adequate screening technique, and with widespread use of roentgen pyelogram unfeasible, we probably will not improve in diagnosing these patients in an asymptomatic stage of the disease. No testicular or penile cancers have been found.

Lymphatic System.—Of six patients found to have lymphatic leukemia, four had palpable lymph nodes at the time of examination. Only one was asymptomatic. Five had symptoms of a mass, pain or weakness. There were forty-six patients with a lymphocyte count above 45 per cent, and thirty-nine patients of this group have not developed a lymphoma to the present time. Four of the six leukemia patients are alive up to five years after treatment.

Thyroid.—One papillary adenocarcinoma of the thyroid was proved at surgery, and one lymphosarcoma of the thyroid was found. These glands were diagnosed as the sites of suspicious nodules. A total of eighty-six thyroid adenomas have been reported. Thirty-five of these have been surgically excised. We believe about seven per cent of palpable, single, thyroid nodules will prove to be malignant (University of Minnesota study).

TABLE VI. PRE-CANCEROUS LESIONS

Total Number	1300
Adenomas and polyps of rectum and colon	624
Gastric polyps	6
Kraurosis vulvae	16
Leukoplakia	144
Pigmented nevi (areas of irritation)	240
Polyps of vocal cords	1
Senile keratosis	183
Thyroid adenomas	86

Pre-Cancerous Lesions.—Table VI presents eight lesions considered to be pre-cancerous and the number of patients detected with these conditions. It is impossible to calculate the potential salvage in this large segment of the over-all group (24 per cent) through *prevention* of malignancy by application of appropriate therapy during the pre-malignant stage of such lesions.

Known Failures of the Detection Center Program During Five Years Operation.—It is to be expected that a few cancers might arise in such a manner that detection center procedure would not screen out the lesion, or that failure of an examinee to co-operate would result in late diagnosis. Furthermore, an occasional misinterpretation of roentgenograms might result in undue delay in establishing the histologic nature of a questionable lesion. Table VII presents nine examinees who developed cancer during the interim period between examinations, and were not picked up at the last negative examination.

Two patients with carcinoma of the kidney were asymptomatic at examination two months and ten months prior to diagnosis. Urinalyses were negative, and abdominal palpation was negative; criteria indicating the need for intravenous pyelograms were lacking. Since intravenous urography is not feasible as a routine procedure, and since most renal neoplasms in adults attain considerable size before invasion of the renal pelvis causes symptoms or signs of disease, a better method of screening for renal neoplasms is urgently needed. These patients demonstrate an inadequacy of the present examination procedure for renal lesions.

The gastric cancer appeared in a patient who previously had a subtotal gastrectomy for duo-

TABLE VII. KNOWN DETECTION CENTER FAILURES DURING FIVE YEARS OPERATION

	Time from CDC Exam. to Positive Diagnosis	Conditions
Kidney	2 months (surgery)	Urine negative. Prostate questionable. Recheck by LMD disclosed renal lesion. Failure of examining method.
Kidney	10 months (surgery)	Examination and urinalysis at CDC negative. Failure of examining method.
Stomach (carcinoma)	25 months (surgery)	Subtotal resection 1923 for duodenal ulcer. X-rays and exam. at CDC negative. Patient refused follow-up exams by CDC & P.P.
Sigmoid (carcinoma)	4½ months (surgery)	Exam. at CDC diverticulitis. Exam. by LMD same. Then developed bleeding and procto biopsy positive. Failure of examining method.
Left Lung (carcinoma)	18 months (surgery)	Jan. 1949—apical lesion seen here. Diagnosed fibrosis. Jan. 1950—diagnosed same. Summer 1950—carcinoma.
Tonsil (undifferentiated carcinoma)	8 months (biopsy)	Negative exam. at CDC 8 months prior to symptoms.
Neck (lymphosarcoma)	8 months (died)	No indications 6 months prior to death.
Prostate (carcinoma)	14 months (died)	Previous trans-urethral resection for prostatism, 1946. Died of heart disease; cancer prostate incidental finding.
Unknown (abdominal carcinoma)	14 months (died)	1949—Papanicolaou Class III—biopsy of cervix negative. Exam. 1950 negative. Failure recheck appointment.

denal ulcer. Gastrointestinal series x-rays were negative for tumor at the time of the CDC examination. The examinee failed over a twenty-five-month period to return to either the Cancer Detection Center or his own physician for check-up. It is reasonable to assume that the gastric malignancy developed during that period of time, for the silent interval of most gastric cancers is in the nature of twenty to twenty-two months. Patient failure appears to be the responsible factor in this instance.

The carcinoma of the sigmoid represents a failure to correctly interpret the colon roentgenograms. A diagnosis of inflammatory stricture secondary to diverticulitis was made at the Cancer Detection Center, and confirmed with further x-ray studies by the family physician. An episode of bleeding shortly after the second x-ray examination led to biopsy of the area through a sigmoidoscope and the maligant character of the constriction was established. The roentgenologist must exercise caution in attempting to differentiate between lesions of the colon causing stricture. Laparotomy with excision of the diseased part is the safest course to follow in any case of large bowel stricture.

The lesion of the left lung was noted on two chest films taken at the Cancer Detection Center one year apart. Six months following the second diagnosis of pulmonary fibrosis the patient developed symptoms, and chest x-ray showed progression of the lesion. Thoracotomy then established the malignant character of the tumor. Failure to strongly advise thoracotomy rests with the Detection Center. Pulmonary lesions must be established histologically at thoracotomy if sputum studies and bronchoscopic biopsy fail to establish the diagnosis. Undue delay is particularly tragic with this lethal carcinoma.

One carcinoma of the tonsil and one lymphosarcoma of the neck appeared eight months after a negative examination at the Detection Center. There was no evidence of tumor in either patient at the time of examination at the Detection Center. Another patient was found to have carcinoma of the prostate as an incidental autopsy finding following death due to cardiac disease. The last patient died of carcinomatosis fourteen months after a negative examination. The source of the primary tumor was obscure at autopsy.

A recent survey of examinees who failed to return for recheck examinations has not brought other cases of "missed cancers" to our attention. This latter group represents 9 per cent of the hundred cancers known to be present in the entire five-year period.

Of the ninety-one cancers correctly diagnosed in the Cancer Detection Center, thirty-two were established by roentgenograms of one kind or another. Only two of the "missed" cases were examined with x-ray methods and these constitute 6 per cent failure for roentgen ray diagnosis during the five years of operation of the center. This compares favorably with diagnostic experience in the x-ray department of the University of Minnesota Hospitals. All roentgen diagnostic methods have specific limitations, and clinicians responsible for patients must appreciate the necessity of bringing other more specific methods (microscopic examination of tissue) to bear on questionable cases.

Sixty-nine of the cancers correctly diagnosed were found either during physical examination or with a laboratory test. If we consider six of the "missed" lesions as failures of physical diagnosis, this represents 8 per cent inaccuracy as far as missing malignancies is concerned. It must be remembered, however, that many lesions were reported as suspicious (prostate, ovary, breast, thyroid) and proved to be benign. Again, the shortcomings of gross palpation or inspection are obvious, and the necessity for histologic and cytologic verification is amplified.

Summary

1. During five years' operation of the Cancer Detection Center at the University of Minnesota ninety-one malignancies were found in 5,352 examinees. A total of 11,343 examinations were performed. This represents seventeen cancers per 1,000 patients examined (1.7 per cent), or 7.9 cancers per 1,000 examinations performed.

2. Forty-six examinees with cancer were symptom free for the organ or organ system in which the lesion was found. Forty-five had symptoms indicating a likelihood of cancer in the organ or organ system involved. In the asymptomatic group twenty-four cancers were found at recheck examination, indicating the value of repeated examinations to detect malignancies in their earliest stages.

3. Only in cancer detection centers, or cancer prevention clinics are large numbers of persons surveyed for cancer while they are without symptoms of disease. It seemed of value to emphasize the difference in survival between symptomatic and asymptomatic examinees found to have cancer in this study.

4. Three of six asymptomatic gastric cancer patients are alive and well up to four years after surgery. There is one survival among five gastric cancer patients who were symptomatic at the time of diagnosis. Of thirteen patients with colon cancer who were free of symptoms at time of diagnosis, eleven are living and well (seven over three years). Only one of the five colon cancer patients with symptoms at time of diagnosis is living (two years). These results appear to substantiate the increased value of early diagnosis in terms of survival.

5. Twenty-four per cent of our patients had pre-cancerous lesions. The value of treatment of these lesions in the prevention of cancer is incalculable.

6. Of all cancers developing in our examinees during this five years, 9 per cent are considered failures of the Detection Center method. Roentgen ray techniques failed in 6 per cent of the thirty-four patients in whom the diagnosis was established by this modality. Misinterpretation of x-ray changes was the responsible factor. Of the cases detected by means of physical examination and laboratory studies 8 per cent must be considered as failures for this method.

7. We believe that several types of serious malignancy can be identified in advance of their usual signs and symptoms through the application of a well conceived, routine, cancer detection examination performed at yearly intervals.

References

1. Ballin, John C.; Feinstein, Robert N., and Warner, Nancy E.: Clinical evaluation of the blood cell carboxypeptidase inhibitor test for malignant neoplasia. Cancer Research, 13:784-788 (Nov.) 1953.
2. Berkson, Joseph, and Comfort, Mandred: The incidence of development of cancer in persons with achlorhydria. J. Nat. Cancer Inst., 13:1087 (March) 1953.
3. Chipps, H. Davis: The Washington state tumor registry and its role in problems regarding gastric cancer and cancer research. J. Nat. Cancer Inst., 10:439-441 (Oct.) 1949.
4. Cooper, William A.: Abstract of Color Film: The exfoliative cytologic method in the diagnosis of gastric cancer. J. Nat. Cancer Inst., 13:1047 (March) 1953.
5. Cromer, Horace E. (Jr.); Comfort, Mandred W., and Butt, Hugh R.: Gastric acidity in cases of adenomatous gastric polyp. J. Nat. Cancer Inst., 10:497-503 (Oct.) 1949.
6. Gardner, Eldon J.; Woolf, Charles M., and Shaffer, J. Ordie: The inheritance of cancer involving different areas of the human gastrointestinal tract. J. Nat. Cancer Inst., 13:1089-1093 (March) 1953.
7. Guiss, Lewis W.: Value of mass chest roentgen-ray survey methods in control of lung cancer. Cancer, 5:1035-1040 (Sept.) 1952.
8. Hubbard, T. Brannon (Jr.), and State, David: Cancer detection, report from the center at the University of Minnesota. Minnesota Med., 35:925-929, (Oct.) 1952.
9. Robbins, Guy F.; MacDonald, Mary C.; Pack, George T.: Delay in the diagnosis and treatment of physicians with cancer. Cancer, 6:624-626 (May) 1953.
10. Segal, Harry L.; Miller, Leon L., and Morton, John J.: Detection of achlorhydria by tubeless gastric analysis. J. Nat. Cancer Inst., 13:1079-1086 (March) 1953.
11. Stillwell, G. G.: Tests for cancer. Minnesota Med., 36:623-624 (June) 1953.
12. Wigh, Russell, and Swenson, Paul C.: Gastric photofluorography for adenomas and cancer. Its place in a cancer detection program. J. Nat. Cancer Inst., 13:1075-1077 (March) 1953.

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FARMER'S LUNG

A Form of Bronchopulmonary Moniliasis

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IN RECENT MONTHS some instances of chronic pulmonary disease in farmers have been observed which share certain features in common. Each gave a history of uncomfortable exposure to the dust from moldy silage, each was ill for a period of months complaining of dyspnea or cough, and each improved on removal from his environment.

Case Reports

Case 1.—The first man was a dairy farmer forty-four years old, who presented himself in April, 1953, with the complaint of abdominal soreness and constipation associated with loss of ambition and a 20-pound weight loss of four months' duration. Physical examination disclosed a few palpable posterior cervical lymph nodes, and some tenderness in the epigastrium and right lower quadrant of the abdomen. The blood hemoglobin was 12.9 Gm., and the white cell count was 8,400 per cu. mm. with 77 per cent neutrophils, 12 per cent lymphocytes, 5 per cent monocytes, and 6 per cent eosinophils. The Wasserman test gave negative findings. The histoplasmin and tuberculin skin test findings were also negative. X-ray examination of the gastrointestinal tract showed only tenderness and spacticity over the duodenal bulb and colon. The chest x-ray examination revealed blunting of the costo-phrenic angles, a small amount of fluid in the right major interlobar fissure, and a mottled infiltration of both mid-lung fields extending out to the periphery. Upon questioning, the patient stated that he had been experimenting with a method of storing silage dry in the open field. He was quite disgusted with the method because every time he went out to the field storage area to get a load of silage for his cattle, he was distressed with suffocating chest pain after inhaling the dust from this moldy silage.

He also stated that two other farmers in an adjoining county had been storing silage in the same manner, and had noted respiratory distress following inhalation of the dust. They had been told at the University of Wisconsin Hospitals that they were probably allergic to something in their silage. When seen four months later, eight months after the onset of his illness, he felt improved, but he had done no farm work in the interval. The chest radiograph showed resolution of the previously noted infiltration into streaks of small nodular densities radiating out from the hilum.

Case 2.—The second case was that of a dairy farmer forty-six years old, who began to note a productive cough and shortness of breath in the fall of 1953. By the end of the year, he was unable to do any work and at times required adrenalin by injection to relieve his nocturnal dyspnea. Treatment with several different antibiotics given one after the other for two to four weeks at a time apparently gave him little relief from his symptoms. No notes concerning the physical examination during the acute phase of the illness were available from the referring physician, but when the patient was first seen in May, 1953, he was rather pinched and anxious in appearance and had a slate gray cyanosis of his skin and mucous membranes. There were no expiratory wheezes audible, but on inspiration there were many sticky and crackling râles. Blood hemoglobin was 18.5 Gms. per cent and the white cell count was 8,550 per cu. mm. with 75 per cent neutrophils, 13 per cent lymphocytes, 9 per cent monocytes, 2 per cent basophils, and 1 per cent eosinophils. The sputum was viscid and contained non-hemolytic streptococci, coagulase negative staphylococci, and some eosinophils. No fungi were demonstrated by smear or culture. Tuberculin and histoplasm skin test findings were negative. X-ray examination of the chest at monthly intervals, commencing in January, 1953, had shown a finely mottled type of density distributed widely through both lung fields. By May, 1953, there was some clearing of the widespread infiltrations without coalescence of nodules or formation of fibrous residuals. This man stated that he had not finished his new silo in the fall of 1952, and had to rely on silage left standing in the cattle barn or trucked into the barn from neighboring farms. Inhalation of the dust from this silage caused so much dyspnea and cough that he was unable to carry on through the winter with the daily chore of pitching of silage out to the cattle. This work was finally delegated to his wife who seemed to handle the job without much trouble.

Case 3.—The third case is that of a farmer, sixty-four years old, who stated that he had noted considerable respiratory distress after handling moldy hay over several winters. He felt that he was suffering from repeated upper respiratory infections. In March, 1953, he noted the onset of a cough productive of two or three ounces of thick yellow sputum daily. Dyspnea on slight exertion was severe, and there were frequent wheezing respirations. On physical examination crackling râles were noted over both lungs, and there was cyanosis of the lips and nail beds. The remainder of the examination was without note of abnormality. Blood hemoglobin was 14.7 Gm. per cent and the white cell count was 6,450 per cu. mm. with 72 per cent neutrophils and 2 per cent eosinophils. Sputum smears and cultures failed to demonstrate pathogenic bacteria or tubercle bacilli, but Candida albicans was isolated from sputum culture on three occasions and a penicillium on one other occasion. X-ray examination of the chest showed finely mottled densities scattered through both lung fields. Over a period of

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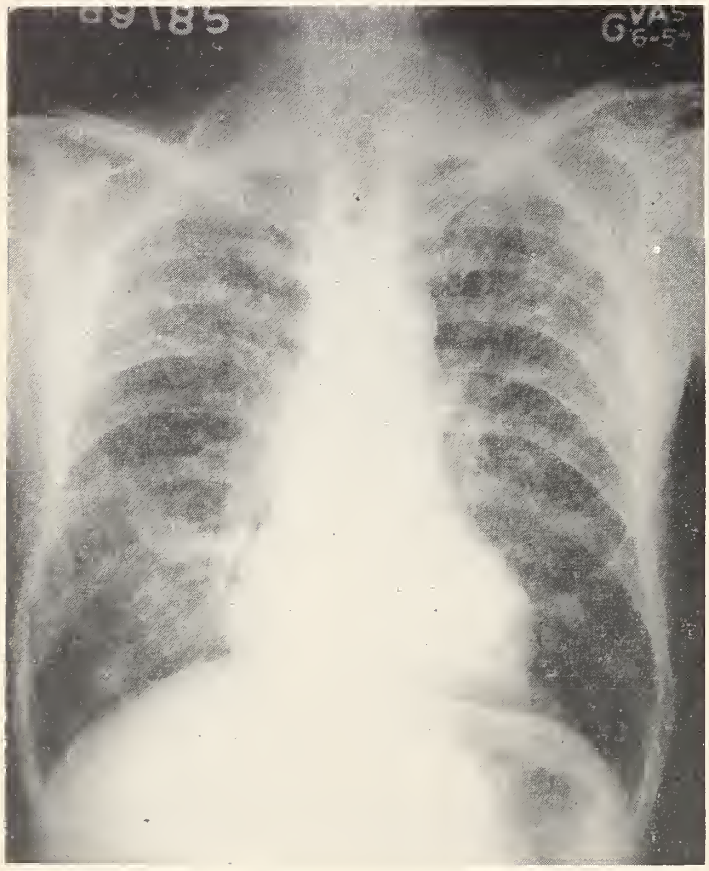
two months in the hospital, there was noticeable clearing of the diffuse infiltration, and the dyspnea was less severe than on admission.



Roentgenogram of chest in third case* after clearing of the acute pneumonia. There are widespread fibronodular infiltrations radiating from each hilum.

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Discussion

These three cases of idiopathic pneumonitis, or pneumoconiosis of farmers, had some features obviously in common, and seemed to resemble those cases of "farmer's lung" recently summarized from the British and Scandinavian literature by Fuller.[3] In England, roentgenologists have been describing an occupational pneumonitis among farmers for over twenty years.[2] They have recognized in farmers exposed to the dust of "burnt" hay or moldy grain at threshing time a bronchitis which may go on, after repeated exposure, to a kind of bronchopneumonia, and, after many years of repeated exposure, to a fibro-cavitary disease roentgenologically indistinguishable from tuberculosis. A high spore count for molds of the Fungi imperfecti was obtained from the airborne dust in either the cattle barns or from the threshing floors where these people worked. In most instances, the Monilia, Candida albicans, was recovered from the sputum of the infected workers.

Whether or not these cases represent instances of primary pulmonary moniliasis is certainly open to question. There is little doubt but that primary pulmonary moniliasis can occur, but it is extremely difficult to make the clinical diagnosis of that disease.

Castellani,[1] in 1905, described "tea-tasters cough" and pneumonitis in persons employed in the dusty tea warehouses of Ceylon. Tea-tasters crushed the dried tea leaves in their hands before sniffing them for aroma while coolie laborers carried bags of dried leaves around within the dusty warehouses. Both groups developed a peculiar cough, and it was known that laborers who became ill while working in the warehouses would recover if employed in some outside work for a time. Candida albicans was found in the dried tea leaves, was recovered from the sputum of sick men, and was made to reproduce the disease in animals. This last was accomplished by forcing a guinea pig to inhale daily over an eleven-month period the tea leaf dust contaminated with Candida albicans.

A brilliant demonstration of the pathogenicity of certain strains of Monilia was made by Dr. Kano Ikeda[4] of Saint Paul and was reported in 1936. He collected five cases of bronchopulmonary moniliasis occurring both as part of systemic moniliasis in debilitated persons as well as primary pneumonia in young individuals exposed to a dusty atmosphere. One particularly virulent strain was isolated from a young man with pneumonia and empyema due to Candida albicans. When this organism was inoculated into rabbits by the intravenous route, they died within a few days of uremia. When the inoculation was intratracheal, a bronchopneumonia developed. When the inoculation was made into the lung by a direct stab through the chest wall, the injured lung offered little resistance to infection and a severely destructive pneumonia resulted. Ikeda also noted the presence of both sub-miliary tubercles and obstructive thickening of the walls of smaller arterioles in the inoculated rabbit lung as well as the human autopsy material he examined. This is particularly interesting in the light of recent work performed by Lennart Zettergren in Sweden.[5]

In 1950, Zettergren reported on an experiment to determine the rôle played by organic dust in the causation of bronchopenumonia in persons working on threshing rigs. He found the dust from a threshing machine to be heavily infested with Candida albicans. The monilia organism was propagated on wort agar while the dust was sifted and sterilized. A chamber was then constructed in which a rabbit could be confined in an atmosphere heavily contaminated with sterile threshing dust. The first group of rabbits was exposed for an hour a day for fourteen days to the dust alone, and the lungs were then examined as the animals were sacrificed at intervals of a few weeks following exposure. A second group of rabbits was exposed to the sterile dust innoculated with the monilia culture, and the lungs were then examined in the same manner as with the first group. In both groups of animals, there was evidence early of irritative bronchitis due to the dust. Both groups showed submiliary granulomata in the neighbhood of the terminal or respiratory bronchioles. These granulomata persisted only a few weeks in the animals exposed to dust alone, but showed caseation and persisted much longer in the animals exposed to dust and the fungus. In addition, the second group of exposed animals showed thickening of blood vessel walls in the terminal arterioles, some being almost obstructed. No fungi were demonstrated in the granulomata, but numerous fat-filled macrophages were seen in the caseating granulomata.

The most interesting thing about Zettergren's descriptions of the pathologic changes in the rabbit lungs was that it was almost identical with Dr. Ikeda's descriptions of the changes in both human and rabbit lungs reported fifteen years ago, although there is no indication that Zettergren was familiar with Ikeda's report. It is apparent that the granulomata accounted for the miliary picture seen on the chest radiograph and it seems reasonable to assume that the arteriolar obstruction accounted for the pulmonary congestion and the cyanosis noted in the subacute phase of the reported cases. The hemoglobin value of 18.5 Gm. per cent in one of the reported cases and a similar elevation in an unreported case seen at the University Hospital since these cases were collected is conceivably a compensatory polycythemia in response to the chronic hypoxia caused by the arteriolar obstruction.

These cases are not presented as authenticated instances of pulmonary moniliasis. As previously stated, diagnosis is extremely difficult to make in the acute phase of the illness and is nearly impossible to make in the chronic state except by lung biopsy and tissue culture. However, these cases do fulfill the requirements for making a presumptive diagnosis of pulmonary moniliasis, each patient having been exposed to an irritating dust from a moldy source and each demonstrating the roentgenological and clinical picture that has been associated with proven cases of pulmonary moniliasis.

Ordinarily, in our temperate climate, we should not expect to find much monilial infection, but with the unusually wet field conditions of recent years, we should anticipate much greater exposure to fungi among our agricultural workers. Mycotic pneumonitis must be considered in any case of persistent pulmonary disease in farmers. Since the fungi grow so freely after antibiotic therapy in such a large percentage of people so treated, it becomes increasingly important to identify pathogens before instituting therapy for pneumonitis. In unusual cases, the bronchoscopic aspiration of secretions is essential to by-pass the usual contaminants in the upper respiratory tract when material is needed for diagnostic culture. It is to be hoped that when atypical pneumonitis in farmers is encountered in the future that interested persons may seize the opportunity to identify the causative organisms and trace them to their source. This procedure will reward the investigator because if fungi are found to be causing the pneumonitis, they will frequently prove susceptible to iodides whereas antibiotics will have little effect and may even enhance the growth of fungi.

Farmers certainly should be dissuaded from employing farm practices that result in the propagation and dissemination of moldy dust, for the pulmonary mycoses are extremely debilitating and may leave permanent residuals, if exposure is repeated.

References

1. Castellani, Aldo: Notes on certain bronchomycoses which may simulate pulmonary tuberculosis. Am. Rev. Tuberc., 16:54, 1927.
2. Fawcitt, Richard: The roentgenological recognition of certain bronchomycoses involving occupational risks. Am. J. Roentgenol., 39:19, 1938.
3. Fuller, C. J.: Farmer's lung: A review of present knowledge. Thorax, 8:59, 1953.
4. Ikeda, Kano: Bronchopulmonary moniliasis. Arch. Path., 22:62, 1936.
5. Zettergren, Lennart: Thresher's lung. Acta Soc. Med., Upsal., 40:257-315, 1950.

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UPPER GASTROINTESTINAL HEMORRHAGE

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A FORTY-FIVE-YEAR-OLD man was first seen in June, 1950, with a history of incomplete recovery from "flu." His complaints were of minor, indefinite aches and pains in the chest and epigastrium. His general appearance and health were good. He did have a past history, however, of excessive use of alcohol. The general physical examination and routine blood studies, as well as x-ray examinations of the chest, colon, esophagus, stomach, and duodenum, all gave negative findings.

Five weeks later, he became weak and faint and was admitted to his local hospital. The next day he vomited blood and had two large bloody bowel movements. During the following three weeks, he had repeated massive upper gastrointestinal hemorrhages requiring thirty-five transfusions. He was seen by many consultants from whom came a variety of opinions. His condition prevented operative treatment and he expired. A small ulcer high on the lesser curvature was disclosed at a postmortem examination. In its base there was a small ruptured aneurysm of a branch of the left gastric artery. A thrombotic mass with signs of organization was attached to the intima of the most superficial part of the blood vessel.

This case and repeated experiences with sudden severe gastrointestinal hemorrhage stimulated a study of such cases admitted to St. Mary's and St. Luke's Hospitals of Duluth during the ten-year period ending December 31, 1952.

The case histories of 424 patients were reviewed (Table I). There were 341 males and 83 females; a ratio of four to one. Approximately 30 per cent of the patients were under forty-five years of age.

The severity of the hemorrhage was arbitrarily graded mild, moderate, and massive (Table II). In mild hemorrhage, the hemoglobin range was from 100 per cent down to 75 per cent, and the red blood cell from five million down to three and one-half million. In moderate hemorrhage, the hemoglobin ranged from 75 per cent down to 50 per cent and the red blood cell count from three and one-half million to two and one-half million. In massive hemorrhage, the hemoglobin ranged from 50 per cent downward and the red blood cell count from two and one-half million on down. Seventy-one cases were graded as mild, 179 as moderate, and 174 as massive hemorrhage (Table III). Patients over forty-five years of age showed essentially the same distribution in the grades as those under forty-five years of age (Table IV). Ninety-four (22 per cent) of the cases had no record of an x-ray examination and in twenty-three (5 per cent) no abnormality could be demonstrated by x-ray examination or exploratory operation (Table V). In those with an established diagnosis, the bleeding was due to duodenal ulcer in 70 per cent, gastric ulcer in 10 per cent, and a variety of pathologic conditions in the remaining 20 per cent.

TABLE I. CASES OF UPPER GASTROINTESTINAL HEMORRHAGE REVIEWED

Males	341
Females	83
Total	424

TABLE II. RANGE OF HEMOGLOBIN AND RED BLOOD CELL COUNT

	Hbg.	RBC
Grade 1 Mild	100—75%	5 M—3.5 M
Grade 2 Moderate	75—50%	3.5 M—2.5 M
Grade 3 Severe (massive)	50— 0%	2.5 M—0 M

A review of these cases also yielded these miscellaneous observations: (1) alcohol and irregular eating habits seemed frequently to predispose to bleeding; (2) esophageal hemorrhage was usually massive and recurrent hemorrhages were common; (3) thirty-seven patients had had forty-two operations mostly for duodenal ulcer or its complications; (4) the x-ray diagnosis of gastritis and gastroduodenitis was fairly common. However, it is possible that in some of these patients the hemorrhage was due to tiny mucosal erosions not detected by x-rays; (5) less common lesions of the stomach, duodenum and jejunum should always be kept in mind. This is especially true of lesions of the jejunum and the second and third portions of the duodenum; (6) fatal hemorrhage occasionally followed reduction of fractures and other unrelated abdominal operations.

Since the treatment of these patients was carried out by many physicians, variations were common. Eighty-one patients were operated upon; seven as emergencies, sixty-six while still in the hospital but after bleeding was controlled, and eight returned to the hospital at a later date for

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From the Department of Surgery, Duluth Clinic.

TABLE III. SEVERITY OF HEMORRHAGE

Grade 1 (mild)	71
Grade 2 (moderate)	179
Grade 3 (severe)	174
Total	424

TABLE IV. UPPER GASTROINTESTINAL HEMORRHAGE BY AGE

Under 45 years			
Grade	1	2	3
Number	24 (20%)	49 (41%)	47 (39%)
			Total 120
Over 45 years			
Grade	1	2	3
Number	52 (17%)	127 (41%)	125 (41%)
			Total 304

TABLE V. LESIONS DIAGNOSED BY EXAMINATION

Duodenal ulcer	217
Presumed duodenal ulcer (previous surgery)	5
Presumed duodenal ulcer (spastic duodenum)	4
Gastric and duodenal ulcer	6
Gastric ulcer	34
Carcinoma stomach	6
Cirrhosis and varices	5
Stomal ulcers	7
Hiatal hernia	3
Banti's disease—Varices	3
Sarcoma stomach	1
Leiomyomata jejunum	2
Fibroma stomach and Meckel's diverticulum	1
Carcinoma duodenum	2
Polyp duodenum	1
Negative examination	23
No examination	94
Total	424

TABLE VI. TIME OF SURGERY

Emergency operation (2 deaths)	7
In hospital	88
Late	8
Total	81

TABLE VII. LESIONS OPERATED UPON

Duodenal ulcer	53
Gastric ulcer	6
Duodenal and gastric ulcer	2
Stomal ulcer	4
Carcinomatous ulcer	4
Banti's disease (splenectomy)	3
Leiomyomas jejunum	2
Sarcoma stomach	1
Carcinoma duodenum	2
Negative	4
Total	81

TALE VIII. SURGICAL DEATHS (SIX) IN SEVENTY-THREE CASES

1.	M (65)	Resection stomach	Blowout duodenal stump.
2.	M (62)	Resection stomach	Erosion of pancreatico-duodenal artery
3.	M (33)	Resection stomach	Evisceration—non-functioning stomach
4.	M (53)	Closure acute perforation resection stomach	Blowout duodenal stump.
5.	M (61)	Exploratory	G. I. hemorrhage—4 days postoperative
6.	M (63)	Resection stomach	G. I. hemorrhage—18 days postoperative

TABLE IX. MORTALITY ACCORDING TO TREATMENT

Operated upon (Grades 2 and 3)	65
Deaths	6
	9%
Autopsies	5
Treated medically (Grades 2 and 3)	282
Deaths	38
	13.5%
Autopsies	21

TABLE X. MORTALITY ACCORDING TO AGE

Under age 45 years	
Patients (Hemorrhage, Grades 2 and 3)	96
Deaths	6
	6.2%
Over age 45 years	
Patients (Hemorrhage, Grades 2 and 3)	252
Deaths	38
	15%

surgery (Table VI). In this group sixty-five had had moderate or massive bleeding. The majority (80 per cent) were found to have benign ulcerations of the duodenum or stomach. In 15 per cent, the lesion belonged to a miscellaneous group, mostly benign or malignant tumors. In 5 per cent, no source of bleeding was found (Table VII).

There were six postoperative deaths, a mortality of 9 per cent. Five followed resection of the stomach for bleeding duodenal ulcer. Of these, two were due to late hemorrhage, the result of erosion of the pancreatico-duodenal artery; two were the result of a blown-out duodenal stump and one patient had a non-functioning stoma and complete wound dehiscence. One death followed negative exploratory operation in an individual who died four days postoperatively from another hemorrhage (Table VIII). Postmortem examination revealed a small ulcer high on the posterior wall near the lesser curvature in the base of which was an open blood vessel, a branch of the left gastric artery.

By comparison, 282 patients with grade II and III hemorrhage were treated medically. In this group, there were thirty-eight deaths, a mortality of 13.5 per cent (Table IX).

There were six (6.2) deaths among ninety-six patients below the age of forty-five with grades II and III hemorrhage, while in the group over forty-five years with grades II and III hemorrhage, there were thirty-eight deaths, a mortality of 15 per cent (Table X). Fatal exsanguination occurred in one half of the cases in the older group. Also, as one might expect in the older age group, co-existing lesions were common. Diabetes, cirrhosis, hypertension, hypertensive cardiovascular disease, coronary sclerosis, and arteriosclerosis were frequently associated.

Discussion

A more aggressive surgical attitude is in order because, in the first place, the past decade has shown a remarkable advance in major surgery as evidenced by a mortality rate of less than 2 per cent for partial gastrectomy for peptic ulceration of the stomach or duodenum. Antibiotics, blood banks, and better anesthesia and techniques are among the factors contributing to this improvement. Secondly, medical management has yielded no comparable improvement.

When bleeding continues or recurs despite adequate medical care, surgical intervention is imperative. At operation, the abdomen should be thoroughly explored. The source of bleeding, if found, should be controlled. If it is a duodenal ulcer, the pancreatico-duodenal artery should be ligated, the ulcer excised if possible, and two-thirds or three-fourths of the stomach resected. If the source of bleeding is a gastric ulcer, two-thirds or three-fourths of the stomach should be resected, including the pylorus, the antrum, and the greater part of the lesser curvature. The left gastric vessels should always be ligated and divided. In those patients in which exploration, including duodenotomy and gastrotomy, does not reveal the source of bleeding, it is mandatory to perform a partial gastrectomy and this must again be a two-thirds or three-fourths resection with ligation and division of the left gastric artery.

Summary

A study of 424 cases of upper gastrointestinal bleeding is presented.

In 80 per cent of the cases, peptic ulceration was the cause of bleeding. In 15 per cent, there were other lesions of the stomach, duodenum, and jejunum. In 5 per cent, routine examinations or exploratory operations failed to disclose the source of bleeding. Sixty-five patients with moderate or severe hemorrhage were operated upon with a mortality of 9 per cent. The cause of death in each case was due to a failure in surgical technique or judgment.

The mortality under medical management was 13.5 per cent.

The death rate for upper gastrointestinal hemorrhage in those below forty-five years of age was 6 per cent as compared to 15 per cent in individuals over forty-five years. Exsanguination was the mode of exit in half of those cases in the older age group.

Conclusion

A more aggressive surgical attitude towards severe upper gastrointestinal hemorrhage is justified.

PSITTACOSIS REPORTED

According to the U. S. Public Health Service, psittacosis virus was recently recovered from two parrots sent to a game farm in Virginia. X-ray examination of the attendant who cared for the birds showed evidence of pneumonitis about one week after discharge from a hospital where he was treated for pneumonia. A specimen of blood from the attendant was positive 1:8 with Lygranum antigen.—Psittacosis infection was reported in three parakeets in Connectciut, one of which had been imported from Florida and two from New York City from a source which had previously been incriminated. Reports have been made of four suspected cases in human beings and of two actual cases of psittacosis, in both of which the blood showed a positive complement fixation test in a titer of 1:128.

Pursuant to information received from the Tennessee health commissioner, indicating that human cases of psittacosis in that state had been traced to contact with parakeets purchased in North Carolina, an investigation by the North Carolina State Board of Health disclosed six cases of atypical pneumonia in the family of the owner of the aviary from which the parakeets had been purchased. It was reported that a large number of persons raise psittacine birds in this area and that many cases of atypical pneumonia have occurred. Blood samples and several birds have been sent to the virus laboratory of the Communicable Disease Center.—J.A.M.A., Vol. 154, No. 12, March 20, 1954.

OBSTRUCTION OF THE SMALL INTESTINE

Survey of Cases in a Small Group Practice

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OBSTRUCTION of the small intestine is an important surgical condition, since almost certain death results if it is left untreated. Special impetus to research problems in obstruction has emanated from the University of Minnesota. Emphasis by Wangensteen on correction of electrolytic and fluid balance and early decompression with or without laparotomy has resulted in a decreased mortality.. His complete and concise summary of this subject is readily available.[1]

All of the cases of small bowel obstruction in the past twenty-five years treated only by operative means at the Mankato Clinic are presented. Major surgical procedures at both the Immanuel and St. Joseph's Hospitals totaled 15,740, and of these 257 patients were operated upon for small bowel obstruction. There were fifty-six hospital deaths in this group (Table I).

The clinical picture of intermittent crampy abdominal pain, nausea, vomiting, and distention is well known. Most patients with suspected obstruction were treated with indwelling nasal catheter drainage, abdominal hot moist packs, intravenous fluids, and analgesics. The degree of distention was evaluated periodically with abdominal x-rays and simply measuring the circumference of the abdomen with a tape. Failure to respond to this regime in twenty-four hours, as evidenced by increasing toxicity and distention, prompted operation. Patients with an obviously strangulated hernia or suspected strangulated gut were immediately operated upon. If the surgeon's description indicated compromising of the blood supply, the obstruction was noted as being of strangulated type; all others were considered simple. The exact site of the obstruction was difficult to determine. Again, the description by the surgeon of the operative findings was used as a means of classification. Duodenal obstruction was not included since this organ for the most part was obstructed at or near the pylorus by gastric or related lesions. In general, if the location of the obstruction was jejunal, it was classed as high, and if in the lower one-third of the ileum it was considered low. Admittedly, this was not too reliable when dealing with distended bowel of questionable viability, but it served to indicate that most of the lesions were in the lower small bowel (Table I). Multiple surgical procedures were utilized and sometimes several in the same situation. It was our operative policy to facilitate decompression by the easiest means, to remove devitalized tissue and re-establish continuity if possible. Before the more common use of intestinal intubation by an indwelling nasal catheter, ileostomy was an accepted method of combating paralytic ileus. The patients in our study commonly had ruptured appendices or other septic conditions and were in extremely poor condition. The additional trauma of this procedure and the absence of antibiotics particularly, contributed heavily to the over-all mortality. The lack of knowledge regarding principles of treatment now well established and the fact that transfusions were not ordinarily used were also contributory to this high figure in early days. The patients expiring in our hospitals had various causes of death, most of which were quite obvious, though few were proven by autopsy. This is unfortunately common in many private institutions.

TABLE I. SMALL BOWEL OBSTRUCTION SURVEY
Mankato Clinic (1927-1952)

	No. Cases	Percent
Total major surgical procedures (through 1952)	15,740	
Small bowel obstruction treated by operative means	257	1.3%
Hospital deaths	56	22% (1947-1952) 6.5%
Site of Obstruction		
Low	250	97%
High	5	2%
Unknown	2	1%
Type of Obstruction		
Strangulated	130	50+%
Simple	127	49+%

The series was equally divided as to sex, the male patients being but slightly more commonly afflicted than female. In this section, there are virtually no colored people so all of the patients were white. As one might surmise, most of the patients were over fifty years of age. Youth and early middle age contributed 24 per cent of the operations, while there were few in the very young (Table II).

TABLE II. SMALL BOWEL OBSTRUCTION SURVEY

	No. Cases	Percent
Sex and Color		
Male	135	53%
Female	122	47%
Black	0	—
White	257	100%
Age		
50 and above	153	59+%
20-50 years	62	24%
5-20 years	18	7%
1- 5 years	8	3%
Below one year	16	6%
Duration of Symptoms		
More than 48 hours	117	45+%
24-48 hours	15	6%
12-24 hours	65	25%
Less than 12 hours	51	19%
Not known	9	3%

TABLE III. ETIOLOGY OF OBSTRUCTION

	No. Cases	Percent
Hernia	119	46%
Inguinal	61	24%
Femoral	29	11%
Incisional	20	8%
Umbilical	7	3%
Internal (omental rent)	2	1%
Adhesions	72	28%
Ruptured appendix	24	10.5%
Intussusception	11	4%
Ileus	9	3.5%
Carcinoma	6	2%
Volvulus	3	1%
Gallstone	3	1%
Congenital bands	3	1%
Mesenteric thrombosis	2	Less than 1%
Pin perforation	1	Less than 1%
Abdominal injury (hematoma)	1	Less than 1%
Pelvic abscess	2	Less than 1%
Regional illeitus	1	Less than 1%
Not known	0	

TABLE IV. OPERATIVE PROCEDURE AND MORTALITY

	No. Cases	Percent
Operative Procedures		
Release bowel	128	50%
Repair hernia	113	44%
Release bowel and repair hernia	108	42%
Release adhesions	63	24%
Ileostomy	61	24%
Bowel resection	15	6%
Remove stone	3	1%
Short circuit	3	1%
Remove pin	1	Less than 1%
Mortality		
Peritonitis	31	55%
Shock	6	10%
Toxemia	5	9%
Carcinomatosis	4	7%
Cardiovascular	4	7%
Not known	3	5%
Pulmonary embolism	2	3%
Gas gangrene	1	1%

Unfortunately, 45 per cent of the patients had symptoms which lasted more than forty-eight hours. This was a definite contributory factor to mortality as will be shown later. A fair number were operated upon in less than twelve hours. These were mostly strangulated external hernia in which the diagnosis was readily apparent and of necessity came to early operation. Although the site of the obstruction was difficult to determine, there was a strikingly high incidence of obstruction in the lower small bowel. The number of strangulated and simple types were equal (Table II).

The obstructions were caused predominantly by herniations. Forty-six per cent of our cases were due to hernia of one type or another. None was included which did not have an obstructed loop of small bowel. Several such loops reduced themselves after spinal anesthetic was administered. We prefer this type of anesthetic. The surgeon's notes, however, indicated that the bowel showed definite evidence of obstruction and usually strangulation. Both of our cases of internal hernia were due to defects in the omentum of the lower ileum. Postoperative adhesions were found in seventy-two cases. Talc used in preparing rubber gloves has proven to be a peritoneal irritant and causes adhesions. For several years now, as elsewhere, nonirritating starch powders have been substituted by our operating staff. Ruptured appendices obstructed either mechanically due to abscess formation or by adhesions in twenty-four cases. This was found chiefly in the early days of clinic practice. All of the intussusception cases were of the ileocecal type. Ileus, as we know it today, was treated at that time by surgical intravention and in nine cases an appropriate ileostomy was performed. Carcinoma of the bowel in every case orginated in the colon and caused blockage by peritoneal spread or metastases to the small gut. Other sources, volvulus, gallstones, bands, et cetera, were found very infrequently (Table III).

The operative procedures in the various cases were dependent upon the type and cause of obstruction. Simple lysis of adhesions was accomplished in one-fourth of the cases. Ileostomy was deemed necessary in one-fourth of this group also, either as a proximal decompression safety valve in resections which were necessary in a fair number of instances, or as a direct attack upon the obstruction *per se*. Because of the high incidence of hernia, release of the bowel and repair of the hernia by one method or another was done most often. As one can readily see, circumstances usually permitted this combination, but in a few instances doubtful tissue viability prevented hernia repair (Table IV).

Investigation showed that peritonitis was by all odds the biggest cause of death. It was difficult to separate shock and toxemia in obstructed patients, but in earlier days shock following oper-

TABLE V. SUMMARY OF MORTALITY
56 Cases

Sex	
Male	53%
Female	47%
Age	
Above 50	66 %
20-50	20.7%
5-20	5.7%
1- 5	2 %
Below 1	5.7%
Type	
Simple	62%
Strangulated	38%
Duration of Symptoms	
More than 48 hours	64%
24-48 hours	6%
12-24 hours	26%
12 hours	3%

TABLE VI. ETIOLOGY—PROCEDURE—MORTALITY
56 Cases

Etiology	
Adhesions	25%
Ruptured appendix	25%
Hernia	20%
Inguinal	9%
Incisional	9%
Femoral	2%
Ileus	7%
Carcinoma	6%
Intussusception	5%
Mesenteric thrombosis	5%
Volvulus	Less than 2%
Gallstone	Less than 2%
Pin	Less than 2%
Congenital band	Less than 2%
Internal hernia	Less than 2%
Procedure	
Ileostomy	58%
Release bowel	22%
Repair hernia	18%
Release bowel and repair hernia	17%
Release adhesions	15%
Bowel resection	9%
Remove gallstone	3%
Remove pin	Less than 2%
Incision and drainage	Less than 2%

ation was common, and that is what this figure represents. Carcinomatosis due to primary carcinoma of the colon in our series was found to be the cause of four deaths. Cardiovascular disturbances, coronary occlusion, and pulmonary embolism were found in a surprisingly small number of cases, considering the high incidence of our patients in the upper age groups (Table IV).

In summarizing our mortality, male and female patients were involved practically identically. Sixty-six per cent of the dead patients were over fifty years of age. Curiously enough, simple obstruction was found in 62 per cent of the fatal cases, while strangulated conditions existed in only 38 per cent. As previously mentioned, the duration of symptoms was a definite factor in mortality, 64 per cent of our patients having symptoms more than two days (Table V). Adhesions were found in 25 per cent and hernias in 20 per cent. The remainder of the etiological agents were of lesser significance. Ileostomy was done in 58 per cent of the patients who died, which would indicate that if such a procedure were deemed necessary, the patient was in very critical condition or tissue viability prevented reestablishment of continuity. It did not mean presumably that the operation itself was a contributing factor in the death, although, in earlier days, the additional insult of such a procedure was a definite factor in the high mortality which we have encountered (Table VI).

It is interesting to note that a male or female patient with small bowel obstruction had a one in five chances of death. In those fifty years of age or above and, curiously enough those with a simple hernia rather than a strangulated hernia, chances of survival were considerably decreased. All of our patients with mesenteric thrombosis expired. Internal hernias, likewise, carried a high mortality. Half of the patients requiring ileostomy expired.

During the past five years, with strict attention to the excellent principles mentioned and earlier operation in questionably secure patients being treated conservatively, our mortality has dropped to 6.5 per cent (Table I).

Reference

1. Wangensteen, Owen: Intestinal obstruction. Ed. 2. Chaps. 5 and 6. Springfield, Illinois: Charles C Thomas Co., 1942.

THE FAMILY DOCTOR

The family doctor has in general a commendable composite of wide interests or receptivity to ever-broadening developments in the various aspects of total medical practice, of an inevitable understanding of his patients that goes beyond Johnnie's pimples and Mary's menstrual cramps and Susie's loss of weight due to lack of appetite—beyond the peptic and other complaints and into life stresses and global biologic considerations. These aspects of medical practice on the part of the family doctor are inevitable because he finds himself as a matter of course in the peculiar position of friend, adviser, and dependency symbol; this is the complete physician, this is the family doctor.—Jess V. Cohn, M.D., in *Pennsylvania Medical Journal.*

THROMBOTIC THROMBOCYTOPENIC PURPURA

Report of a Case

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THROMBOTIC thrombocytopenic purpura is characterized pathologically by generalized hyaline arteriolar and capillary thrombi and clinically by a fulminating thrombocytopenic purpura accompanied by hemolytic anemia and transient renal, cardiac, and neurological manifestations. It was first reported as a newly recognized disease by Moschcowitz[19] in 1925. The next report of the condition appeared in 1936 when Baeher, Klemperer, and Schifrin[2] clearly defined the syndrome and presented four additional cases. It has been described with increasing frequency in American medical literature during the past five years. An excellent review of the reported cases was made in 1952 by Barondess[3] and he added three new cases of his own. At present, the literature contains descriptions of about forty cases,[1-24] in only a small percentage of which was the diagnosis even considered antemortem. In 1952, Cooper, Stickney, Pease, and Bennett[10] reported two cases in which the diagnosis was confirmed antemortem by examination of aspirated bits of bone marrow. In most instances, the disease has occurred in young adult white women, although it has also been reported in men and in colored people.

The onset of illness is usually abrupt, although occasionally there has been reported a preceding period of vague ill health. The symptoms include malaise, weakness, fever, and frequently suggestions of an upper respiratory infection. Often there have been abdominal pains, anorexia, nausea, and vomiting. Anemia may be present at the onset or may develop during the course of the illness and is usually independent of the bleeding which accompanies the thrombocytopenic purpura. Transfusions have been of little benefit. Frequently jaundice and hyperbilirubinemia have accompanied the condition. Purpura and other hemorrhagic manifestations, such as melena, hematemesis, and hematuria, invariably have eventually been part of the picture. Aside from the hemorrhagic manifestations of the thrombocytopenic purpura, transient and bizarre neurological disturbances of almost any type have been distinctive clinical features. There may be headache, dizziness, delirium, confusion, drowsiness, and progressing coma. Transient paralysis or paresis are very common. The disease, in all reported cases, has been fatal, usually in from two to four weeks, varying from one week to three months. Engle[12] reported a case which had a partial remission of about one month's duration, but this is extremely rare.

In thrombotic thrombocytopenic purpura, the blood shows the typical manifestations of a hemolytic anemia, although the severity of the anemia and the rate of the hemolysis have varied greatly. The erythrocyte count and hemoglobin have dropped simultaneously, resulting in a normocytic, normochromic type of anemia. The mechanism of the hemolytic process has been carefully studied by Singer.[21,22] In the cases where the osmotic fragility has been reported, it was normal or increased. There has been no evidence of auto-immunization, transfused cells being destroyed as rapidly as the patient's own cells. The Coomb's test findings, when reported, have been negative. Reticulocytes have increased, and in many cases abnormal young erythrocytes of the normoblastic series have appeared in considerable numbers in the peripheral blood. Bone marrow in most patients in whom it has been studied has shown a moderate hyperplasia with normal megakariocytic activity. There has been, with rare exceptions, a uniformly persistent thrombocytopenia, accompanied by hemorrhagic phenomenon, and prolonged bleeding and clotting times.

The etiology of this condition remains as great a mystery today as it was to Moschcowitz in 1925. The possibility of an allergic reaction has been suggested,[15] but there is little evidence to support this theory. It has also been suggested that the condition may be a primary subendothelial, vascular lesion with secondary accumulation of platelets on the injured endothelial area.[15] The idea has also been advanced that this illness belongs to the so-called collagen group of diseases.

The hyaline thrombi have been described in

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almost every organ but, as Clough[9] pointed out, there is a surprising lack of information regarding the occurrence of lesions in the skin and skeletal muscles.

Treatment thus far has been useless. All cases have terminated fatally. Although there is no apparent evidence of hypersplenism in these cases, splenectomies were done in several instances and were ineffective. Final evaluation of the effect of splenectomy cannot be made at this time, however, particularly since most of the patients on whom the operations were performed were terminally ill at the time the surgery was done.[18] The results of treatment with ACTH, Cortisone, and various antibiotics have been unimpressive. Since there is no evidence, as mentioned above, of hyperimmune bodies, we should not expect benefit from ACTH and Cortisone as pointed out by Hagen.[17]

Report of Case

A forty-one-year-old white housewife was admitted to the Northwestern Hospital on October 9, 1952, primarily fearing that she was having a stroke.

For six months prior to admission, she had been working and living with painting and staining projects in her home. She had been very active in the painting work, handling paints, stains, and solvents. For about three months before the onset of acute symptoms, she had complained of weakness and exhaustion out of proportion to the amount of work she did. For a month prior to admission, she had been suffering from severe frontal headaches to which she had previously not been subject. She suffered from aches and pains in her legs. Because of these complaints, she had gone to a rest hospital for five days "to get a good rest away from her family." On October 8 her hemoglobin was 45 per cent. On the night of admission to Northwestern Hospital, she had experienced a transient aphasia and right-sided paresis.

Review of her past history revealed that the patient had had the usual childhood diseases without complication. She had always enjoyed good health and had been a very active, vigorous woman. She had an appendectomy in 1926, a tonsillectomy in 1946, and plastic surgery on the fallopian tubes in 1938. Menses started at age thirteen, and occurred every twenty-seven to thirty-five days with a normal flow for six days. Her systemic history was entirely negative and her routine medical examinations had always been reported as negative. When she was examined on March 11, 1952, there were no abnormal findings. At that time urinalysis was negative. The hemoglobin was 12.3 grams; erythrocyte count, 4,240,000; leukocyte count, 9,800 with 78 per cent neutrophils, 20 per cent lymphocytes, 1 per cent eosinophils, and 1 per cent monocytes. Erythrocyte sedimentation rate was 20 mm. in one hour. Stool was negative for occult blood.

Physical examination on admission to Northwestern Hospital revealed a well-developed, fairly well-nourished, white woman who appeared to be very apprehensive. Her skin and mucous membrane were pale. The eyes, including the fundi, were normal. The ears, nose, and throat were within normal limits. The tongue was normal. Examination of the neck revealed no adenopathy and no limitation of motion. Examination of the chest revealed no abnormality. The heart tones were normal; the cardiac rate was 100 per minute with a regular sinus rhythm. Examination of the abdomen revealed no palpable masses or viscera. There was no tenderness or rigidity. The abdomen was soft. Neurological examination was entirely negative. Blood pressure was 120/80. There were small hematomas on the shins of both legs. There were no petechiae noted.

On admission, the electrocardiogram was normal. The hemoglobin was 5.7 grams; erythrocyte count, 1,590,000, and the leukocyte count, 10,000 per cu. mm.

Shortly after admission, the patient complained of precordial pain. Because of this pain and low hemoglobin, she was given 1000 cc. of whole citrated blood intravenously during the first twelve hours following admission. On October 10, 1952, a bone marrow biopsy was done. Study of the bone marrow revealed a hematocrit of .5 per cent fat, 66.5 per cent plasma, 1 per cent myeloid-erythroid layer, 32 per cent erythrocytes. Study of the smears from the myeloid-erythroid layers showed a marked increase in the developing red cells. These were normoblastic cells. A mild increase in the plasma cells was also present and a normal number of megakariocytes was noted. There was no evidence of leukemia, tumor, or granulomata.

Further laboratory studies on October 10, 1952, revealed a hemoglobin of 8.4 grams; erythrocyte count, 2,400,000, and leukocyte count, 7,300. Differential study of the white blood cells revealed 73 per cent neutrophils, 24 per cent lymphocytes, 1 per cent monocytes, and 2 per cent eosinophils. The reticulocyte count was 14 per cent. The platelet count was 39,000. A urinalysis was normal. The bleeding time was 7 minutes 45 seconds; clotting time, 3 minutes. The prothrombin time was normal. The erythrocyte sedimentation rate was 27 mm. in 60 minutes.

Further laboratory studies within a few days after admission revealed normal blood urea nitrogen, normal cephalin cholesterol flocculation test, normal thymol turbidity test, no occult blood in the stool, and free hydrochloric acid in the gastric analysis. Coomb's test findings were negative on two occasions.

X-ray examination showed a normal chest, normal esophagus, stomach, and duodenum, following the ingestion of a barium meal, and a normal colon except for marked redundancy throughout. Repeated electrocardiograms showed no change and were essentially the same as those made in March, 1952, and February, 1951.

In spite of repeated transfusions (a total of 6,000 cc. of blood) during her hospital stay, the hemoglobin and erythrocyte count remained low. The platelet count varied between 18,000 and 52,000. The patient's red blood cell osmotic fragility was tested. Her cells began to hemolize at .54, and hemolysis was complete at .32.

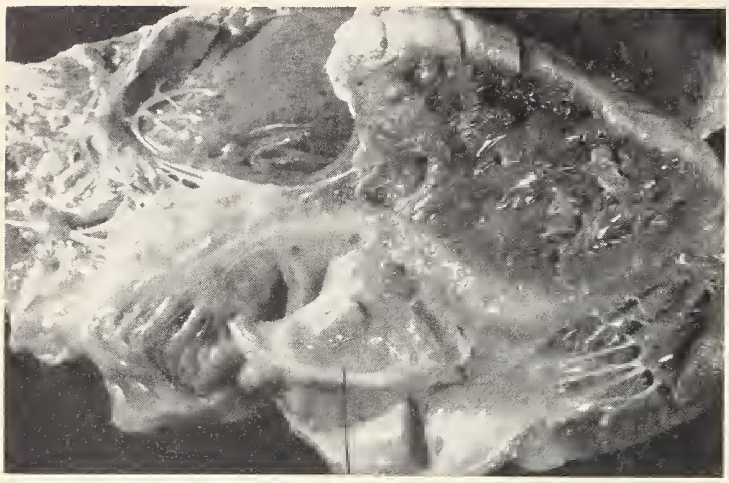

Fig. 1. Heart. Note the multiple petechial hemorrhages on the endocardial surface.

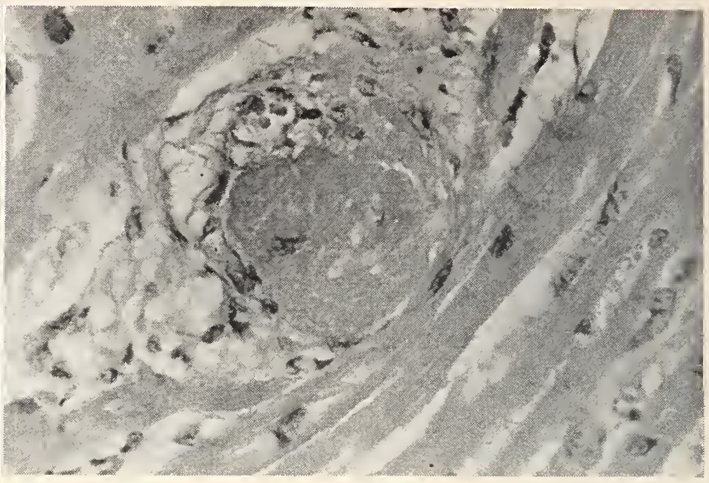

Fig. 2. Heart muscle with a typical lesion. Note the hyaline thrombus in the arteriole.

Control cells began at .46 and were complete at .34. There was no evidence of spherocytosis in any of the blood smear studies. The serum bilirubin was slightly elevated to 1.3 mg. Of this total, 0.12 mg. was direct acting. The thymol turbidity test showed 1 unit. Cephalin cholesterol flocculation test revealed one plus in twenty-four hours and 2 plus in forty-eight hours. On October 13, 1952, the quantitative urine urobilinogen was .9 units per 100 cc. of urine. Examination of the gastric contents on October 14, 1952, revealed normal free hydrochloric acid of 50 degrees and a total acid of 59 degrees.

On October 17, 1952, purpura was first noted on the patient's legs. On October 20, the Guaiac test on the stool was positive for the first time. On October 20, 1952, the possibility of thrombotic thrombocytopenic purpura was considered in the differential diagnosis. At this time Cortone was started by mouth, 25 milligrams four times a day.

On October 22, 1952, the patient started to menstruate with excessive blood loss. She also started to have bloody emesis at this time. Her liver became very tender and palpable at the costal margin. At times it was thought that the spleen could also be palpated in the left upper quadrant.

On October 25, 1952, the patient had repeated bloody emeses and was unable to keep the Cortone down. This drug was discontinued and ACTH, 100 mg., was given at once and 50 mg. every eight hours thereafter, intramuscularly.

During the evening of October 25, 1952, the patient was seen in consultation with Dr. C. J. Watson, who concurred in our diagnosis of hemolytic anemia and thrombocytopenic purpura and also with the possibility that this might be a thrombotic thrombocytopenic purpura. On October 27, 1952, the bone marrow slides were examined by Dr. Bennett and Dr. Stickney of the Mayo Clinic. While there were no thrombi visible in the sections we examined, Dr. Bennett thought there were some suggestive changes in some of the vessels, and suggested further bone marrow studies be made in an attempt to find the diagnostic lesions. The patient expired suddenly on October 28, 1952, nineteen days after admission to the hospital. During the nineteen days her temperature ranged from 98 to 100 degrees.

Autopsy (four hours after death) was performed and described by Dr. Frederick H. Lott.

General Appearance: The body was that of an adult white woman, measuring 158 cm. in length. Estimated weight was 125 pounds. The body was well developed and well nourished. Postmortem rigidity was present, and there was a mild postmortem lividity in the dependent parts. There was no edema or jaundice. The pupils were sound and equal. The ears and nose showed nothing of note. The teeth were in a good state of repair. The external genitalia showed evidence of bleeding, and the anus showed nothing of note. There were numerous small petechial hemorrhages over much of the body surface, many of which were secondary to injection. There was a well-healed lower abdominal scar which measured 16 cm. in length. There was a well-healed McBurney's scar measuring 12 cm. in length.

Abdominal and Thoracic Cavities: On opening the abdomen, the subcutaneous fat was found to measure 2 cm. in thickness. The peritoneal surfaces were smooth and shiny, and there was no excess fluid present. The appendix had been surgically removed. The liver extended 10 cm. below the xiphoid process and 7 cm. below the costal margin in the right midclavicular line. The diaphragm arched to the fourth rib on the right, and to the fourth intercostal space on the left. On opening the thorax, the lungs were found to be free and the pleural cavities empty.

Heart: The pericardial sac showed nothing of note save for a slight excess of clear amber fluid. The heart measured 13 cm. in width. Examination of the larger branches of the pulmonary artery showed no evidence of thrombi or emboli. The heart weighed 310 grams. The valves were normal in appearance. Examination of the coronary arteries showed nothing of note. The myocardium was somewhat flabby in consistency, and on cut section had a markedly mottled appearance, with small petechial hemorrhages separated by small greyish-yellow areas, which suggested fibrosis. This was extremely marked in both ventricles and in the interven-

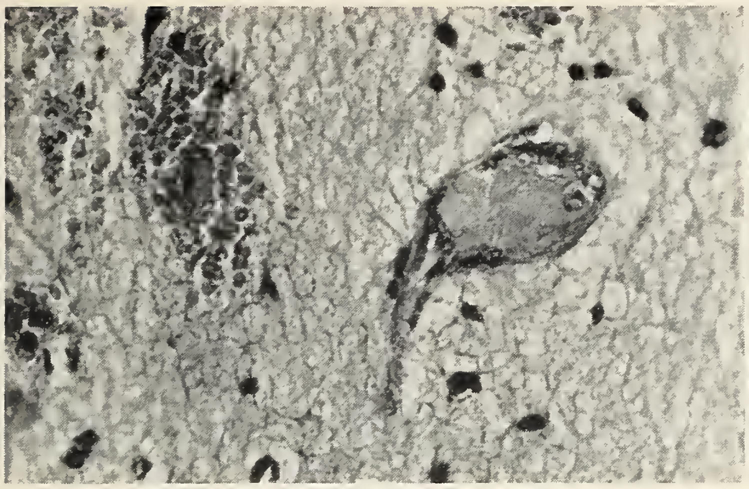

Fig. 3. Brain. Showing typical hyaline thrombi in the arteriole.

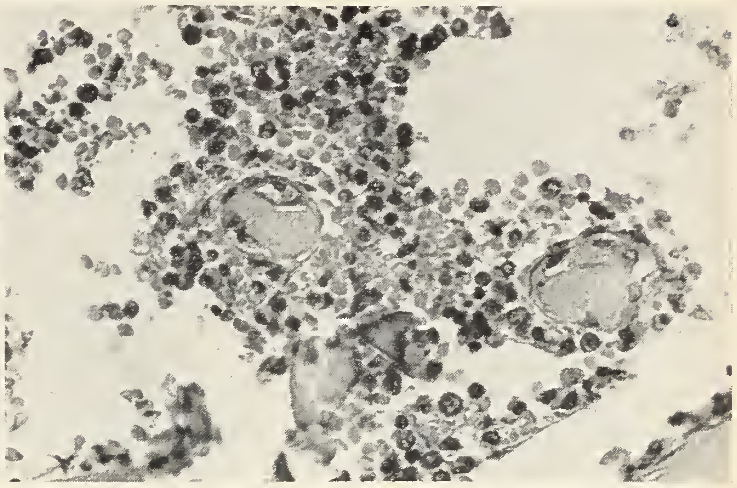

Fig. 4. Bone marrow. Showing two arterioles occluded by hyaline thrombi. This specimen was obtained at autopsy. The antemortem specimens unfortunately failed to show the lesions.

tricular septum. The petechial hemorrhages showed on the endocardial surface of both the right and left ventricles. The root of the aorta was free from atheromatous degeneration. The auricular appendages contained no thrombi.

Lungs: The right lung weighed 200 grams. The surface of the organ was smooth and crepitation was slightly reduced throughout. On cut section, the cut surface showed a very slight excess of blood and fluid, but there was no evidence of consolidation. The left lung weighed 180 grams. The surface of the organ was smooth, and crepitation was slightly reduced throughout. On cut section, the cut surface showed a slight excess of blood and fluid, but there was no evidence of consolidation.

Liver and Gallbladder: The liver weighed 1,900 grams. The surface of the organ was smooth and the edges sharp. On cut section, the cut edge everted, and the cut surfaces showed the markings to be indistinct. There was no gross evidence of darkening of the centers of the lobules. The gallbladder wall was normal in thickness. No stones were present. The common bile duct was patent.

Spleen: The spleen weighed 175 grams. The capsule wrinkled easily. On cut section, the edge everted and the cut surface was red in color. The pulp was soft in consistency. The splenic corpuscles were barely visible.

Pancreas and Adrenals: The pancreas and adrenals showed nothing of note.

Kidneys: The right kidney weighed 150 grams. The capsule stripped easily, leaving a smooth surface. On cut section, the cut edge everted, and the cut surfaces showed the markings to be indistinct. There was no evidence of atrophy of the cortex. The pelvis and ureters showed nothing of note. The left kidney weighed 165 grams. It showed a picture similar to that of the opposite organ.

Bladder and Genital Organs: The bladder contained a few cubic centimeters of a clear yellow fluid, but the bladder mucosa showed numerous hemorrhagic areas. The uterus was normal in size, and, on cut section, contained a small amount of clotted blood. The endometrium was smooth and the cervix showed nothing of note. The fallopian tubes were free and the fimbriated ends appeared patent. On cut section, they were normal in caliber. The ovaries were normal in size, and, on cut section, showed nothing of note.

Abdominal Aorta: The abdominal aorta showed a very mild degree of atheromatous degeneration.

Gastrointestinal Tract: The gastrointestinal tract showed numerous petechial hemorrhages.

Head: On opening the head, the scalp and calvarium were found to be normal in appearance. Examination of the brain showed approximately twenty petechial hemorrhages measuring up to 2 mm. in diameter in the cortex of the brain. These were most pronounced on the right side. The entire brain substance was slightly softened, but otherwise there was no evidence of gross hemorrhages, tumor, or softening. The pituitary gland was normal in appearance.

Microscopic examination showed innumerable hyaline thrombi within the capillaries and arterioles of all the major organs including the bone marrow. These were especially numerous in the myocardium and brain. Associated with these thrombi were scattered small hemorrhages which were surrounded by beginning degeneration of the myocardium and brain. The balance of the sections showed nothing of note.

Diagnoses: Thrombotic thrombocytopenic purpura, with petechial hemorrhages in the heart and brain.

Discussion

While this disease is undoubtedly rare, it is probably more common than the number of cases which have been reported since 1936 would lead one to believe. Diagnosis is probably missed because of our unawareness of the syndrome. We feel that we are justified in reporting this case in order to bring the syndrome to the attention of a greater number of physicians, pointing out again the salient diagnostic features: transitory bizarre neurological disturbance, thrombocytopenic purpura, and hemolytic anemia. Awareness of the syndrome may facilitate earlier diagnosis of the condition and bring about a better understanding of the etiology, pathogenesis, and treatment.

Summary

1. A case of thrombotic thrombocytopenic purpura with clinical and pathological findings is reported.

2. The clinical picture is manifested by a fulminating thrombocytopenia, hemolytic anemia, and transient focal neurological abnormalities.

3. The postmortem examination is characterized by widespread hyaline thrombotic lesions involving the arterioles and capillaries. The exact nature of these thrombi has not been determined.

4. Its etiology is unknown, and to date all reported cases have been fatal.

5. The importance of antemortem studies has been pointed out.

References

1. Altschule, M.D.: A rare type of acute thrombocytopenic pupura: Widespread formation of platelet thrombi in capillaries. New England J. Med., 227, 477, 1942.
2. Baehr, G., Klemperer, P., and Schefrin, A.: An acute febrile anemia and thrombocytopenic purpura with diffuse platelet thrombosis of capillaries and arterioles. Tr. A. Am. Physicians, 51:43-58, 1936.
3. Barondess, J. A.: Thrombotic thrombocytopenic purpura (Review of the literature and report of three cases.) Am. J. Med., 13:294-310, 1952.
4. Beigelman, P. M.: Varients of the platelet thrombosis syndrome and their relation to disseminated lupus. Arch. Path., 51:213-223, 1951.
5. Bernheim, A. E.: Widespread capillary and arteriolar platelet thrombi. J. Mt. Sinai Hosp., 10:287-291, 1943.
6. Blackman, N. S.; Cohen, B. M., and Watson, J.: Thrombotic thrombocytopenic purpura: report of a case. J.A.M.A., 148:546-548, 1952.
7. Brown, E. B., and Norman, J. W.: Multiple platelet thrombi. New York S. J. Med., 46:2167-2193, 1946.
8. Carter, J. R.: Generalized capillary and arteriolar platelet thrombosis. Am. J. M. Sc., 213:585-592, 1947.
9. Claugh, P. W.: Editorial: Thrombotic thrombocytopenic purpura. Ann. Int. Med., 33:739-743, 1950.
10. Copper, T.; Stickney, J. M.; Pease, G. L., and Bennett, W. A.: Thrombotic thrombocytopenic purpura (confirmation of clinical diagnosis by bone marrow aspiration). Am. J. Med., 13:374-383, 1952.
11. Ehrich, W. E., and Seifter, J.: Thrombotic thrombocytopenic purpura caused by iodine. Arch. Path., 47:446, 1949.
12. Engel, G. L.; Scheinker, I. M., and Humphrey, D. C.: Acute febrile anemia and thrombocytopenic purpura with vasothrombosis. Ann. Int. Med., 26:919-933, 1947.
13. Fitzgerald, P. J.; Auerbach, O., and Frome, E.: Thrombocytic acroangiothrombosis (platelet thrombosis of the capillaries, arterioles and penules). Blood, 2:519-541, 1947.
14. Gendel, B. R.; Young, J. M., and Kraus, A. P.: Thrombotic thrombocytopenic purpura. Am. J. Med., 13:294-310, 1952.
15. Gore, I.: Disseminated arteriolar and capillary platelet thrombosis: A morphological study of its histogenesis. Am. J. Path., 26:155-176, 1950.
16. Green, M. A., and Rosenthol, S.: Generalized blood platelet thrombosis report of three cases with necropsy findings. J. Mt. Sinai Hosp., 16:110-120, 1949.
17. Hagen, Paul: Hemolytic anemia. Minn. Med., 36:128-132, 1953.
18. Meacham, G. C.; Orbison, J. L.; Heinle, R. W.; Steele, H. J., and Schaefer, J. A.: Thrombotic thrombocytopenic purpura: a disseminated disease of arterioles. Blood, 6:706-719, 1951.
19. Moschowitz, E.: An acute fibrile pleomorphic anemia with hyaline thrombosis of the terminal arterioles. Capillaries: an undiagnosed disease. Arch. Int. Med., 36:89-93, 1925.
20. Muirhead, E. E.; Grass, G., and Hill, J. M.: Diffuse platelet thrombosis with thrombocytopenia and hemolytic anemia (thrombotic thrombocytopenic purpura). Am. J. Clin. Path., 18:523-532, 1948.
21. Singer, K.; Borstein, F. P., and Wile, S. A.: Thrombotic thrombocytopenic purpura. Blood, 2:542-554, 1947.
22. Singer, K.: Motulsky, A. G., and Shanberge, J. N.: Thrombotic thrombocytopenic purpura II, studies on the hemolytic syndrome in this disease. Blood, 5:434-448, 1950.
23. Trobaugher, F. E., Jr.; Markowitz, M.: Davidson, C. S., and Crowley, W. F.: An acute febrile illness characterized by thrombocytopenic purpura, hemolytic anemia and generalized platelet thrombosis. Arch. Path., 41:327, 1946.
24. Wallace, D. C.: Diffuse disseminated platelet thrombosis (thrombotic thrombocytopenic purpura), with a report of two cases. M. J. Australia, 2:9-13, 1951.

1531 Medical Arts Bldg.

MALNUTRITION

It is well known that hookworm disease, malaria, and tuberculosis are intimately related to malnutrition in many persons who suffer from these diseases, but whether or not the relationship is causal defies affirmation except in certain individual situations. The sufferer of malaria or hookworm disease may be malnourished because of anemia that reduces his productive and earning capacity and therefore his ability to provide for his food needs; or the malnourished person, as a result of diminished resistance, may fall an easy victim to such diseases. Whatever may be the relationship, it is clear that the well-being of the individual is compromised by malnutrition as well as by certain specific diseases.—Institute of Inter-American Affairs, Public Health Reports, November, 1953.

HYPERSPLENISM: LEUKOPENIC-THROMBOPENIC SYNDROME

Report of Two Cases

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LEUKOPENIA with splenomegaly usually presents a troublesome diagnostic and therapeutic problem. In 1924, Felty[4] described the syndrome of chronic arthritis associated with splenomegaly and leukopenia. In 1932, Hanrahan and Miller[5] reported the first case of splenectomy for Felty's syndrome. In 1939, Wiseman and Doan[10] described a syndrome which they called "primary splenic neutropenia." This syndrome was characterized by severe neutropenia of the peripheral blood, splenomegaly, and hyperplasia of the myeloid elements of the bone marrow. The mechanism by which these pathological changes take place has been and still is a much disputed point. One school of thought holds that the cells are removed by abnormal phagocytosis by the hypertrophied reticulo-endothelial cells of the spleen.[3,11] Another school contends that the condition is due to an unusual inhibitory effect on the bone marrow of a hormonal substance secreted by an overactive spleen.[2] Both groups agree that these interpretations are at the present time highly theoretical. In 1946, Doan and Wright reported hypersplenism secondary to other chronic diseases of the spleen.[3] These cases show the same clinical picture as those described in the earlier paper as primary splenic neutropenia by these investigators. A survey of the available literature revealed many cases which fit the primary and secondary syndrome. In 1948, Smith and McCabe[8] and in 1950, Rogers and Langley[6] reported examples with hypoplasia of the bone marrow with complete recovery of hematopoiesis following splenectomy. Since Doan and Wright's publication, several cases have been reported which may be classified as "secondary hypersplenism," which apparently benefited from splenectomy. The problem then becomes: Is there actually a primary splenic neutropenia or is the syndrome a symptom of a disease process in which the etiological factor is not recognized?

It is the purpose of this paper to report two cases of apparent hypersplenism, one of which presents an unusual bone marrow morphology which we believe is unique and instructive, and the other demonstrates a portal hypertension with secondary hypersplenism.

Case Reports

Case 1.—A white woman, aged fifty-four years of age, was referred to the authors by Dr. H. Kath of Wood Lake, Minnesota. Her principal complaints were weakness, fatigue, 42-pound weight loss, and repeated bouts of chills and fever over an eight months' period.

Physical examination showed a thin white woman who appeared to be listless and chronically ill. Her temperature was 100.6°, the pulse 102, and the blood pressure 122/82. The spleen was palpable 6 cm. below the left costal margin. The liver was not palpable.

Laboratory studies showed a leukopenia and neutropenia. The range of the leukopenia is shown in Graph A. Examination of the peripheral blood smear showed 6 per cent immature cells, which were interpreted by various pathologists as atypical immature myeloid cells of the type found in leukemia. Although there was no clinical evidence of leukemia, a bone marrow study done on May 23, 1947, was characteristic of a myeloid leukemia. Marrow preparations and the peripheral blood smears, obtained on June 1, 1947, were then sent to two hematologists, Dr. Hal Downey, University of Minnesota, and Dr. E. M. Schleicher, Minneapolis General Hospital, who independently interpreted the immature cells in the peripheral circulation as non-malignant reticulo-plasma cells and the myeloid hyperplasia of the marrow as myeloid maturation arrest rather than myeloid leukemia. Figure 1 shows a reticulo-plasma cell in the peripheral blood. In addition to the myeloid hyperplasia, the marrow sections showed several epithelioid granulomas, an example of which is shown in Figure 2. Plasma cells and eosinophils formed a loose cuff about the epithelioid structures. No giant cells were seen. We have not been able to find a paper which reports such a lesion in the bone marrow in a case of hypersplenism. This granulomatous lesion was somewhat similar to those described by Schleicher in herpes zoster.[7] Demonstrations of these granulomatous lesions led us to believe that we were dealing with a virus infection superimposed upon or causing the hypersplenism.

On July 10, 1947, in spite of our patient's serious condition and the presence of high fever, it was decided that a splenectomy should be carried out. An 800 gram spleen was removed by Drs. O. J. Campbell and N. C. Plimpton. Microscopic examination of many spleen sections revealed a rather severe reticulum hyperplasia, although no proliferation of malignant cells was seen. Many sinusoids were open and somewhat congested with red blood cells and contained from a few to many reticulo-plasma cells morphologically identical to those seen in the peripheral blood. Since only a few of these reticulo-plasma cells were seen in the bone marrow preparation, it is believed that the majority of cells were proliferated in the spleen.

Bone marrow obtained one month after the splenectomy

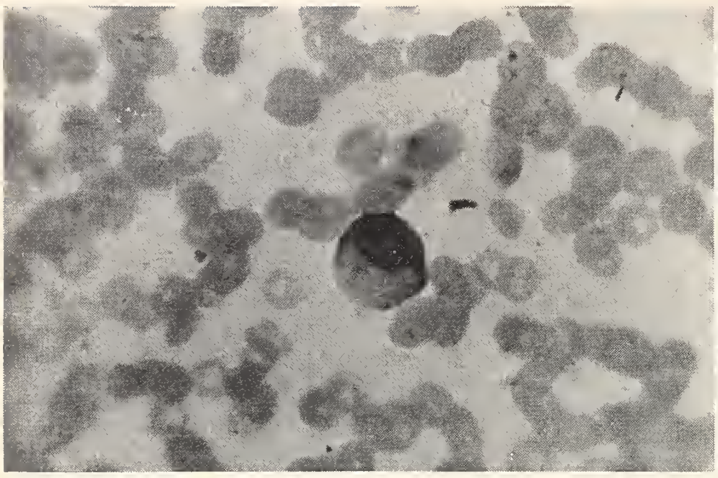


Fig. 1. Blood smear showing a reticulo-plasma cell. Six per cent of the leukocytes were this type of cell.

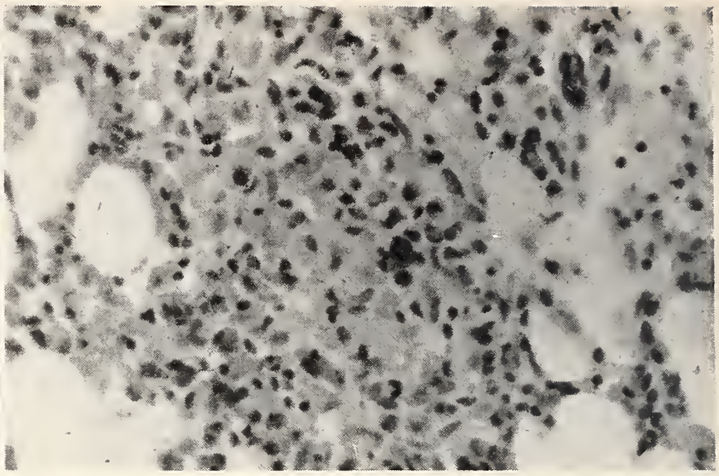


Fig. 2. Epithelioid granuloma in the bone marrow preparation.

showed none of the granulomas previously described, and normal maturation of the myeloid cells was found. Bone marrow aspiration on April 4, 1949, two years after the surgery, showed a normal marrow. The patient made an uneventful recovery and six years postoperatively is enjoying good health. We feel that this patient had hypersplenism without thrombocytopenia. The hypothesis may be advanced that the leukopenia which was corrected following splenectomy may have been in part the result of a chronic infection. We believe that this case points out the necessity of examining sections of the bone marrow as well as smears in cases of hypersplenism for conclusive interpretation of the character of myeloid hyperplasia.

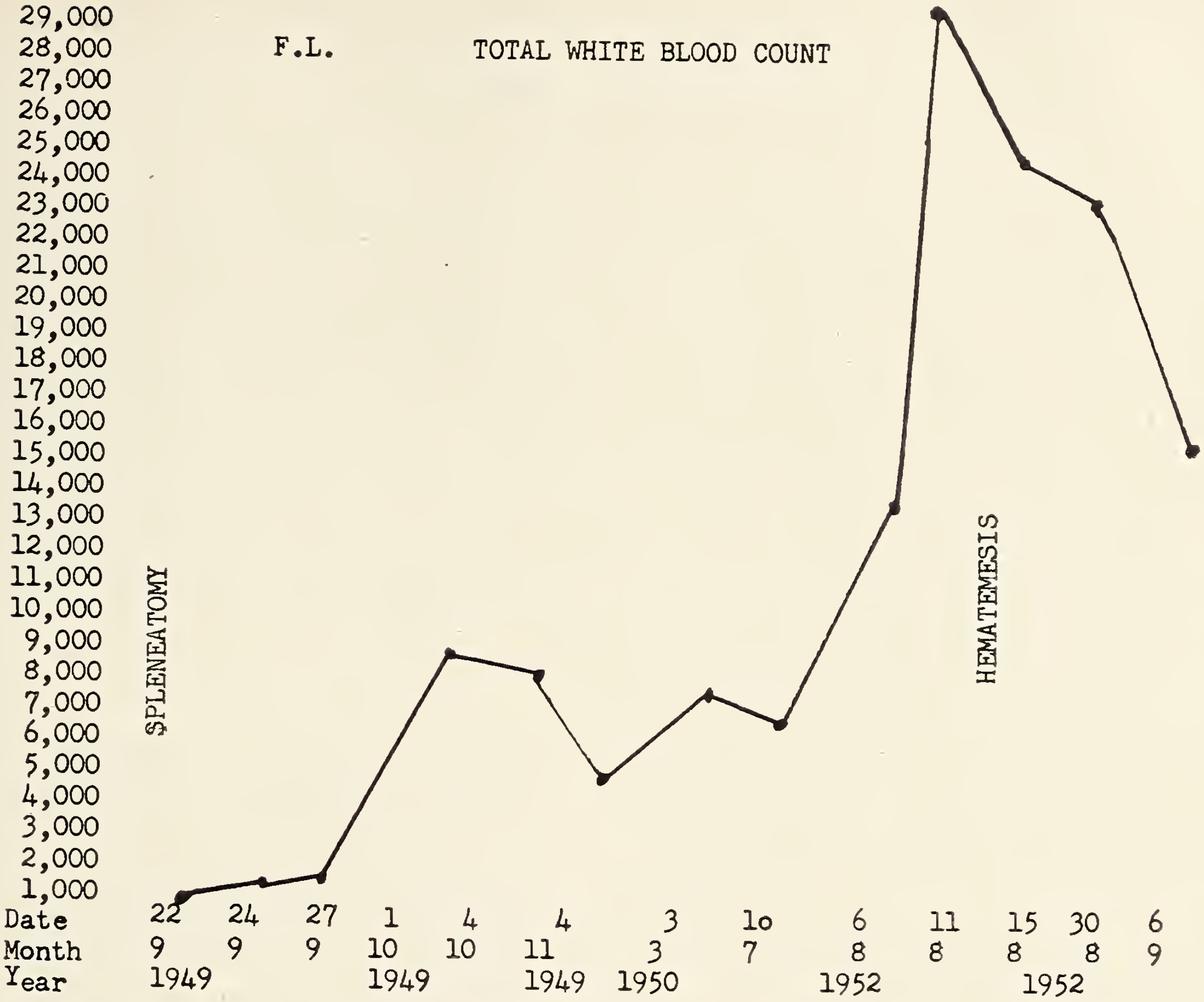
Case 2.—A fourteen-year-old boy was admitted to the Northwestern Hospital on December 23, 1949, referred by Dr. E. A. Thayer and Dr. O. E. Wandke of Fairmont, Minnesota. The patient's present illness dated back to September 14, 1949, at which time he had fractured his left forearm while playing football. The fracture had been reduced satisfactorily and the patient returned to his home. On September 17, 1949, he had a hematemesis, losing approximately one quart of blood. He was hospitalized and given two blood transfusions. It was then discovered that he had a marked leukopenia and a splenomegaly. A review of the patient's past history revealed that he had the usual childhood diseases without complications. He had a fracture of the left forearm at age four which healed uneventfully. In October, 1948, he had had a hematemesis but otherwise had enjoyed perfect health. He had been an active child and since entering high school had taken part in athletics.

Physical examination on admission to the hospital revealed a well-developed, fourteen-year-old boy who was pale but otherwise did not appear to be in any distress. Examination of the abdomen revealed the spleen to be palpable 5 inches below the umbilicus. The spleen was hard but not tender. There were no further remarkable physical findings. The left arm was in a well-fitting plaster cast. Laboratory studies on admission to the hospital revealed the patient had a hemoglobin of 7.6 grams; an erythrocyte count of 2,560,000; a leukocyte count of 950 with 31 per cent neutrophils, 64 per cent lymphocytes, 3 per cent monocytes, and 2 per cent eosinophils. His reticulocyte count was 4.6 per cent; the platelet count was 10,000. The bleeding time was 3 minutes and 50 seconds; the clotting time, 5 minutes and 30 seconds. His sedimentation rate was 22 mm. in one hour. The stool was positive for occult blood. The total serum protein was 5.3 grams with 3.4 grams of albumin and 1.9 grams of globulin. The serum bilirubin level was 1.4 mg. per cent. The urinalysis was negative. Bone marrow was obtained by sternal aspiration on September 23, 1949; and smears, imprints, and histological sections were prepared. The preparations showed a marked myeloid hyperplasia with maturation arrest.

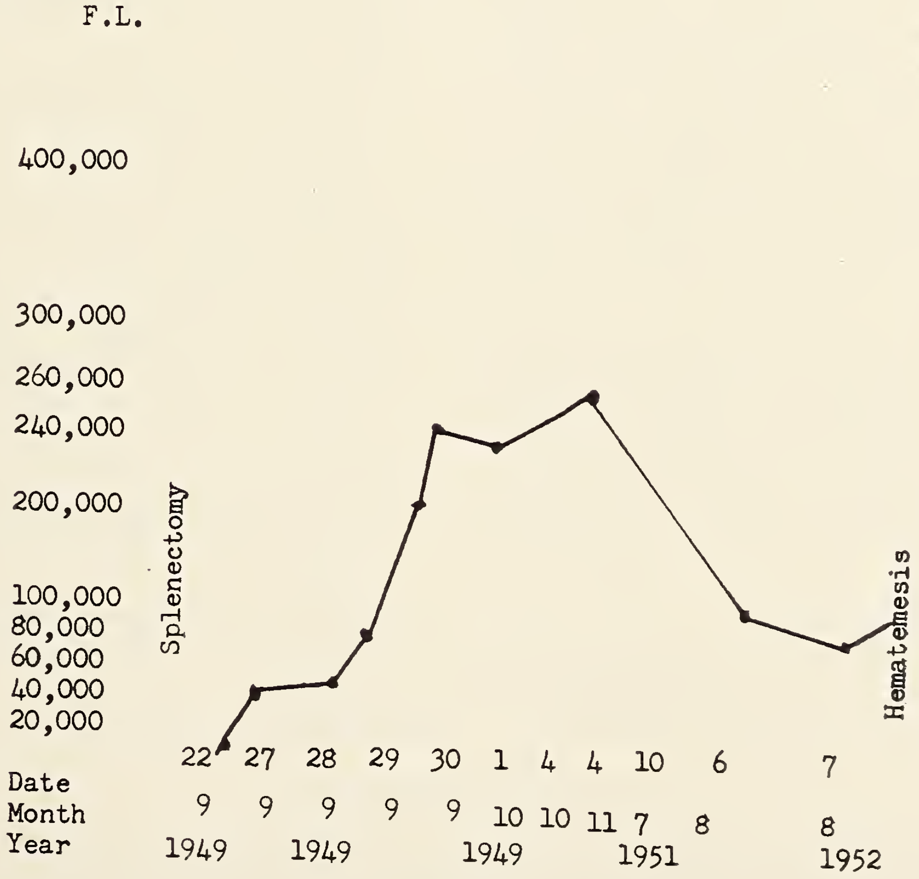
On September 28, 1949, a 1,247 gram spleen was removed by Dr. M. C. Nelson. On gross examination, the spleen was not considered remarkable. The microscopic examination disclosed the sinusoids to be small and not congested. An insignificant amount of fibrosis was present. The lymphoid follicles were relatively small and widely spaced. There was an occasional hemorrhagic area ranging from one to two mm. in diameter. There was nothing seen to suggest a malignant lymphoblastoma or any specific disease.

Postoperatively, the patient's course was uneventful. His hematological response is shown in Graphs 1 and 2. On October 4, 1949, his hemoglobin was 16.8 grams. His erythrocyte count was 5,290,000. His leukocyte count was 8300 with 74 per cent neutrophils, 23 per cent lymphocytes, and 1 per cent monocytes. The platelet count was 232,000. His urine remained normal. The patient was seen again on November 4, 1949; and physical examination was entirely negative. On March 3, 1950, he was seen again, at which time his hemoglobin was 15.6 grams; red blood cell count was 4,560,000; white blood cell count was 8000; and the red blood cell sedimentation rate was 14 mm. in one hour. Differential studies of the blood at this time showed 23 per cent neutrophils, 76 per cent lymphocytes, and 1 per cent eosinophils.

From March 3, 1950, until August 4, 1952, this young man was well and was working as a truck driver. At 11:30 P.M. on August 4, 1952, he was awakened by an emesis of a large quantity of blood. He was taken to the local hospital, where he was given blood transfusions; and then because the hematemesis recurred he was



Graph 1.



Graph 2.

brought to the Northwestern Hospital at 3 A.M. on August 6, 1952. Physical examination on admission to the hospital was entirely negative. The hematemesis continued, necessitating repeated blood transfusions. Eighteen hours after admission, x-ray examination following a barium meal revealed a marked degree of varices extending from the aortic arch to the distal end of the esophagus. Laboratory studies revealed an anemia but no other diagnostic findings. Liver function tests and bone marrow studies were normal.

The hematemesis persisted in spite of balloon tamponage; so, although the patient's condition was poor, an attempt to do a venous shunt to relieve the portal hypertension was made on August 16, 1952. The pressure was measured in a large vessel in the omentum and found to be 385 mm. of blood. The veins in the omentum and on the peritoneum were large and tortuous. The esophagogastric junction was exposed and all the veins from the diaphragm and about the junction were clamped and ligated. The esophagus was pulled down about 4 cm. and packed around its entire circumference with gelfoam. The patient's condition was such that further exploration and shunting procedures were deemed unwise. A liver biopsy was taken and reported as normal.

On August 30, 1952, laparotomy was again undertaken with the plan in mind to establish a portacaval or a splenorenal shunt. The splenic vein was exposed by Blakemore's technique[1] and found to be completely thrombosed, as was the portal vein. In place of the portal vein were a few collateral venus channels which were small and very friable. None of these veins was considered suitable for a shunting procedure.

Following this second operation the hematemesis and melena subsided, and the patient was discharged by ambulance to the hospital in his home town on September 6, 1952, one month after admission on August 6, 1952. From August 4, 1952, to September 6, 1952, he received a total of 71 pints of blood. His hemoglobin varied from 5.3 grams to 16.8 grams and was 13.4 grams at the time of discharge. Leukocyte counts, differential studies, and platelet counts were all normal. After September, 1952, he had three massive gastrointestinal hemorrhages, each necessitating several transfusions. He had a massive hemorrhage in June, 1953, and expired. An autopsy was refused.

It was our original impression that this patient had a hypersplenism with leukopenia and thrombocytopenia preceding his first hematemesis in October, 1948. Now we wonder if this hematemesis and the splenomegaly may have been due to portal hypertension, and whether the portal hypertension should not have been relieved at the time of the original operation.

Summary

Two cases of hypersplenism are presented—one patient had anemia and neutropenia and the other anemia, neutropenia, and thrombocytopenia. Case 1 had granulomatous lesions in the bone marrow, suggesting a secondary type of hypersplenism. Case 2 simulated a primary portal hypertension with secondary hypersplenism. This second case demonstrated the value of the warning made by Blakemore and Fitzpatrick[1] and earlier by A. O. Whipple,[9] that when a splenectomy is attempted the surgeon should always look for portal hypertension and, if possible, determine its cause. He should be prepared to do a splenorenal or a portacaval shunt if it is indicated. These cases lead us to speculate that perhaps, as more information accumulates, all the cases of so-called primary hypersplenism will prove to be secondary to some other primary condition.

References

1. Blakemore, Arthur H., and Fitzpatrick, Hugh F.: The surgical management of the post-splenectomy bleeder with extra-hepatic portal hypertension. Ann. Surg., 134:420-432, 1951.
2. Dameshek, Wm.; Miller, E. B.: The megakaryocytes in idiopathic thrombocytopenic purpura, a form of hypersplenism. Blood, 1:27-52, 1946.
3. Doan, Charles A., and Wright, Claude-Starr: Primary congenital and secondary acquired splenic panhematopenia. Blood, 1:10-26, 1946.
4. Felty, A. R.: Chronic arthritis in adults associated with splenomegaly and leukopenia. Bull. Johns Hopkins Hosp., 35:16-20, 1924.
5. Hanrahan, E. M., and Miller, S. R.: Effect of splenectomy in Felty's syndrome. J.A.M.A., 99:1247-1249, 1932.
6. Rogers, H. M., and Langley, F. H.: Neutropenia associated with splenomegaly and atrophic arthritis (Felty's syndrome): report of a case in which splenectomy was performed. Ann. Int. Med., 32:745-754, 1950.
7. Schleicher, Emil M.: Reticulo-epithelioid cell granulomas in bone marrow in herpes zoster. Am. J. Clin. Path., 19:981-984, 1949.
8. Smith, Sol, and McCabe, E. S.: Primary splenic neutropenia with arthritis: its treatment with splenectomy. Ann. Int. Med., 29:445-455, 1948.
9. Whipple, Allen O.: Rationale of portacaval anastomosis. Bull. N. Y. Acad. Med., 22:251, 1946.
10. Wiseman, B. K., and Doan, Charles A.: Primary splenic neutropenia; a newly recognized syndrome, closely related to congenital hemolytic icterus and essential thrombocytopenic purpura. Ann. Int. Med., 16:1097, 1942.
11. Wright, C. S.; Coan, C.; Bouroncle, B. A., and Dolinger, R. M.: Hypersplenism. Blood, 6:195-211, 1951.

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The most important elements in the diagnosis of cancer remain those natural attributes of the physician—he must see, hear, and feel the manifestations of malignant processes before utilizing his store of complex tools and methods.

FLUID REGULATION IN NEPHRITIS AND NEPHROSIS OF CHILDHOOD

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THE KIDNEY constitutes the main organ for the regulation of fluids and electrolytes. By an active process of filtration, absorption and excretion the kidney defends the normal chemical pattern of the plasma. The maintenance of body stores of water, the acid-base ratio of the blood and tissue cells and the elimination of products of metabolism cannot continue indefinitely in the face of chronic renal disease.

All efforts in fluid and electrolyte therapy for renal disease are based on the assumption that renal function is temporarily or partially inadequate. There is, as yet, no artificial means to maintain indefinitely, an individual with progressive renal failure, recent developments in the artificial kidney notwithstanding.

The efforts of Gamble[4,5] to acquaint members of the medical profession with the fundamental concepts of fluid and electrolyte metabolism are outstanding among those of many researchers in this field. I shall draw upon his contributions in this consideration of the requirements of children with renal disease. What is written here will be confined to a few physiologic aspects of fluid and electrolyte metabolism, on which can be based a rational course of treatment as regards administration of salt and water.

The glomerulus, by a process of filtration, produces a protein-free ultrafiltrate of plasma. The volume of filtrate produced daily approximates 180 liters. Roughly 25 per cent of the cardiac output passes through the glomeruli at any given time. It is generally agreed that the rate of filtration by the glomeruli is relatively constant.

Urea is one of the most important substances removed from the plasma by glomerular activity. Glomerular filtrate has the same acid-base ratio as plasma; that is, a pH of 7.4.

As glomerular filtrate courses past the cells of the renal tubules all but about 2 liters are reabsorbed. In the renal tubules the pH changes from 7.4 to acidity or alkalinity as dictated by the state of metabolic activity and renal function. The limit for acidity of the urine is a pH of 4.8 while the limit of alkalinity of urine is a pH of 7.8.

Two mechanisms for acidification of the urine and conservation of base are employed in normal renal function. The first of these functions consists in substitution of a hydrogen ion supplied by the renal tubule cell for a sodium or potassium ion in the tubular fluid. Glomerular filtrate contains a phosphate with two sodium ions. By substituting a hydrogen ion for a sodium ion half of the base is conserved. The monosodium phosphate is an acid salt and thus tubular fluid is acidified. The degree of acidification may be determined by titration with tenth normal sodium hydroxide to a pH of plasma. The resultant value is usually referred to as "titratable acidity." Above a pH of 7.4, therefore, there would be no titratable acidity, by definition.

A second mechanism for economy of base involves the production of ammonium by tubule cells. The operation of these two adjustments effectively compensates for the excretion of large amounts of acid substances without the depletion of body stores of base. Figures 1 and 2 show quantitatively the adjustment of the production of ammonium and titratable acidity to cover the excretion of acid radicals in the urine.

It is possible to foresee the consequences of chronic renal disease if consideration is given the aforementioned facts. Damage to the glomeruli results in failure to excrete urea, phosphates, sulfates and organic acids. The plasma therefore has a relative deficiency of base; a state of azotemia and acidosis is the consequence. When the renal tubular function is impaired the tubules cannot continue to conserve base and then it becomes necessary for larger amounts to be excreted to maintain the reaction of the urine within the physiologic range. The renal tubules also lose the ability to reabsorb water and stores of body water are rapidly lost.

There is yet another feature of tubular failure which produces distortion of fluid and electrolyte balance. Due to the loss of concentrating ability of the renal tubules as the load of substances to be excreted from the plasma has become greater,

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From the Section of Pediatrics, Mayo Clinic.
Read at the meeting of the Southern Minnesota Medical Association, Mankato, Minnesota, September 14, 1953.

removal of the load demands removal of larger volumes of water.

One sees then, that in advanced renal disease large volumes of urine of low specific gravity, and of alkaline reaction, are excreted.

Alkalosis is an unusual occurrence in renal disease, acute or chronic; therefore it merits no special consideration at this time.

The minimum amount of water required to form urine depends on the amount of substance or solute to be excreted. This need for water is usually defined as the renal obligatory water requirement. In children the renal obligatory need varies from 200 to 500 cc. per day. This requirement must be met even in starvation or thirsting and if not provided for by intake the need will be met by metabolism of the body stores of protein. Oliguria, anuria, or dehydration and death are the consequences of inadequate available water for renal function. In figure 3 the relationship of urinary volume to load of solute is depicted. On the right side of the graph the volume of water for intake and output is indicated for various routes.

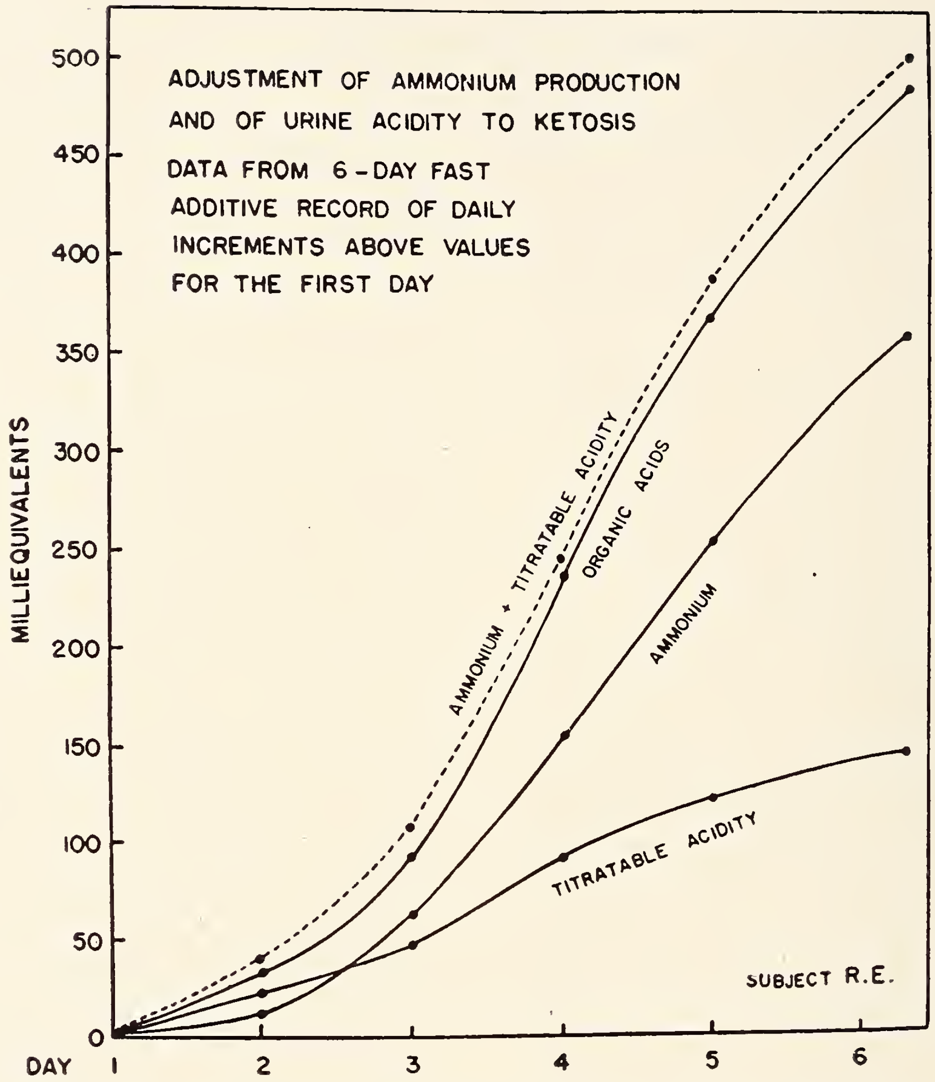

Fig. 1. Reprinted from "Companionship of Water and Electrolytes in the Organization of Body Fluids" by J. L. Gamble[5] with permission of the author and of the publishers, Stanford University Press. Copyright 1951 by the Board of Trustees of the Leland Stanford Junior University.

To the daily water requirement for renal function must be added the volume of water lost through the lungs and skin. This is designated as the insensible loss of water. The 24-hour water requirement for children varies from 150 cc. per kilogram for the infant to 75 cc. per kilogram for the older child. Fever and high environmental temperature increase the daily water requirements. The importance of the consideration of water requirements and renal function will become evident in discussion of acute and chronic states of kidney disease.

Acute Nephritis

The most common form of renal disease in children is acute hemorrhagic nephritis or glomerulonephritis. Varying degrees of severity of the disease are known to occur but in general the recovery rate approaches 98 per cent.[6] Two-thirds of the cases of acute nephritis in childhood occur under the age of 7 years. The disease is rare in infancy. Most clinicians agree that there is a distinct difference in the prognosis for acute nephritis as it occurs in adults. Similarly, the nephrotic syndrome is a significantly different disease in the adult than it is in children.

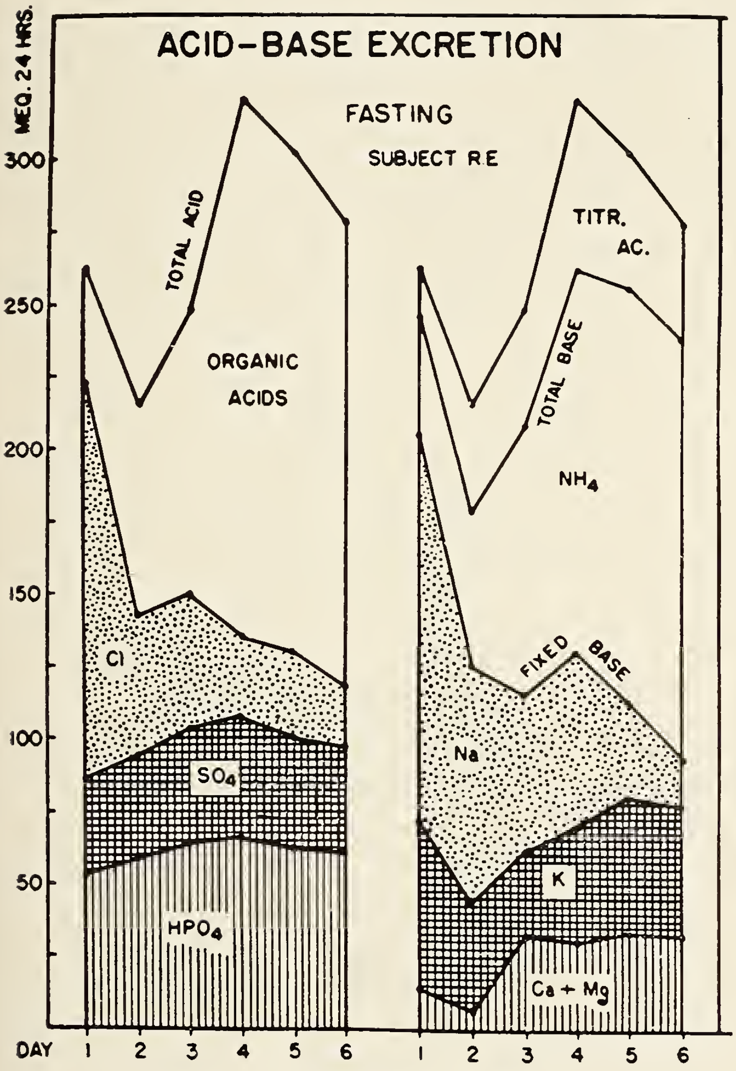

Fig. 2. (For legend, see Fig. 1.)

Generally, the onset of acute nephritis follows a streptococcus infection and is heralded by hematuria. In the average case of acute glomerulonephritis there is no need for limitation of the intake of fluid, salt or protein; therefore the concern here will be with only two complications of acute nephritis associated with severe manifestations which demand special therapeutic considerations.

The first of the two types is referred to as “hypertensive encephalopathy.” In this rather dramatic and acute illness the child suddenly has a convulsion. The blood pressure is markedly elevated and the child is comatose or confused. The concentration of blood urea usually is not elevated.

Prompt treatment to prevent convulsions and to reduce the hypertension is essential. Intramuscular administration of a 50 per cent solution of magnesium sulfate is effective as an anticonvulsant. The dosage is usually 0.2 cc. per kilogram of body weight, given every four hours until the adequate response in blood pressure is noted.

Magnesium relaxes arteriolar spasm to lower the blood pressure. Ettledorf and Tuttle have shown that it also increases the glomerular filtration rate, which is reduced in the acute phase of nephritis. The toxic effects of magnesium manifested by respiratory depression and narcosis can be counteracted by intravenous administration of calcium gluconate.

There is little need for rigid restriction of oral intake of fluids but the children frequently are comatose, in which case they require parenteral administration of fluid. Intravenous injection of fluid should be limited to an amount equivalent to the urinary output plus the estimated insensible loss. Sodium chloride should not be given, either intravenously or orally. The hypertensive phase usually abates within seven days and as a rule recovery is complete.

In the second group of patients the blood pressure may be found to be normal but oliguria and azotemia are apparent. The patients represent a small percentage of the total cases of acute nephritis but they are acutely ill and treatment is a special consideration. Oral intake of fluids may be limited somewhat and ingestion of salt should be restricted. As was mentioned previously, when oliguria or anuria is encountered intravenous administration of solutions must be restricted to replacement of daily losses from the skin, lungs and kidneys.

It used to be common practice to “flood” the anuric patient with parenterally given fluids to induce formation of urine and alleviate azotemia. The fallacy of this is obvious. Administration of an excess of fluid can only result in edema, dilution of the plasma electrolytes and aggravation of a serious clinical syndrome. Occasionally rapid administration of fluid results in convulsions. This occurrence has been termed “water intoxication.”

Presumably, cerebral edema is the cause of the convulsions.

The value for serum potassium may rise sharply in anuric states to produce signs of potassium toxicity of hyperkalemia. The concentration of serum potassium is typically more than 5 mEq. per liter in this state and an electrocardiogram shows characteristic shortening of the Q-T interval, with marked peaking of the T wave. If sodium chloride in water is administered cautiously the effects of hyperkalemia or hyperpotassemia may be prevented.

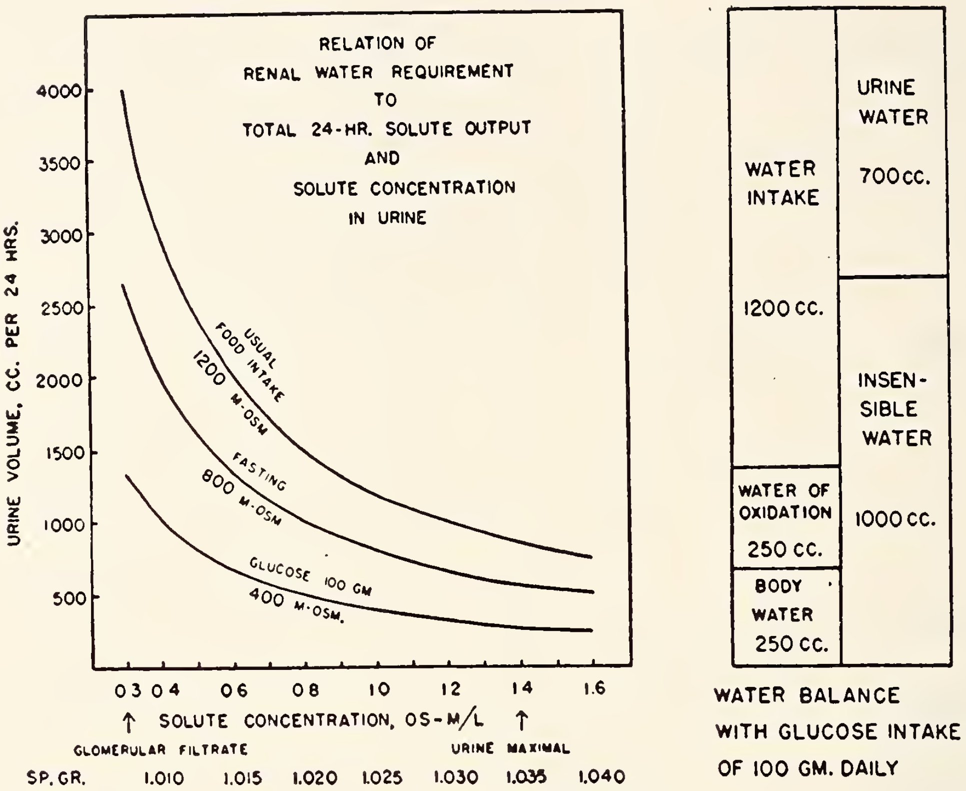

Fig. 3. (For legend, see Fig. 1.)

Chronic Nephritis

Contrasted with acute nephritis, edema in chronic nephritis is exceedingly rare. The chronic phase of nephritis is unusual in children and there are differences of opinion as to the relationship of the acute nephritis of childhood to chronic nephritis in adults. Most likely there are rare cases of chronic nephritis of adults attributable to acute nephritis which occurred in childhood.

Edema[9] in chronic nephritis usually is found only as an accompaniment of cardiac failure. Proper treatment with digitalis is indicated, but restriction of the intake of salt must be cautious.

Characteristically, the patient with chronic nephritis excretes large volumes of dilute urine which has a low specific gravity and an alkaline reaction. The large output of urine is the result of diseased renal tubules. The glomeruli also are damaged; hence, azotemia is produced.

Tubular deficiency produces excessive loss of stores of fixed base; that is, sodium, potassium, calcium and magnesium. The tubule cell is unable to substitute hydrogen ions for base ions and an alkaline urine results. The production of ammonium by the tubule cells is deficient; therefore, large amounts of base are excreted with the acid radicals (sulfate, phosphate and organic acids).

The titratable acidity is absent in an alkaline urine with a pH above 7.4. The tubules also have been impaired to the degree that reabsorption of water is altered and the result is polyuria.

Gamble maintains that a high level of blood urea is protective to a certain extent in that this extra load of solute demands an added output of water for its removal. Oliguria or anuria following trial with the artificial kidney for removal of urea and azotemic products from the plasma has been commonly observed.

Loss of base in the urine over a prolonged period results in the mobilization of calcium from bone with consequent demineralization of the skeleton. Renal rickets, though not a common accompaniment of this type of tubular disease, can occur and tetany may be noted.[2]

Albuminuria contributes to the possibility of tetany since calcium is in part protein-bound. Excessive treatment with alkali to correct the acidosis of renal disease may also produce tetany because of the reduction of available ionized calcium in an alkaline medium.

The treatment of chronic nephritis therefore consists in the provision of an adequate intake of water. Alkalinizing treatment with Shohl's solution (citric acid, 190 cc.; sodium citrate, 90 cc.; water sufficient to make 1,000 cc.), is indicated. The usual oral dosage of this mixture is 1 ounce four times a day.

Calcium may be supplied by a large intake of milk. Very little calcium will be absorbed from the intestine if any calcium salt other than calcium lactate is used. To be effective, calcium lactate must be dissolved in boiling water and cooled.

Some clinicians advocate the use of antacids on the theory that antacid blocks the absorption of phosphates from the gastrointestinal tract. Caution must be used in prolonged use of antacids, and occasional determinations of the pH or carbon dioxide content of the serum should be made to avoid gross disturbances of electrolytes.

While death from renal insufficiency is the usual outcome of chronic nephritis, many years may be added to the patient's life by careful management of the disease.

Nephrotic Syndrome

The term "lipoid nephrosis" has been supplanted by the term "nephrotic syndrome." The latter is preferable because the clinical course of each case is similar to that of every other. Pure or lipoid nephrosis is less commonly encountered and supposedly is not accompanied by hematuria or azotemia. The diagnosis "nephrotic syndrome" applies to cases with or without microscopic hematuria, casts, albuminuria and mild azotemia.

Typically, the onset of the nephrotic syndrome is insidious, with edema of the periorbital region or of the ankle as the presenting symptom. From the onset the syndrome progresses to massive edema, ascites, hypoproteinemia, oliguria and anemia. Remissions and exacerbations are the rule and the predisposition to intercurrent infection is well known.

Kohn and Obrinsky's series of nephrotic patients has been followed over a period of twenty-six years. This series included fifty-seven children of whom thirty-six died. Most of the fatalities occurred before the advent of satisfactory treatment with sulfonamides and antibiotics.

In a more recent survey of cases by Philips, Calvin and Kagan, twenty-nine patients were followed from 1944 to 1950 and a more encouraging report is presented. These authors advanced the idea that the survival of twenty-three of the twenty-nine patients was primarily due to the prevention of infection. Four of the six children who died succumbed to bronchopneumonia. The importance of effective use of antibiotics and chemotherapy is apparent.

The physiologic alterations in the nephrotic syndrome are loss of protein through the damaged glomerular basement membrane, a decreased glomerular filtration rate and excessive retention of salt and water by the renal tubules.

In the past few years, significant developments in the treatment of the nephrotic syndrome have resulted through the use of corticotropin (ACTH) and cortisone. While foremost authorities in study of the nephrotic syndrome make no claim that these potent hormones cure the disease, there is increasing evidence that corticotropin and cortisone are effective in prolonging remissions. From this clinical observation has come careful speculation that the ultimate outcome will be a higher incidence of complete recovery.

In the past several years, the general attitude of pediatricians has changed from one of "let nature take its course" to one of more active treatment of the nephrotic syndrome. Dextran, a polysaccharide of high molecular weight and polyvinylpyrolidon (PVP) also have been employed as effective means of producing diuresis.

Metcoff and associates have advocated a ten-day course of corticotropin in a dosage of 150 mg. per square meter of body surface per day to be given in four divided injections to effect diuresis. Approximately 70 per cent of children so treated will undergo diuresis following this program. On a second, later trial diuresis will result in an additional 13 per cent. Other investigators have advocated oral administration of cortisone for several days a week for a period of six to eight weeks following diuresis.

The electrolyte pattern of the nephrotic syndrome has been well defined by the studies of Metcoff and associates. Before diuresis with corticotropin, the concentration of sodium, potassium, chlorine and carbon dioxide in the serum were normal. Following diuresis, no change of significance occurred except for a minor rise in serum sodium and a slight fall in serum potassium. Extensive studies of renal function showed that the diuresis accompanied significant improvement in the glomerular filtration rate and in renal tubular function. A few patients experienced gross disturbance of electrolytes which demanded prompt treatment of alkalosis. Several other patients suffered infections. The intake of fluid or salt was not restricted during treatment. Excessive salt was not permitted in the diet, however.

In the cases of Metcoff and associates, when sodium was needed potassium also was administered despite the lack of excessive loss of potassium during diuresis.

Those with whom I am associated believe that definitive treatment for patients with the nephrotic syndrome requires a course of corticotropin early in the disease, followed by oral administration of cortisone three days per week, in a dose of 100 to 200 mg. per day for a period of six to eight weeks after diuresis. It is essential that prophylaxis against infection with antibiotics be offered during treatment with corticotropin. Penicillin should be given orally during treatment with cortisone.

There is no need for rigid limitation of intake of fluid in remissions and a general diet without "added salt" may be employed. Barnett expressed the belief that there should be as few hospitalizations in the course of the disease as possible, and that activity should be restricted as little as practicable. As regards the use of antibiotics, Barnett has advocated liberal use of these agents when threats of infection occur but he has cautioned against continual administration.

Brief mention should be made of two additional syndromes wherein renal failure is concerned, and their associated fluid and electrolyte problems. The first of these syndromes is lower nephron nephrosis. This condition may be the consequence of various insults to renal integrity such as transfusion with incompatible blood, infection and shock. Considerable damage to the renal tubule cells occurs and tubular and epithelial casts appear in the urine. Anuria follows during the period of regeneration of the tubule cells.

Administration of fluids must be held to the replacement of the daily estimated losses until renal function is resumed. A period of four to seven days usually is required for recovery.

A second condition is the low-salt syndrome which occurs as a result of an inadequate intake of sodium. Edema is apparent and renal function ceases. Prompt replacement therapy with hypertonic sodium chloride, in 2 per cent or 6 per cent solution, is effective in restoring renal function.

Summary

Only in anuric or oliguric states associated with renal disease should intravenous administration of fluids and salt be rigidly restricted. Fluids, when ingested orally, seldom produce aggravation of oliguric or anuric states. In chronic nephritis, if edema is absent, the intake of salt and water need not be restricted. Slight hope is offered for increased recovery rates among patients who present the nephrotic syndrome, through judicious use of corticotropin and cortisone. Additional help toward improvement of recovery rates can be expected from the antibiotics. Complete exclusion of salt from the diet alone can produce edema and oliguria. Treatment of the low-salt syndrome requires administration of hypertonic salt solutions.

References

1. Barnett, H. L.: Management of nephrosis. Symposium on renal function in infants and children. Child Hosp., Buffalo, Mar. 2 and 3, 1953. (Copies distributed to audience.)
2. Bland, J. H.: The Clinical Use of Fluid and Electrolytes. 259 pp. Philadelphia: W. B. Saunders Company, 1952.
3. Ettledorf, J. N., and Tuttle, A. H.: The effects of magnesium sulfate on renal function in children with acute glomerulonephritis. J. Pediat., 41:524-527 (Nov.) 1952.
4. Gamble, J. L.: Chemical Anatomy, Physiology and Pathology of Extracellular Fluid. Cambridge: Harvard University Press, 1947.
5. Gamble, J. L.: Lane Medical Lectures. Companionship of Water and Electrolytes in the Organization of Body Fluids. Vol. 5, 90 pp. Stanford, California: Stanford University Press, 1951.
6. Hebert, H. J.: Acute glomerulonephritis in childhood: a study of the late prognosis of twenty-seven cases. J. Pediat., 40:549-557 (May) 1952.
7. Kohn, J. L., and Obrinsky, William: Lipid nephrosis in children. Observations over a period of twenty-six years. Am. J. Dis. Child., 84:587-600 (Nov.) 1952.
8. Metcoff, Jack; Rance, C. P.; Kelsey, W. M.; Nakasone, Nobuyuki, and Janeway, C. A.: Adrenocorticotropic hormone (ACTH) therapy of the nephrotic syndrome in children. Pediatrics, 10:543-564 (Nov.) 1952.
9. Nelson, W. E.: Mitchell-Nelson Textbook of Pediatrics. Ed. 4, 1350 pp. Philadelphia: W. B. Saunders Company, 1945.
10. Philips, I.; Calvin, J. K., and Kagan, B. M.: Nephrotic syndrome in children. Am. J. Dis. Child., 85:451-461 (Apr.) 1953.

CHEMOTHERAPY OF THE LEUKEMIAS AND LYMPHOMAS

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A FEW YEARS AGO a title such as this would have been meaningless because the treatment of leukemias and lymphomas was confined to roentgen therapy. Such treatment is still the standard by which any other must be measured, but at present several drugs have earned a place in everyday treatment and many others are under investigation.

Mustards and Related Compounds

Since World War I, the mustard gases have been known to depress lymphocytic tissue. As the result of investigation during World War II, the nitrogen mustards were selected for clinical trial in the treatment of neoplastic diseases. Methyl-*bis* (β-chloroethyl)-amine, referred to as HN_2, has proved least toxic and easiest to manage. It is clinically effective in Hodgkin's disease, lymphosarcoma and leukemia. HN_2 probably exerts its action by interfering with mitosis and is similar in action to roentgen therapy. HN_2 is soluble in water but disintegrates rapidly; therefore, it must be given immediately after being dissolved. Because HN_2 may produce thrombophlebitis with ease, it is wise to inject this material into the tube of an intravenous infusion set in which a 5 per cent solution of dextrose is being used. The dose per course consists of 0.3 to 0.5 mg. per kilogram of body weight, the average total dose being 25 to 40 mg.; it is given usually in two to four divided doses. The immediate reactions are nausea, vomiting, fever and headache. Sedation with barbiturates before and after the injection and the use of dimenhydrinate (dramamine) may help to modify these reactions. Depression of the neutrophils and platelets may occur in one to twenty-one days. Seldom is this severe enough to be troublesome, although blood transfusions and antibiotic therapy occasionally are necessary. A course of HN_2 may be repeated in four to six weeks.

This drug has been most effective in the treatment of Hodgkin's disease. Remissions such as occur after roentgen therapy are common, but on the average they are not maintained for so long a period. The best indication for treatment with HN_2 is widespread and systemic involvement. The more localized lesions of Hodgkin's disease are better treated with roentgen therapy unless viscera are involved. HN_2 has been effective when radiation has lost its effectiveness; at times, tissues that have become resistant to radiation appear to respond again after treatment with HN_2. The value of combined treatment has not yet been determined. Evidence exists to indicate that the results of treatment with HN_2 followed immediately by roentgen therapy, may be superior to the results of either agent used alone.

The mustards are somewhat less effective in the treatment of other lymphomas but what has been said about Hodgkin's disease applies to them. Although improvement may follow the treatment of chronic leukemia with HN_2, the drug has been used only occasionally in this disease. It is of no value in the treatment of acute leukemia.

A closely related compound is triethylene melamine (TEM—Lederle Laboratories Division, American Cyanamid Company). Its effect on neoplastic tissue appears to be identical to that of HN_2 but it can be given orally and is effective in smaller doses. The standard dose is 5 or 10 mg.; it is given in the morning before the patient has had anything to eat or drink. Nausea and vomiting may occur but are not common. Administration is not repeated until the effect of the preceding dose is ascertained. Daily determinations of leukocytes and platelets are essential because some patients, particularly those who have lymphatic leukemia and lymphosarcoma, are extremely sensitive to this drug. Patients who have Hodgkin's disease or chronic myelogenous leukemia tolerate larger doses. A maintenance dose of 5 mg. every seven to ten days is usually possible but careful adjustment of the dose is required in each patient. Severe depression of the bone marrow may occur rapidly and renal damage also may result.

Another group of compounds, the triethylenephosphoramides (TEPA—Lederle Laboratories Division, American Cyanamid Company) have similar properties and belong in the mustard

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From the Section of Medicine, Mayo Clinic.

group. One of these, triethylene thiophosphoramide (thio TEPA), is fairly stable in solution and may be given orally in doses of 10 mg. It appears to have a wider margin of safety than does TEM. Its effectiveness has not been fully evaluated. It is of no value in the treatment of malignant melanoma.

Drugs of the mustard group have been used in the treatment of polycythemia vera, mycosis fungoides and metastatic carcinoma of the breast with variable but sometimes good results.

Antimetabolites

Another group of drugs that act in a different way has found a place in the management of leukemia. These agents are the antimetabolites, the best known of which are the folic-acid antagonists. Of many such compounds, 4-aminopteroyl glutamic acid (aminopterin) and amethopterin are now in general use and are available. Unlike most of the other drugs discussed in this paper, these agents are of value in the treatment of acute leukemia. These drugs are extremely toxic and easily produce signs that resemble acute deficiency of folic acid. Administration of citrovorum factor or folinic acid will counteract this reaction. Most workers agree that toxic effects and remissions in acute leukemia are closely related, so that these drugs must be given until the toxic state is at least closely approached. The type of leukemia that responds most often is acute or subacute lymphocytic leukopenic leukemia of childhood or adolescence. All types of acute leukemia, however, have responded and occasionally an adult will be helped.

Aminopterin is given in doses of 0.5 to 1.0 mg. and amethopterin in doses of 2.5 to 5.0 mg. daily. It is important to watch for signs of toxicity during administration of folic-acid antagonists. Alopecia is annoying but not serious. Stomatitis with shallow ulceration is often the first sign of toxicity and soon may be followed by severe abdominal pain accompanied by bloody diarrhea. The latter manifestations are caused by ulcerative enteritis, which is usually of grave prognostic significance. Severe depression of the bone marrow with neutropenia, leukopenia and thrombocytopenia may occur but often are also part of the natural course of the disease. Stomatitis or abdominal pain is an indication to discontinue administration of the drug or reduce the dose. As previously mentioned, folinic acid in doses of 1 to 3 mg. may be used to counteract the toxic effects. When any improvement is to occur, reticulocytosis and regeneration of erythrocytes in the bone marrow often precede the disappearance of leukemic cells. About 25 per cent of children who have acute leukemia undergo remissions after use of these folic-acid antagonists; the average remission lasts for three months. However aminopterin and amethopterin are difficult to handle, and in many instances an exceedingly sick patient is made even more uncomfortable.

Folic-acid antagonists may be used in the treatment of chronic leukemia and the lymphomas but the hazards do not justify such use because other methods are available.

Another drug that probably should be classified as an antimetabolite is also useful in the treatment of acute leukemia. It is 6-mercaptopurine (purinthol—Burroughs Wellcome & Co.), which apparently is an antagonist of hypoxanthine and adenine. It is given in a dose of 1 mg. per pound of body weight and is administered orally in tablets containing 50 mg. each. Although nausea and vomiting may occur after its use, stomatitis is rare. The incidence and duration of remissions produced by this drug are not yet fully known.

Hormones

Cortisone and corticotropin also have found a place in the treatment of acute leukemia. The doses used are large; 200 mg. of cortisone or 100 mg. of corticotropin is given as long as these agents are tolerated. Lymphocytic leukemias are most likely to respond and again most of the remissions occur in children. While use of these steroids induces good remissions in about 25 per cent of patients, the duration of the remissions averages about half of that obtained with the folic-acid antagonists, namely, six weeks instead of three months. One advantage of these hormones is their absence of severe toxic reactions. The folic-acid antagonists may be given simultaneously with the hormones or alternate courses of each agent may be used.

Cortisone also may be used in the treatment of Hodgkin's disease, in which it causes shrinkage of involved tissue but only short remissions. In chronic lymphocytic leukemia it may favorably affect the cutaneous lesions even though the numbers of blood cells are not altered. It also should be tried in hemolytic anemias secondary to lymphoma or chronic leukemia.

Miscellaneous Drugs

Urethan (ethyl carbamate) has been used in the management of chronic leukemia, particularly myelogenous leukemia. Administration of doses up to 3 gm. per day produces a decrease in the number of leukocytes and in the size of the spleen. The great difficulty with this drug is the nausea and vomiting which it may induce; however, restriction of doses to 1 or 2 gm. per day usually prevents this effect. My experience, however, has been that urethan is not so effective in inducing and maintaining remissions in chronic myelogenous leukemia as is roentgen therapy; it offers little benefit when the disease is radioresistant.

English workers recently have introduced a new compound, namely, 1,4-dimethanesulfonoxybutane (myleran—Burroughs Wellcome & Co.), which is reported to be effective in the treatment of chronic myelogenous leukemia. While my experience indicates that the drug may reduce the number of leukocytes, I cannot say whether it will prove more useful than urethan.

Many years ago, Fowler's solution was used in the treatment of leukemia, particularly in the chronic myelogenous type. Although this preparation is subject to the disadvantages of arsenical therapy, it is still a useful drug in this disease.

Comment

My colleagues and I have noted, as have others, that multiple remissions may occur in acute leukemia when the same drug is used on different occasions and that leukemia which becomes resistant to one drug may respond later to another. While such occasions are not common, they do occur. At all times, general supportive care, such as blood transfusion and antibiotic therapy, is carried out. While these factors may influence survival times, I do not think they are responsible for remissions. It must be emphasized that no curative agent for acute leukemia has been found and that remissions in children are not the rule and are rare in adults. At the present time I consider that the best program of treatment, if the patient or the parents understand the whole picture and still desire to have such treatment, is a trial of a folic-acid antagonist combined with or followed by use of corticotropin or cortisone. Administration of 6-mercaptopurine may be of value when the disease is resistant to use of folic-acid antagonists. It is questionable whether any drugs are useful in patients more than thirty years of age.

The availability of drugs that will augment or supplement roentgen therapy in Hodgkin's disease, the lymphomas and the chronic leukemias should be welcomed by the general practitioner, the pediatrician and the internist. It means that renewed interest may be taken in patients, formerly dismissed to the care of the radiologist and it should lead to closer co-operation between the family physician and the specialist. This should result in better care for the patient.

Summary

Roentgen therapy remains the standard treatment for Hodgkin's disease, lymphoma and chronic leukemia.

Nitrogen mustard is effective in the treatment of these diseases, particularly Hodgkin's disease. Related compounds that may be given orally are triethylene melamine and triethylene thiophosphoramide. In selected cases they are useful agents.

The folic-acid antagonists, as well as corticotropin, cortisone and 6-mercaptopurine cause remissions in acute leukemia in children, and young adults but such remissions are not long maintained.

SCHOLARSHIPS IN PUBLIC HEALTH

The Harvard School of Public Health is initiating postgraduate scholarships in amounts ranging up to $5,000 to qualified candidates desiring to study at the school during the academic year 1954-1955. These scholarships are available to (1) physicians, dentists, and veterinarians interested in preventive medicine and seeking training in one or more public health specialties leading to a master of public health or a doctor of public health degree; (2) industrial physicians seeking training in industrial medicine leading to a master of industrial health degree; (3) public health nurses with a college degree and satisfactory field experience who wish additional public health training leading to a master of public health degree; (4) public health engineers seeking additional training and research experience in one or more public health specialties leading to either a master of science in hygiene or a doctor of science in hygiene degree; and (5) college graduates with academic experience in the natural sciences who desire training and research experience in one of the sciences related to public health and leading to either a master of science in hygiene or a doctor of science in hygiene degree. Applications must be filed by April 30. Winners will be announced June 1. Information may be obtained from the Secretary, Harvard School of Public Health, 55 Shattuck St., Boston 15.

Laboratory Aids to Medical Practice

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OFFICE HEMATOLOGY

JAMES L. JAECK, M.D.
La Crosse, Wisconsin

"OFFICE HEMATOLOGY" refers here to routine blood tests performed on outpatients *in the doctor's office.* Few physicians can obtain or maintain a registered medical technologist. Many rely on the "office nurse," who may be neither nurse nor technician. The doctor himself, therefore, is responsible for the reliability of his office laboratory. Unreliable laboratory data are worse than none at all!

Office hematology should provide adequate screening for primary hematologic disease and should furnish dependable quantitative information for clinical evaluation and treatment of secondary hematologic problems. Realize the limitations of the office laboratory and freely seek hematologic consultation. Do not be guilty of the diagnosis of anemia by therapeutic trial. Have respect for persistent leukopenia with anemia. Use caution in diagnosis and prognosis of adenopathy with lymphocytosis in children and young adults; in such cases, send blood for determination of heterophil antibodies once a week for three weeks and send good blood smears to a clinical laboratory.

Choose office blood tests for maximal dependable information, rapidity and minimal cost but never trade time for accuracy. A single determination has much less significance than a series, which may show a "trend." Repeat tests whose results are inconsistent with clinical impressions. No one hematologic finding is pathognomonic. A minimal hematologic screening for every new office patient should consist of: (1) hemoglobin or hematocrit; (2) total leukocyte count and (3) peripheral blood smear. An erythrocyte count gives little information and has great personal and technical error; it may well be omitted unless cell indices are to be done. The erythrocyte count may be normal in hypochromic anemia. The hematocrit is more valuable than either hemoglobin or erythrocytes in following the treatment of anemia.

I would do a differential count only when indicated by the number of leukocytes or the appearance of the blood smear. The differential count requires a well-stained smear and proper training in morphologic hematology or it is worthless. I often receive blood smears for diagnosis accompanied by total and differential leukocyte counts, yet find the smear so poor that I cannot identify cells for a differential count, to say nothing of returning a morphologic diagnosis.

Depending on the size of the office laboratory and on trained personnel, one could set up certain unit or group tests to be done on the basis of clinical diagnosis and results of basic screening tests. For the study of anemias, such a unit might include: (1) complete blood counts; (2) hematocrit; (3) cell indices and (4) reticulocyte count. For a study of hemorrhagic tendencies the unit also would include: (1) bleeding time; (2) clotting time; (3) clot retraction; (4) platelet count and (5) prothrombin time.

Hemoglobinometry

Determination of hemoglobin is the most commonly used of all office blood tests, but it also has the greatest range of error (5 to 40 per cent). Error is referable to: (1) differences in methods and large inherent error; (2) lack of uniform standard of reference; (3) pipette and instrument error; (4) personal technical errors in visual colorimetric methods and (5) instability of color of hemoglobin and acid hematin.

The Tallqvist scale and Dare method have the

This is the fourteenth in a series of editorial reports sponsored by the Minnesota Society of Clinical Pathologists and designed to foster closer relationships between clinicians and pathologists.

Dr. Jaeck is Pathologist and Director of Laboratories, Gundersen Clinic and Lutheran Hospital, La Crosse, Wisconsin.

greatest inherent error. The popular Sahli method with careful technique and calibrated pipettes and tubes can attain a satisfactory accuracy of ±5 per cent. The following precautions reduce technical error in the Sahli method: (1) use a *dry*, bubble-free pipette; (2) use clean diluting fluid; (3) wipe off blood on outside of pipette; (4) fill pipette accurately (if overdilution occurs, discard and start over); (5) discard first drop of diluting fluid; (6) rinse acid in Sahli tube back and forth several times; (7) stir constantly while delivering pipette sample into Sahli tube; (8) allow a minimum of twenty minutes at room temperature before diluting the acid hematin to match color standard; (9) stir while diluting and match color in same good light each time; (10) read meniscus in Sahli tube in grams per 100 cc. of blood and (11) use tubes made by same manufacturer as standard.

The Haden-Hausser hemoglobinometer (larger laboratory model) has the advantage of allowing use of the blood sample in the white pipette. It also allows approach of the end point from either side without the danger of passing it. Both Sahli and Haden-Hausser techniques are visual colorimetric acid-hematin methods subject to inherent errors of fading and the use of artificial permanent color standards. Each physician must determine, for the instrument used, the equivalent in grams per 100 cc. for 100 per cent hemoglobin on a series of healthy, normal adults with erythrocyte counts near 5,000,000. A value of 15 gm. per 100 cc. of blood is the average acceptable standard for this region.

Hematocrit

The volume per cent of packed erythrocytes is the most accurate index of circulating hemoglobin and erythrocytes. The hematocrit is quantitative, simple, informative and does not require unusual technical skill. A good method is that of Wintrobe, as detailed in Wintrobe's *Clinical Hematology*. The equipment required includes Wintrobe hematocrit tubes, capillary pipettes and any standard centrifuge capable of 2,500 to 3,500 rpm. Some precautions for accurate results include: (1) dry anticoagulant in clean tubes; (2) invert blood sample in oxalate tube ten to twelve times; (3) fill hematocrit tube immediately after mixing; (4) let blood settle in hematocrit tube for one hour before centrifuging (this gives sharper demarcation of layers and also permits a sedimentation rate reading in the tube); (5) centrifuge for thirty minutes and (6) use hand lens to read level of packed erythrocytes.

The normal hematocrit value in men is 47 ± 7 per cent and in women is 42 ± 5 per cent. In anemia, the value may go as low as 8 per cent, whereas in polycythemia it may be as high as 80 per cent.

Blood Cell Counts

A likely source of error in cell counts is wet pipettes. A dry pipette is one in which the bead is free in the bulb. The best way to assure a dry pipette for each count is to have enough on hand for the entire day's use without employing any pipette twice. A clean dry counting chamber is also imperative. Discard pipettes with even a tiny chip at the tip. We have greatly reduced technical errors in dilution by replacing all conventional red and white pipettes with Tri-Lyne pipettes. Bard-Parker pipettes probably are the best of the conventional types because they are calibrated individually and marked with a correction factor. The counting chamber must not be overfilled or underfilled for an accurate count.

Blood Smears

These are the most important and also the poorest done of all office hematology procedures. Any smear might be the one you want to send to a pathologist or hematologist. With a little personal supervision, Wright-stained blood smears done in the office can be just as good as those done in the clinical laboratory.

Clean slides are essential. Slides in a freshly opened box are not clean. Wash up a batch in ordinary soap and water. Rinse in plenty of hot clean water, with a final rinse in distilled water. Store in 95 per cent alcohol (ethyl) until ready for use. The best smears use a small drop of blood (pinhead size) spread lightly, evenly and without a final push or lift. Dry smear rapidly in air; do not blow or use a fan. High humidity in summer makes poor smears and poor stain affinity. I know of no remedy. Use only certified Wright stain (prepared or powder). If you make your own, use only acetone-free methyl alcohol. Keep stock Wright stain in tightly stoppered bottle. Dispense it with a small dropper bottle. Filter stain each day. Keep bottle of stain away from strong acids or alkalies.

(Continued on Page 292)

President's Letter

WE MEET AGAIN

The tradition of fine medical meetings will be carried on again in 1954. The annual meeting of the Minnesota State Medical Association is scheduled for Duluth on June 7, 8, and 9, and from all indications, the convention promises to be outstanding in all respects.

The scientific program will be one of unusual value to the medical profession of this area, specialist and general practitioner alike. Subjects run the gamut of medical interests, including such worthy subjects as ulcerative colitis, a symposium on fractures, a symposium on cortisone and ACTH, treatment and diagnosis of hypertension, diagnosis of pulmonary diseases, a symposium on anesthesiology, hydronephrosis, treatment of blood dyscrasias, anemias in pregnancy, plasma expanders, and a section on what's new in medicine, surgery, radiology, pediatrics, obstetrics and gynecology.

The usual wide selections in subject matter will again be found in the round-table discussion luncheons. Many guest speakers from the general session will lead these discussions, giving expert advice and opinion to the physicians who look on the annual meeting as an excellent opportunity to gain additional information as an adjunct to their everyday practices.

Assurance of excellent hotel accommodations has been given by the Duluth Chamber of Commerce.

Business sessions scheduled during the meeting include the following. The Council meets daily, beginning on Saturday, June 5. The House of Delegates will begin its sessions on Sunday, June 6. Association committees will meet on Tuesday and Wednesday, June 8 and 9.

A new feature this year will be one or two "consultation" booths, to be found on the convention floor at the Duluth Armory. Plans include one booth on general fracture subjects, another on obstetrics and gynecology. Here, the physician can bring his own special problems, outline them to experts in the field, and discuss their implications and treatment.

Many important social events have been planned. Special organizations will be meeting simultaneously with the State Association, holding special luncheons and dinners for their members.

Recreation has been arranged by the Committee on Local Arrangements. Golf, lake fishing and skeet and trap shooting will give the busy doctor a fine chance for relaxation away from the heavy schedule of scientific and business sessions. Duluth offers many good accommodations for such affairs, and physicians are urged to take time out to enjoy them.

There are many things one might mention in giving a general outline of the meeting of the year. With all the various events scheduled, it is a matter of choice on what to concentrate. But there is one important thing to remember: your attendance at the meeting in Duluth will be well worth the few days away from your practice. It offers postgraduate education from experts in all fields; it offers meetings with your special interest group; it offers time for relaxation with your colleagues throughout the profession; and it offers opportunity to meet and discuss Association problems and policy which are common to the nation's entire medical profession.

For interesting and informative medical sessions, your meeting calendar should be marked for the Minnesota State Medical Association annual convention, Duluth, Minnesota, June 7, 8 and 9, 1954.

President, Minnesota State Medical Association

Editorial

Arthur H. Wells, M.D., *Editor;* Henry G. Moehring, M.D., and John F. Briggs, M.D.

CANCER DETECTION COST vs. VALUE OF LIFE

AFTER almost twenty years of experience with Cancer Detection Centers, the medical profession should soon be able to properly evaluate many of the practical aspects of these periodic examinations of apparently healthy individuals. Perhaps the closing of large numbers of the Centers throughout the country and the recent shift of emphasis by the American College of Surgeons to Cancer Diagnostic Clinics (patients come with complaints) are signs of practicality.

Three principal questions have confronted the profession. (1) Which of the individual tests performed in the examinations is worth its salt in terms of lives saved per dollar spent? (2) Is the specialty clinic approach to cancer detection more economical than the "Every Doctor's Office a Cancer Detection Center" plan? (3) What new tests for cancer detection are there to be discovered? The answer to the last question could make almost any amount of money expended in this type of research worthwhile.

There is some value in nearly all of the clinical and laboratory procedures generally performed in these examinations. The question is, are they economically sound? An x-ray examination of the chests of more than a million people in the Los Angeles area was worth the cost in numbers of cases of tuberculosis discovered, isolated and treated. On the other hand, if the study had been carried out solely for the purpose of discovering bronchogenic cancer, rough calculation leads one to the conclusion that it would cost approximately $100,000 per life saved from this disease. As a matter of fact, there is some question as to the economy of any type of roentgen study conducted solely for the detection of cancer in the symptomless and signless individual.

From the figures of 11,000 examinations for cancer presented by Hitchcock and Aust elsewhere in this issue, one can see that only ten possible curable, asymptomatic cancers were found which were not easily accessable to the examining fingers and eyes of a physician in general practice. The salvage from these ten cases of deep-seated cancer may have been five lives. Had the ten eventually developed symptoms and gone to their family physicians in the conventional manner, at least two would have been saved. Would the difference of three lives cost an additional $50,000? We are badly in need of a dollars and cents approach to this subject concurrent with the scientific study.

Physicians are on solid economic ground when the cancer detection (in the apparently healthy person over forty years) is limited to a study of cancer symptomatology and a careful physical examination including cervical and rectal visualization. Any clues of "internal" cancer picked up in the clinical history must receive the proper diagnostic attention. The corrected cost of this type of cancer detection will be between $1,000 and $2,000 per life saved from cancer. In addition, there will be a bountiful disclosure of "precancerous" and non-neoplastic diseases. The "Every Doctor's Office . . ." program of your Committee on Cancer deserves the whole-hearted support of the profession.

BLUE SHIELD'S PLAN B

CONCLUDING that open, written debate on important issues serves the profession best, your Board of Editors publishes the following editorials on the Pro and Con of Plan B of Blue Shield for your careful consideration.

PRO

Both Blue Shield and Blue Cross were organized to provide people with a means of paying their medical-care and hospital bills at a cost within their financial reach. During the depression of 1932, many people throughout the United States were unable to pay their hospital bills, as a result of which the occupancy of hospitals dropped to a hazardously low level. To remedy this situation, Blue Cross devised a plan whereby it accepted small monthly payments from those who joined the movement for the payment of their hospital bills. It was a non-profit, prepayment plan to provide hospital care at a cost which people could afford.

Similarly, the depression of 1937 created serious problems in the payment of medical care bills.

Many people were unable to pay their doctor bills, and both the medical profession and the government became interested in what was referred to as "the high cost of medical care." The government's solution appeared in the form of the first Wagner-Murray-Dingell bill introduced in Congress in 1939. The medical profession's answer appeared the same year in the organization of the first Blue Shield medical-care plan fashioned after the Blue Cross hospital plan.

In this problem of the so-called "high cost of medical care," classification of people according to their incomes and their ability to pay for medical care has been necessary. Indigent persons receive their medical care at the cost of some unit of government, local, state or federal, and, consequently, are not concerned in pre-payment plans. The wealthy are able to pay the costs of their medical care, and do not constitute part of the problem. This leaves those in the low and modest income brackets as the ones who must be provided medical care at a cost they can afford. If medicine, through Blue Shield or some other equally effective means, does not furnish the answer of providing medical care for those with low or modest incomes, the solution might be forthcoming from government, labor or industry. Thus, Plan B has the rôle of furnishing part of the answer by offering medical care to those with modest incomes within the reach of their pocketbooks.

As a first step in the development of Blue Shield's, Plan B, the issuance of a contract with service benefits for families with annual incomes less than $5,000 was first discussed with the Council and the Houe of Delegates of the Minnesota State Medical Association at the Radisson Hotel in May, 1952. No action was asked nor taken regarding it, but neither were any adverse comments made regarding it. The matter was again presented to the Council at the meeting of the Minnesota State Medical Association in May of 1953. At this time, the contract's increase of fees to approximately 50 per cent more than those of the standard contract, and the contract's income limits for service benefits of $3,600 for single persons and $5,000 for families were fully presented. No objections were raised at the Council meeting, and the matter was referred to the House of Delegates. Here again, these same two features of Plan B were clearly discussed before the House of Delegates, which then approved the plan with only one dissenting vote. Plan B, then, was developed only after it was approved by the governing body of organized medicine in Minnesota.

After the announcement of its issuance, two objections to Plan B were voiced by some members of the medical profession. The first of these concerns fees. As a first step in the deteremination of an adequate fee schedule, a questionnaire was sent to the component medical societies asking them to list the fees customarily charged by their members for some of the more common medical-surgical-obstetrical services. Replies were received from nineteen of the thirty-two component medical societies. Then, four of the highest Blue Shield fee schedules of such higher income contracts as Plan B were assembled. The fees selected for the Plan B schedule consisted principally of the highest or next highest fees of the four Blue Shield schedules. By comparison with the fees listed in the questionnaires, the Plan B fees were usually above those customarily charged by members of the various component societies. The final Plan B fee schedule has fees, in general, 50 to 55 per cent higher than those of Plan A. Also, the Plan B fee schedule ranks among the highest Blue Shield fee schedules in the United States.

Despite its generally favorable comparison with other fee schedules, it has been pointed out that it still contains some inequities. To overcome these objections, the Board of Directors has appointed a special committee whose purpose it is to meet with the authorized representatives of any group of physicians for the review and adjustment of inequities. Efforts are now being made to adjust inequities of fees and other Plan B matters in this manner.

The second objection to Plan B is that Plan B might establish the fees for 75 per cent of the population. This statement is based upon the fact that an average of approximately 75 per cent of single persons and families in Minnesota have incomes less than the service income limits of the Plan B contract.

Enrollment under Plan B contracts in other states, as well as Plan A enrollment here in Minnesota, fails to support such a statement. In one of the states having such a contract, after several years of comparatively successful Plan B enrollment, only a relatively small percentage of the population had Plan B coverage. Were this same rate enrollment of Plan B to occur in

Minnesota, is it likely that Plan B might establish fees for 75 per cent of the population?

It has been calculated from income data available in 1949 that an average of approximately 70 per cent of single persons and families then had incomes less than the standard contract's service income limits. This is only five per cent less than the percentage now said to be potentially eligible for service benefits under Plan B, yet no statements have been made that the standard contract established the fees for 70 per cent of the population.

Other economic factors have an important bearing on these service income limits. For instance, since Blue Shield was organized there have been five general, national wage and salary increases. These salary raises have elevated the incomes of many workers to the point where they are no longer eligible for Plan A service benefits. One of the Building and Construction Trades Council in Minnesota lists twenty-nine types of construction workers whose lowest hourly wages make them ineligible for Plan A service benefits. The Agriculture Department and the Farm Management Office of the University Extension Service found the average annual farm labor income in the southern half of Minnesota in 1952 to be $3,950. Are not these wage earners and farmers the types and groups of people who were originally intended to have service benefits under the Blue Shield program?

Inflation also has developed to the point where the dollar today has only 60 per cent of its value in 1938. This means that a family with an annual income of $5,000 today has the same purchasing power as a family with a $3,000 annual income sixteen years ago. Families of three or four persons, and usually families in this income bracket are larger, are often unable to pay for medical and hospital care. Here again, it is apparent that these are the same people for whom Blue Shield's service benefits were originally planned.

Thus, Plan B is a necessary part of the answer to the problem. As a complement of Plan A, and with a catastrophic illness rider, Plan B provides the answer to the broad problem often referred to as the high cost of medical care. Plan A alone will not solve the problem. Plan B is an essential part of the program of providing medical care for people with low or modest incomes at costs they can afford. Recommendations have emanated from high authorities in both the past Democratic and the present Republican administrations that voluntary prepaid medical care plans provide greater benefits for more people. And, if this challenge is not met by the medical profession through such a medium as Minnesota Blue Shield's Plan B, there is little doubt that the answer may be forthcoming from some other source.

E. J. Simons, M.D.

CON

Blue Shield of Minnesota has rightfully claimed the support of the preponderant majority of physicians in Minnesota since its inception in 1947. Members of the State Medical Association have recommended this form of voluntary pre-paid medical insurance as the best buy in its field. The medical profession has supported the Blue Shield as its own medical insurance plan believing that it would materially relieve the financial burden of sickness upon Minnesota families.

The Blue Shield contract has proved its value in the succeeding years. It has earned the support of the public and the profession generally. Although in many instances the fees paid were inadequate, the physician dealing with a patient in reasonably good financial circumstances could ask for supplemental payment. Participating physicians were required to accept the Blue Shield payment as full settlement of medical charges for indigent and low income groups: single individuals with an income less than $2,000.00 per year or families with less than $3,000.00. Minnesota physicians participating in this plan subscribed to these limitations and restrictions. The profession accepted the inevitable inequities that occur when a third party sets the fee, occasionally providing medical care without fee in some cases which required the services of more than one physician. The percentage of cases falling into such a category was small.

This happy state of affairs has been severely disturbed by a letter sent out December 23, 1953, by Minnesota Medical Service Incorporated (Blue Shield), outlining a new plan, the Plan B contract, to be available to the public after March 1, 1954. Quoting from this letter:

> "Service benefits under this Plan B contract will be available to single persons with annual incomes less than $3,600.00 and to family participant subscribers whose annual family income is less than $5,000.00 The Blue Shield allowances for eligible services provided the subscribers whose incomes are below the level stated for each type of subscribers—single or family—*will consti-*

tute payment in full. If the annual income is above these levels the subscriber is responsible for the balance of the doctor's usual fee."

This means that the Blue Shield allowance represents payment in full to the participating physician for the medical obligations of family subscribers with annual incomes not in excess of $5,000.00 per year and single subscribers having annual incomes not in excess of $3,600.00 per year. It is estimated that approximately 75 per cent of the gainfully employed people of the State of Minnesota have annual earnings within these income levels. It is reasonable to assume therefore that this plan, if it finds wide public acceptance, will in effect establish fees for 75 per cent of the population of this state.

Large segments of Minnesota physicians are alarmed by the introduction of this drastic change in Blue Shield coverage. The so-called full coverage under the proposed plan means absolute regulation of physicians' fees not only for its indigent and low income groups but also for the average man and necessarily for the above average as well! Inequities that were of little consequence affecting a small indigent group can no longer be ignored. In particular, the nonoperating specialties, internal medicine, dermatology, neuropsychiatry, pediatrics, and anesthesiology will suffer. However variously the new plan may create economic burdens, groups within the entire profession must suffer equally the loss of independence. Several groups have already voiced their opposition to the plan. Both the Minneapolis and Saint Paul Societies of Internal Medicine have had special meetings expressing their opposition. When the proposed plan was presented by officers of the Blue Shield recently before a special meeting of the Minneapolis Asbury Hospital Staff, every comment was critical.

It is evident that large sections of the membership of the Minnesota State Medical Association, probably the majority, are opposed to extension of the complete fee service to a $3,600.00 and $5,000.00 level.

It seems proper, therefore, to suggest to the Board of Directors that this is not an opportune time to inaugurate this new plan. It might be further suggested to them that they more carefully take soundings of medical opinion before making drastic changes in the Blue Shield contracts.

R. Berman, M.D.

THE ANNUAL MSMA MEETING

The annual meeting of the Minnesota State Medical Association to be held June 7, 8, and 9 in Duluth shows every promise of being a successful one by everyone's standards. You can help make it successful by sending in your reservations for housing, for specialty meetings, and for special events *early*.

The hotel managers have given assurance that housing for the meeting will be even better than in 1950. You are urged to minimize any causes of discontent by:

1. Mailing or telephoning in your reservations early.
2. Stating your preference, and an alternate or two, of type of accommodations.
3. Stating your checking out time, if possible.

Since reservations will be held until 4:00 P.M., it is suggested that you send a deposit with your reservation request, if you think you will sign in *after* 4:00 P.M. (Deposit will be returned, if the reservation is cancelled before 4:00 P.M.)

Minnesota State Medical Association

101st Annual Meeting

Duluth, Minnesota
June 7, 8 and 9, 1954

Preliminary Program

BUSINESS SESSIONS

SATURDAY, JUNE 5
Hotel Duluth

2:00 P.M.—Council.................... English Room
6:00 P.M.—Council.................... English Room

SUNDAY, JUNE 6
Hotel Duluth

8:30 A.M.—Council.................... English Room
10:00 A.M.—Reference Committees................ Rooms to be assigned
1:30 P.M.—House of Delegates.............. Ballroom
8:00 P.M.—House of Delegates.............. Ballroom

MONDAY, JUNE 7
Hotel Duluth

8:30 A.M.—Council.................... English Room
12:15 P.M.—House of Delegates............. Ballroom

TUESDAY, JUNE 8
Hotel Duluth

8:30 A.M.—Council.................... English Room
8:30 A.M.—Committees......................... Rooms to be assigned

WEDNESDAY, JUNE 9
Hotel Duluth

8:30 A.M.—Council.................... English Room
8:30 A.M.—Committees......................... Rooms to be assigned

SOCIAL EVENTS

SUNDAY, JUNE 6

Buffet Supper

American Medical Women's Association, Inc., Minnesota Chapter

6:30 p.m., Home of Elizabeth Bagley, M.D., 2524 Branch Street
Open to all visiting women doctors. Dr. Marie Bepko will show colored slides from the Fourth Congress of the Pan-American Medical Women's meeting in New York City.

MONDAY, JUNE 7
Luncheons

Minnesota Chapter, American College of Chest Physicians
12:15 p.m.

Minnesota Medical Foundation
12:15 p.m.
Speaker: Clarence Dennis, M.D., New York

Dinners

Minnesota Academy of General Practice

6:00 p.m., Ballroom, Hotel Spalding

Open House, Minnesota State Medical Association
9:00 p.m., Ballroom, Hotel Duluth

Minnesota Radiological Society
6:00 p.m., Social Hour
7:00 p.m., Dinner
Speaker: Philip J. Hodes, M.D., Philadelphia—"An Evaluation of Body Section Radiography"
Northland Country Club, 39th Avenue East and Superior Street. Tickets, $5.00. Reservations should be made with Dr. John R. McNutt, 824 Medical Arts Building, Duluth.

TUESDAY, JUNE 8
Dinners

Minnesota Society of Clinical Pathologists
6:00 p.m., Annual Dinner and Business Meeting—House of Dr. A. H. Wells

Annual Banquet, Minnesota State Medical Association
7:00 p.m., Ballroom, Hotel Duluth

12:15 ROUND TABLES

Duluth and Spalding Hotels

MONDAY, JUNE 7

Anesthesia as a Maternal Mortality Factor
Frederick J. Hofmeister, Milwaukee, Wisconsin

TUESDAY, JUNE 8

Use of Tracheotomy in Chest Injuries
W. P. Eder, Minneapolis
Treatment of Burns in Children
Tague C. Chisholm, Minneapolis
Emergency Treatment of Nerve Injuries
Robert L. Merrick, Saint Paul
Indications and Contraindications for the Use of the Hormones (ACTH and Cortisone) in Gastrointestinal Disorders
J. Arnold Bargen, Rochester

Skin Manifestations in Systemic Disease
John F. Schmid, Duluth

Acute Urinary Obstruction in the Male
Otto C. Phares, Saint Cloud

Drugs in Peptic Ulcer
E. E. Gambill, Rochester

The Injured Hand
J. C. Feuling, Duluth

WEDNESDAY, JUNE 9

Diagnostic Difficulties in Carcinomas of the Lungs
F. F. Callahan, Saint Paul

Evaluation of Operations for Peptic Ulcer
C. W. Lillehei, University of Minnesota

Relation of Blood Volume to Gastrointestinal Hemorrhage
J. E. Twomey, Minneapolis

Practical Application of Cytologic Technique for Malignancy
A. H. Wells, Duluth

Anesthesia in Tonsillectomy
F. C. Jacobson, Duluth

Functional Uterine Bleeding
R. F. Sturley, Saint Paul

Treatment of Plantar Warts
R. W. Goltz, Minneapolis

Diagnosis and Treatment of Blood Dyscrasias
Paul S. Hagen, Saint Paul

GENERAL SESSION

Duluth Armory

MONDAY, JUNE 7

A.M.

8:30 Visit Scientific and Technical Exhibits.... Armory

9:00 TREATMENT OF HYPERTENSION
Moderator—R. M. Shick, Rochester
Henry A. Schroeder, Saint Louis, Missouri
A. C. Corcoran, Cleveland, Ohio
Robert W. Hollenhorst, Rochester
Question and Answer Period

Intermission

10:15 Visit Scientific and Technical Exhibits.... Armory

11:00 Plastic Operations for Hydronephrosis
Frederic E. B. Foley, Saint Paul
Comments
Ormond S. Culp, Rochester

11:40 Staphylococcal Sepsis; Control with Antibiotics
Robert I. Wise, Assistant Professor, Department of Medicine, University of Minnesota

P.M.

12:15 Miscellaneous Luncheons
Round-Table Luncheon

1:30 Visit Scientific and Technical Exhibits..Armory

2:00 (To be announced)

2:30 RUSSELL D. CARMAN MEMORIAL LECTURE
Pitfalls in the Diagnosis of Pulmonary Diseases
Philip J. Hodes, Professor of Radiology, School of Medicine, University of Pennsylvania, Philadelphia

Intermission

3:15 Visit Scientific and Technical Exhibits..Armory

4:00 SYMPOSIUM ON ANESTHESIOLOGY
Moderator—Ralph T. Knight, Minneapolis

Hypotension During Surgery and the Immediate Postoperative Period
Ferdinand C. Jacobson, Duluth

Cardiac Arrest During Anesthesia
James H. Matthews, Minneapolis

Will Analgesia Evolve from a Century of Anesthesia?
John S. Lundy, Rochester

Trichlorethylene—Its Possibilities for the General Practitioner
O. Sidney Orth, Professor of Pharmacology, University of Wisconsin, Madison

5:00 Visit Scientific and Technical Exhibits..Armory

6:00 Special Dinners

9:00 Open House....................Hotel Duluth

GENERAL SESSION

Duluth Armory

TUESDAY, JUNE 8

A.M.

8:30 Visit Scientific and Technical Exhibits..Armory

9:00 SYMPOSIUM ON PROBLEMS ARISING IN THE CLINICAL USE OF CORTISONE, HYDROCORTISONE AND CORTICOTROPIN

Problems Arising in the Treatment of Adrenal Insufficiency
Edmund B. Flink, Minneapolis, Moderator

Untoward Effects Upon the Gastrointestinal Tract
Eric E. Wollaeger, Rochester

The Use, Abuse and Limitations of Steroid Therapy in Dermatology
Francis W. Lynch, Saint Paul

Problems of Dosage in Treatment of Rheumatoid Arthritis
L. Emmerson Ward, Rochester

Problems Arising From Suppression of Adrenal Cortical Function by Cortisone and Hydrocortisone
Robert M. Salassa, Rochester

Pathology of the Pituitary and Adrenals
Warren A. Bennett, Rochester

Question and Answer Period

Intermission

10:15 Visit Scientific and Technical Exhibits..Armory

11:00 The Clinical Management of Fulminant Infections in Childhood
Edward B. Shaw, Clinical Professor of Pediatrics, University of California Medical School, San Francisco

11:30 ARTHUR H. SANFORD LECTURESHIP IN PATHOLOGY
John R. Schenken, Professor of Pathology, University of Nebraska Medical School, Omaha

P.M.

12:15 Miscellaneous Luncheons
Round-Table Luncheons

1:30 Visit Scientific and Technical Exhibits..Armory

2:00 ULCERATIVE COLITIS

Medical Aspects
J. Arnold Bargen, Rochester

Psychiatric Aspects
L. R. Gowan, Duluth

Surgical Aspects
Clarence Dennis, New York

Question and Answer Period

Intermission

3:15 Visit Scientific and Technical Exhibits..Armory

4:00 SYMPOSIUM ON FRACTURES OF THE SPINE AND LOWER EXTREMITIES

Fractures of the Spine and Pelvis
John C. Ivins, Rochester

Fractures About the Knee Joint
Harry B. Hall, Minneapolis

Fractures of the Tibia and Fibula in Adults
Malvin J. Nydahl, Minneapolis

Fractures of the Lower Extremity in Children
S. S. Houkom, Duluth

5:00 Visit Scientific and Technical Exhibits..Armory

7:00 Annual Banquet......Ballroom, Hotel Duluth

GENERAL SESSION

Duluth Armory

WEDNESDAY, JUNE 9

A.M.

8:30 Visit Scientific and Technical Exhibits..Armory

9:00 Diagnosis and Treatment of Hemolytic Anemia
Paul S. Hagen, Saint Paul

Anemias in Pregnancy
Roy G. Holly, University of Minnesota

Plasma Expanders
A. R. Curreri, Associate Professor of Surgery, University of Wisconsin Medical School, Madison

Question and Answer Period

Intermission

10:15 Visit Scientific and Technical Exhibits..Armory

11:00 Minnesota Division, American Cancer Society

P.M.

12:15 Round-Table Luncheons

1:30 Visit Scientific and Technical Exhibits..Armory

2:00 WHAT'S NEW?

Moderator—F. J. Hirschboeck, Duluth

In Medicine
C. J. Watson, Professor and Head, Department of Medicine, Medical School, University of Minnesota

In Surgery
Owen H. Wangensteen, Professor of Surgery and Chairman, Department of Surgery, Medical School, University of Minnesota

In Radiology
Leo G. Rigler, Professor and Head, Department of Radiology, Medical School, University of Minnesota

In Pediatrics
Irvine McQuarrie, Professor and Head, Department of Pediatrics, Medical School, University of Minnesota

In Obstetrics and Gynecology
John L. McKelvey, Head, Department of Obstetrics and Gynecology, Medical School, University of Minnesota

Question and Answer Period

SPECIAL SESSIONS

MONDAY, JUNE 7

A.M.

10:00 Minnesota Academy of Ophthalmology and Otolaryngology, St. Luke's Hospital Auditorium

Hearing Loss in Children—Detection, Treatment and Rehabilitation....................
Kinsey M. Simonton, Rochester

Chronic Middle Ear Suppuration—Today......
L. R. Boies, Minneapolis

Retrolental Fibroplasia.......................
Erling Hansen, Minneapolis

Some Types of Retinal Vascular Occlusions....
Henry P. Wagener, Rochester

P.M.

1:00 Round Table Luncheon
Discussion on Vertigo.......................
Terrance Cawthorne, London, England

TUESDAY, JUNE 8

A.M.

9:00 Minnesota Society of Clinical Pathologists
Tumor Seminar..............St. Mary's Hospital

Moderator—Benjamin Castleman, Professor of Pathology, Harvard Medical School, Boston, Massachusetts

11:30 Arthur H. Sanford Lectureship in Pathology..
Duluth Armory

P.M.

12:00 Luncheon....................St. Mary's Hospital

2:00 Tumor Seminar (Continued)..................
St. Mary's Hospital

6:00 Annual Dinner and Business Meeting

Plan to attend the

annual meeting

at Duluth

You will learn much

that is new and

of interest

The Dean's Page

THE DEPARTMENT OF MEDICINE

From time to time on this page, we want to bring you information concerning the teaching and research activities of the various departments and divisions of the Medical School. The Department of Medicine is one of the larger departments and carries an extemely important responsibility for teaching. It is composed of three divisions: Internal Medicine, Dermatology, and Clinical Laboratory Medicine. These divisions are directed, respectively, by Doctors Cecil J. Watson, Henry E. Michelson, and Gerald T. Evans. Dr. Evans is also in charge of the Laboratory Service of the Hospital. The Division of Clinical Laboratory Medicine is responsible for teaching clinical chemistry and microscopy to the sophomore medical students and for teaching the application of laboratory methods to clinical medicine during the junior year.

In addition to the course in clinical chemistry and microscopy, the department is responsible for the following teaching:

1. *Physical Diagnosis,* given in the winter quarter of the sophomore year. Because this course constitutes the student's first contact with patients, it is of particular importance. Students study physical signs at the bedside at the University Hospital and either at the Veterans Hospital, the Minneapolis General, or the Ancker Hospital in St. Paul.

2. *Introduction to Clinical Medicine.* This is a "didactic" course which meets for two hours weekly during the first two quarters and one hour weekly during the third quarter of the Junior year. Students are provided with a lecture syllabus (*The Outlines of Internal Medicine*) which covers the basic material and, for the most part, the class hours are devoted to clinics and demonstrations and discussion of new advances.

3. *The In-Patient Clinical Clerkship* in the Junior year. Students are assigned for ten weeks to the Department of Medicine. Each student spends half of this time at the University Hospital studying patients under close supervision on the wards. Their detailed histories and physical examinations are incorporated into the charts. A student at the University Hospital is responsible also for a considerable amount of the laboratory work. This is carefully supervised. Each student devotes the other half of his medical clerkship to similar clinical work either at the Minneapolis General Hospital or the Veterans Hospital.

4. *The Out-Patient Medical Clerkship* in the Senior year. The student is assigned a new patient presenting for diagnosis and treatment at the Out-Patient Medical Clinic. After taking a careful history and making the physical examination in detail, he sees the patient with one of the staff. After a thorough discussion of the problem and a review of the findings, a decision is reached as to disposition of the patient, i.e., whether the patient should be admitted to the hospital, what further

laboratory or x-ray studies are desired, etc. On the patient's next visit to the Out-Patient Clinic, he will be seen by the same student and staff member who will again attempt to arrive at a definite diagnosis and plan of therapy It may be that a third visit will be required because additional laboratory work or x-ray studies are necessary. Often the patient can be discharged to return to the local physician. In this event, the student prepares the letter to the referring physician, which is reviewed and signed by the staff member. This system provides continuity in follow-up.

In addition, the Department of Medicine is responsible for the admissions service of the hospital. All new patients are worked up either through the system just referred to or, if emergencies, by the medical intern and fellow on call in the admissions room of the hospital.

The staff of the Department represents a number of interests in the broad field of Internal Medicine, and it should be emphasized that every effort is made to sustain the philosophy of internal medicine as a broad discipline in which the patient is regarded as a whole and not from the standpoint of narrow, segregated specialties . There are no specialty services in medicine at the University Hospital. It is true that one of the services in the Variety Club Heart Hospital places more emphasis on cardiovascular disease but students, both undergraduate and graduate, and staff rotate through all services so that a broad integration is maintained.

Research activities in the department are on a rather broad scale. These include studies of brucellosis, staphylococcus infections, chemotherapy, various aspects of liver disease, pigment metabolism, coagulation mechanisms, hypertension (both therapy and fundamental aspects), electrocardiography, metabolic problems, cancer biology, and steroid and mineral metabolism. The fellows in the department are invited to conduct research in collaboration with graduate advisors or with their supervision and advice, either in the areas which have just been mentioned or along some other avenue which is mutually acceptable.

Teaching, service to patients, and research are co-ordinate and interdependent activities in the Department of Medicine as they are in other clinical departments. Each week the department holds a number of conferences and seminars as well as ward rounds. All physicians are cordially invited to attend any of these sessions.

Harold S. Diehl, M.D.
Dean of the Medical Sciences

Medical Economics

Edited by the Committee on Medical Economics
of the
Minnesota State Medical Association
George Earl, M.D., Chairman

ROCHESTER DOCTOR RECEIVES NATIONAL AWARD

Dr. Frank H. Krusen, Rochester, Minnesota, received the Presidential Award of 1953 for his contributions to the employment welfare of the handicapped.

The citation was presented to Dr. Krusen at the annual dinner of the Industrial Health Congress in Louisville, Kentucky, February 24 and 25. Dr. Krusen received the award from Dr. Ross T. McIntire, Washington, president of the President's Committee on Employment of the Physically Handicapped.

In accepting the award, Dr. Krusen said that "proper restoration of handicapped persons depends on three things: (1) appropriate; definitive treatment and physical rehabilitation of the handicapped person in the hospital; (2) proper vocational rehabilitation, and (3) well-established programs for employment of the handicapped." Dr. Krusen is head of the section on physical medicine at the Mayo clinic.

New Light on Problems

The 14th annual Congress on Industrial Health featured discussions of wide interests to physicians, management representatives and labor leaders. Problems encountered in the maintenance of the health of factory and white-collar workers were topics of interest to those attending. Panel subjects included the stress effects on workers in industry, covering disorders such as neuroses, heart conditions, and ulcers; the effect of prolonged illness occupied another panel subject; the preventive medicine program carried out in some of the duPont plants; industrial implications of emphysema, including the clinical aspects, pathologic physiology, histopathology, radiography and therapy; the difficulties of small plant operators in providing medical care.

Improvements Noted

Frank G. Dickinson, Ph. D., Chicago, director of the AMA's Bureau of Medical Economic Research, reported that improvements in medical service have greatly increased the economic value of industrial workers. Said Dr. Dickinson:

> "In 1900, perhaps one-half of the industrial workers twenty years of age could expect to live to the age of sixty-five. Now, more than four-fifths of them are destined to reach that retirement period.
>
> "The decline in mortality during the working years of life has contributed greatly to economic progress. It has contributed to the solution of some social problems—the probability of becoming an orphan, for instance, has been cut in half."

A joint conference of the Council on Industrial Health and chairmen of state medical society committees on industrial health was held on the day before the opening of the congress. The meeting was sponsored by the American Medical Association's Council on Industrial Health, and physicians and others from the entire country attended to hear of these important phases of industrial health and medicine.

UNION HEALTH PLANS GET "ONCE-OVER"

A greatly intensified interest in health and welfare plans of labor unions has sprung up within recent years. An excellent example of this interest is seen in a new survey published by the American Medical Association called "Union Health Centers." This survey traces the growth of present plans and notes in the introduction that during and since World War II a much broader and deeper system of such plans has been growing.

It is interesting to note that the pros and cons of such plans have also been presented recently by the *Wall Street Journal.* In a series of articles on the Teamsters Union, the *Journal* states:

> "A deep concern for 'health and welfare' has seized leaders of the giant Teamsters Union.
>
> "In the brief period since World War II they've rushed to arrange a bewildering variety of insurance and direct medical service programs."

The article goes on to relate that the interest does not confine itself to the unions themselves, but is manifested in local grand juries and by no less than three Congressional committees. Most of this interest is directed toward finding out about the disposition of certain union funds. One such investigation is being made into the Labor Health Institute in St. Louis, whose finances are being questioned.

According to the *Journal*:

> "The institute has its own elaborately equipped five-story clinical building where union members and their families can get their teeth fixed, have allergies treated, their throats sprayed, their bones x-rayed, and buy drugs. It finances service in hospitals, as well. Nearly a million dollars annually is paid in by 175 companies and employer groups whose contracts with the union provide that either 5 per cent or 3½ per cent of their gross payrolls shall go to L.H.I. The larger figure appears in contracts providing full coverage for the dependents of union members."

No Trouble Elsewhere

While the *Journal* goes into detail about union leaders being jailed for failure to produce books on health plans, another similar plan, with infinitely different purposes and consequences, has been reviewed. The *Wall Street Journal* itself has featured a story about a mass check-up which is praised by firms, unions and employes in Los Angeles. Called a project in preventive medicine, the plan consisted of one thousand workers at two manufacturing plants being given comprehensive, on-the-job physical checkups that didn't cost them anything. In the *Journal's* words:

> "Hoffman Radio Corp.'s television division, with 500 employes, and its neighbor, Leach Relay Co., Inc., with an equal number of workers, offered the health survey as a means of ferreting out prevalent, but often unrecognized, ailments ranging from defective vision to heart trouble or lung cancer."

The company's personnel manager praised the plan: "It's a benefit to the employes, and we believe in the long run it will also benefit the company in terms of better employe relations and reduction of lost time due to illness."

Medical Support Given

The idea for the mass employe health screening came from the University of California at Los Angeles, which suggested it to union representatives. It was tried without fanfare and turned out to be successful enough to warrant a thorough test. The *Journal* reports:

> "An unusual demonstration of community teamwork by local health organizations kept the actual cash outlay for the survey to $500—or $100 apiece from each of the two companies and the three unions."

Participating organizations and their contributions were: American Cancer Society (pamphlets, and review of some chest x-rays); California Department of Health (equipment for blood tests); Los Angeles City Health Department (equipment and medical advice); Los Angeles County Diabetes Association (equipment, and blood sugar tests at minimum cost); Los Angeles County Heart Association (educational material, electrocardiograms); Los Angeles County Medical Association (co-ordination of projects); Los Angeles County Tuberculosis and Health Association (x-ray equipment, film, technician); University of Southern California Research Laboratory (co-ordination of diabetes tests); University of California at Los Angeles (its Institute of Industrial Relations acted as catalyst in getting companies, unions and other agencies together).

The plan drew favorable comments from all of those participating, union officials, company officials, agency officials and employes themselves. The plan was a sample of what can be done if all concerned work together to promote a worthy project for the good of all. Those who worked out the project feel that if used in other areas, care should be taken to see that all agencies are in on it and that all have a part in planning and operation.

COMMISSION BRINGS RECOMMENDATIONS

The Commission on Financing Hospital Care has recently published its recommendations, after two years of intensive study and investigation. The thirty-four member commission completed its study with contributions amounting to $556,000 from the Blue Cross commission, Health Information Foundation, John Hancock Mutual Life Insurance company, W. K. Kellogg Foundation, Michigan Medical service, Milbank Memorial Fund, the National Foundation for Infantile Paralysis and the Rockefeller Foundation.

Pointing out that in eight states three-fourths of the population are participating in voluntary prepayment plans for defraying hospital expenses, the Commission states:

> "The financing of hospital care and the maintenance of standards of hospital service will depend increasingly upon maximum coverage of the population with voluntary prepayment at the lowest possible cost compatible with the inclusion of adequate benefit provisions."

The Commission report then goes on to make the following recommendations:

1. Hospitals should take the initiative in establishing local study committees to evaluate all prepayment plans in their area. Recommendations for improvement should be made to the prepayment agencies, the hospitals and the public.

2. Prepayment agencies should take the initiative in mobilizing community leadership to inform the public concerning the frequency of hospitalized illness, the rôle of voluntary prepayment in meeting costs, factors affecting cost, and benefit provisions.

3. Methods should be developed to enroll the following who are not presently covered by prepayment in sufficient number: dependents of present contract holders such as infants, children over 18, parents and other relatives; the self-employed; farm operators and employes; employes of small firms; employes who retire under pension plans; employes temporarily without employment because of short layoffs or change of employers; domestic employes; migratory workers; persons receiving workmen's compensation benefits.

4. Employer participation in costs of prepaid protection should be encouraged.

5. Methods should be explored for the coverage of individuals and families by prepayment during periods of temporary unemployment.

6. Methods should be explored for continuation of coverage during periods of prolonged disability.

7. Programs might be developed for coverage of individuals and families who are without income from employment.

8. Low-income families should be encouraged to budget through prepayment for the cost of adequate protection. Experimental programs should be instituted to reduce the cost of prepayment to bring protection within reach of this group.

9. Hospitals, prepayment agencies and community groups should support, promote and interpret administrative practices in the operation of prepayment which help attain maximum coverage.

10. Hospitals and community groups should aid in enrollment policies and techniques to permit those not covered to attain prepayment protection.

11. Hospitals should help conduct area-wide studies of prevailing benefit provisions and their effect upon the financing of hospital care. Recommendations should then be made to the public regarding basic benefit provisions required for adequate protection of public and hospitals.

12. Hospitals, co-operating with others, should assume leadership in developing measures and educational programs designed to assure most efficient and economical use of prepayment funds. Consideration should be given to efficiency in provision of hospital services, unnecessary use of hospital services, appropriate use of hospital services and efficiency in operation of the prepayment agency.

OFFICE HEMATOLOGY

(Continued from Page 279)

The majority of stain trouble is *buffer* trouble. We use the following buffer adjusted to a pH of 6.4: potassium phosphate, monobasic, 6.63 gm.; sodium phosphate, dibasic, anhydrous, 2.56 gm.; distilled water to 1,000 cc. Check the pH of the buffer frequently with several crystals of hematoxylin in a test tube of buffer solution. A pink-lavender color develops in two minutes if the buffer is satisfactory. Red-purple means it is too alkaline; yellow means it is too acid. Adjust with weak acid or bicarbonate as indicated. The stain-buffer times varies with each new batch of stain. A good stain is pink on the slide. Erythrocytes should be light buff; the nuclei are blue-purple and are in sharp contrast to the cytoplasm. Eosinophilic granules are bright red and neutrophilic granules are pink to lavender.

No degree of diagnostic acumen can correct for a poor smear or a poor stain. When sending blood smears for consultation, send one stained and one unstained smear with pertinent clinical and hematologic data. Give the age of the patient. Bone-marrow preparations require exacting and special technique and are not office procedures. It's not "old fashioned" to look at your own blood smears. You cannot afford to ignore them!

Minneapolis Surgical Society

Meeting of December 3, 1953

The Vice President, Maynard Nelson, M.D., in the Chair

SPONTANEOUS RUPTURE OF THE LIVER

A Case Report with Recovery

L. A. STELTER, M.D., and G. L. PETERSEN, M.D.
Minneapolis, Minnesota

THE FIRST known case of spontaneous rupture of the liver was reported by Vesalius at some undetermined date. Andral, in 1829, sketchily reported two cases with complicating lesions which may have been gummata or carcinoma. However, in many of these earliest cases, there is some probability that gastric hemorrhage, secondary to liver disease, may have been mistaken for actual rupture of the liver. According to Devic and Beriel and later Mazel, the following theory had been formulated: "In traumatic rupture, the rupture is the cause of hemorrhage. In spontaneous rupture, the hemorrhage is the cause of rupture."

Rupture of the liver is a result of spontaneous tearing or trauma. The chief traumatic causes are: (1) blunt force, (2) open injuries, such as stab wounds, gunshot wounds and war injuries.

Of all the organs in the abdominal cavity, the liver is the most likely to be injured when the abdomen is hit in any part by blunt force. Street and automobile accidents; falls from scaffolding; burying accidents; war injuries and hoof beats are the main causes. In 494 postmortem examinations of rupture of abdominal organs through blunt force, liver rupture rates first with 59.9 per cent. The right lobe has been found ruptured much more frequently than the left. The relationship is about 65 to 35.

Spontaneous rupture of the liver is due to enlargement of the liver from disease within stretching Glisson's capsule, which has a certain degree of tension. The chief diseases which may cause the liver to burst are fatty degeneration, malaria, tuberculosis, syphilis and malignancy. It occurs when the liver is more friable and during digestion when it becomes hyperemic and enlarges.

The greatest danger in liver rupture is hemorrhage. In fact, this is the main cause of a fatal outcome. The large intrahepatic veins have very thin walls, no valves are present, and there is no retractibility, therefore the bleeding is persistent and spontaneous control is rare. Because of the large venous blood supply in the liver, the hemorrhage is more likely to be of venous nature and enormous amounts of blood may be found in the abdominal cavity.

Extensive liver injuries have a very high mortality rate and statistics from the war give a lower mortality rate (60 to 80 per cent), which may be erroneous, as a considerable number die immediately on the battle field because of massive hemorrhage.

Clinical signs of ruptured liver and differential diagnosis are: (1) symptoms of general shock, (2) general symptoms of abdominal injuries, (3) special symptoms caused by the injured liver.

Shock. The patient lies still with an expression of fear in the face. The skin is pale and clammy; the mind is clear, but mental activity is diminished. The respiration is shallow and frequent, deep sighs interrupting normal respiration. The pulse at first is full and bounding, later thready and rapid, and the blood pressure progressively falls.

Special Signs of Liver Injury. When loss of blood occurs in the abdominal cavity, peritoneal irritation occurs immediately, the intensity depending on the rapidity of intra-abdominal hemorrhage. Pain after rupture of the liver, is quite characteristic. It is especially felt when pressing the finger into the intercostal space. Pain radiates downward and backward and also into the right shoulder. This referred pain is due to diaphragmatic irritation through the phrenic nerve and into the cutaneous branches of the dorsal scapular nerve. Breathing is shallow, more fre-

TABLE I. CAUSES OF RUPTURE OF THE LIVER OTHER THAN VIOLENT MUSCULAR EFFORT*
*From Annals of Surgery, 118: 397, (Sept.) 1943.

Date	Author	Age	Cases	Operation	Cause	Result
1829	Audral		2	Autopsy	Carcinoma or gumma	Died
1831	Honore		1	Autopsy	Multiple caries	Died
1836	Gerard	52	1	Autopsy	None	Died
1844	Sacquepec	44	1	Autopsy	Syphilis	Died
1847	Fauconneau & Dufrense		1	Autopsy	Typhoid fever	Died
1849	Abercrombie	35	1	Autopsy	Pregnancy	Died
1864	Heinzdman		2	Autopsy	Tuberculon Peritonitis	Died
1884	Chrari	40	1	Autopsy	Cancer	Died
1886	Heinzdman	18	1	Autopsy	Pneumonia	Died
1890	Laureneau	16	1	Autopsy	Syphilis	Died
1892	Pauly	47	1	Autopsy	Biliary colic	Died
1892	Poljakoff		1	Autopsy	Epilepsy	Died
1898	Bar & Reim & Neubert		2	Autopsy	Syphilis	Died
1899	Carnabel	26	1	Autopsy	Osteitis	Died
1902	Bernard	62	1	Autopsy	Hemiplegia	Died
1905	Nauwerck		1	Autopsy	Common duct stone	Unknown
1906	Derri & Beriel	41	1	Autopsy	Syphilis	Died
1921	Walz		1	Autopsy	Aneurysm	Died
1932	Rethon & Cadein	34	1	Operation	Exercise & cough	Recovered
1933	Mast & Streamer		1	Autopsy	Pneumonia: Ca. of liver	Died
1934	Robin	28	1	Operation	Trauma	Recovered
1936	Bradley & Garrett	3	1	Operation	None	Recovered
1937	Sciacia		1	Autopsy	Toxic infection	Died
1938	McEwan	8	1	Operation	Malaria	Died
1940	Coniden	9	1	Operation	Slight trauma	Recovered
1943	Rademaker	32	1	Exploratory	Pregnancy	Recovered
1946	Links	42	1	Operation	Pregnancy	Recovered
1946	Sanes and Kaminiski		1	Autopsy	Pregnancy and eclampsia	Died
1951	Haller, Abels & Straus	34	1	Autopsy	Eclampsia	Died
1953	Wright and Ball	61	1	Operation	Hypertension	Recovered

quent, and may be painful. Dullness in the right side of the abdomen gradually increases and spreads downward as the blood flows down the right abdominal gutter. The dullness may shift if the patient is turned to the left. Rectal or vaginal or rectovaginal examination reveals fullness in the pelvis and tenderness. Bradycardia may last longer than in other types of intra-abdominal hemorrhage and this is due to resorption of bile from the abdominal cavity. Bile salts have a depressing influence on the heart. Diagnosis of bile salts may be made by tapping the abdominal cavity with a No. 20 spinal needle; a procedure which I have done on various occasions of abdominal injury or suspected ectopic pregnancy. No harm will be done to the small or large bowel where the abdomen is free from adhesions. The examination of the blood for bile and bile salts and for liver cells will be helpful. X-ray examinations, upright and flat abdomen, will only help in ruling out air under the diaphragm in rupture of gas containing viscera. Auscultation reveals a near silent abdomen.

Complications. (1) death from hemorrhage, (2) cholascos (effusion of bile into the abdominal cavity) causing bile peritonitis, (3) permanent biliary fistula, (4) infection of the liver wound, (5) embolism of liver tissue, (6) infarction of the liver, (7) lung complication—pneumonia (due to emboli), (8) hepatorenal syndrome (nephrosis of the kidneys), (9) eye symptoms, simulating albuminuria retinitis.

Treatment. From the statistics of Elder, Terrier, Auvray and others, who have shown that the mortality in non-operated cases was as high as 85.9 per cent, while in the past decade, with modern surgical intervention, the mortality has been reduced from 53.8 per cent to 28.5 per cent, we feel that this is an acute urgent surgical entity. The patient should be transfused by multiple portals of blood and operated upon immediately.

Case Report

The patient, a white female teacher, aged twenty-three, a student at summer school, gave the following history: While eating her Sunday dinner with a friend at 2:00 P.M., she was suddenly stricken with an acute pain in the right shoulder. She was unable to finish her meal and had to be assisted to a lounge where she lay for a short time having a lady massage her shoulder because of the pain. She then experienced pain in the upper abdomen—severe and intense—vomited her dinner and retched several times, after which she felt relieved for about an hour. During this time she was taken to her room, at a Girls' club, and went to bed, complaining to her roommate that her abdomen was getting more and more painful, that she felt faint and weak, wanted to vomit, and that she had a severe backache.

I saw the patient at 5 o'clock p.m., a tall, fairly well-developed female, holding her hand on her upper abdomen. She was somewhat pale, the examination revealed she had a normal pulse, blood pressure was 70/30, heart and lungs were normal, abdomen was held tense and with boardlike rigidity above the navel and across to the right lower quadrant. Vaginal examination was negative. She was sent to Fairview Hospital by ambulance and upright and flat x-rays revealed no air under the diaphragm. Hemoglobin—70 per cent; R.B.C.—3,700,000; W.B.C.—16,000; P.M.N.'S—94 per cent; lymphocytes—6 per cent. Urinalysis revealed sugar—four plus also acetone and diacetic acid. There were 40-50 R.B.C.s and pus cells—20-30 per high powerfield pus cells. Re-examination of her abdomen at 8 o'clock p.m. by Dr. Petersen and myself, revealed a benign abdomen. There was no rebound tenderness and no rigidity at this time. The patient laughingly remarked, "Why do I have to stay in the hospital? My pain all left me when I came here in the ambulance."

We decided to treat the patient conservatively on the basis of a diagnosis of sealed perforated posterior duodenal ulcer, complicated by a severe diabetes and pyelitis. Nasal suction was instituted and Dr. Petersen treated the diabetes by I.V. glucose and large doses of insulin. Re-counts of laboratory work were ordered for the following morning. Her blood pressure and pulse were observed at hourly intervals during the night and no change was noted. At 7:30 a.m. the next morning no change was noted except for some abdominal distention. On rectal examination some fullness was felt in the lower pelvis. Re-check of x-rays and I.V. pyelogram were ordered.

At 11 o'clock a.m. an acute change in her condition was apparent. The pulse became rapid. She had become pale and clammy. The vaginal examination, at this time, revealed marked tenderness. I then made a diagnosis of acute abdominal catastrophe, as the laboratory at this time, reported that her hemoglobin had dropped to 55 per cent and the red blood cell count was 2,500,000. A diagnosis of intra-abdominal hemorrhage was evident. An emergency operation was performed.

Under spinal anesthesia, with two units of bloods and normal saline running in the veins, a "peak" right paramedian incision was made. On finding the abdomen full of blood, the incision was continued down to the pelvis and both tubes and ovaries explored and found normal. Exploring the right side and following up to the liver, I found that the girl had a ruptured right lobe of the liver. The incision was extended to the xyphoid and the right rectus muscle cut across to the right costal margin, the blood evacuated by suction, approximately 1 quart, and a rent, the size of my hand, was found in the right lobe, extending up to the dome of the liver. The entire surface of the right lobe was dark from subscapsular hemorrhage and echymosis. The active bleeding, which was profuse from the liver rent, was then controlled by large gauze packs. These were then gradually removed after the patient's pulse and blood pressure stabilized and replaced by oxycel and gel foam, seven such packs were used to fill the rent and the liver was then sutured with mattress sutures. This, however, proved very difficult because of the friability of the organ. Two large cigarette drains were placed between the chest wall and the liver rent and the abdomen was then closed, the peritoneum with chromic 0 doubled, the fascia with through and through wire and cotton. A third transfusion was given after surgery and her immediate postoperative condition was good. Her postoperative progress was satisfactory and her diabetic condition was controlled by I.V. fluids and insulin under Dr. Petersen's supervision. Nasal suction was continued.

A liver biopsy, at the time of surgery, was considered and would have been helpful, but in view of the critical condition of the patient, and the black, discolored liver tissue, I felt the procedure would only add insult to an already badly injured organ.

The remaining postoperative condition of the patient progressed satisfactorily. After the third day, the nasal suction was removed. One drain was removed on the fourth day and the second on the eighth day. The patient left the hospital on the thirteenth day postoperatively and was seen subsequently in the office.

A letter from her on the second month after surgery, reported her to be in fine physical condition.

Comment

In 1943, Rademaker collected twenty-nine cases of spontaneous rupture of the liver, including the case of Vesalius. Since then four cases have been added, three associated with pregnancy and one with hypertension.

Because a biopsy of the liver was not made at the time of surgery, I believe one is justified in assuming that this case of spontaneous rupture was due to some metabolic disturbance of the liver, associated with a mild diabetes.

References

1. Arden, Felix, M.D., M.R.C.P.: Medical Superintendent, Brisbane Children's Hospital, Brisbane, Australia.
2. Bradley, C. E., Garrett, D. L.: Pediat., 776 (July-Dec.) 1936.
3. Haller, A. P., Abel, D. W., Straus, R.: Ingelwood, California. Spontaneous rupture of the liver in patient with non-convulsive eclampsia. Am. J. Obst. & Gynec. Vol. 62, No. 5 (Nov.) 1951.
4. Leuke, Harold, M. B., B.Ch., B.A.O., R.S.A.: King Edward VII Hospital Windsor, "Rupture of the liver, complicating pregnancy." Brit. M. J., 275 (Feb. 23, 1946).
5. Papen, George W., Mikal, Stanley: The treatment of ruptured liver with absorbable hemostatics. New England J. Med. 239:290 (Dec.) 1948.
6. Rademaker, Lee, M.D., Salisbury, M.D.: Spontaneous rupture of the liver complicating pregnancy. An. Surg., 118 (July-Dec.) 1943.
7. Sanes and Kaminiski: Pregnancy and eclampsia, American Journal of Obst & Gynec., 52, 325 (Aug.) 1946.
8. Wright, Gilbert L., M.D., and Ball, John M., M.D.: Salt Lake City, Utah. Rocky Mountain M. J., Vol. 50, No. 4 (April) 1953.

Minnesota Department of Health

GAMMA GLOBULIN (IMMUNE SERUM GLOBULIN)

For Poliomyelitis

Gamma globulin is available for direct intimate contacts, forty years of age and under, of reported cases of clinical poliomyelitis on receipt of the name, age and weight of the contact, and identification of the case of polio. The dose of globulin for poliomyelitis contacts is 0.14 cc of globulin per pound of body weight, to a maximum of 20.0 cc and should be given as early as possible following exposure. It is probably of little value if *ten* days or more have elapsed after exposure.

For Infectious Hepatitis

Gamma globulin is regularly available for close and continued contacts of cases of infectious hepatitis (jaundice). Since the product is of no value once symptoms of the disease have begun, and even late in the incubation period, this means that the product should be used largely in household contacts and those living in small groups where the contact is essentially the same. The dose of globulin for contacts of infectious hepatitis depends on the body weight and is 0.02 cc of globulin per pound of body weight, with a maximum of 2.0 cc for any one individual. Globulin should be given to all household contacts who do not give a history of having had the disease, including adults.

For Measles

Gamma globulin for measles has been available for a number of years and most physicians are familiar with its use for this purpose. Those at greatest risk are children three and under. As a general rule, the amount given is aimed at modification of the disease and the development of permanent immunity by a mild attack. Occasionally it is wise to aim at complete prevention, even though no permanent immunity results. Only contacts who have had a recent definite exposure should receive globulin. Those who have had measles already should not be inoculated.

For modification, the dose of globulin for children definitely exposed to measles is 0.02 cc of globulin per pound of body weight. When given during the first six days following exposure, this amount will usually produce a modified case of measles. If complete prevention is urgent, the dose is five times as large. Gamma globulin should not be given if ten days or more have elapsed after exposure, since its value at that late date is negligible.

For Other Conditions

Globulin is not distributed for other conditions for which its value has not been established. This includes mumps, german measles, diarrhea, or for treatment of symptoms once a disease has developed.

Reporting of Cases

Since the Department's supply of gamma globulin is allocated on the basis of numbers of reported cases, it is important that all cases of the above diseases be reported to the State Board of Health as required by regulation.

LABORATORY EXAMINATION OF GASTRIC WASHINGS FOR TUBERCLE BACILLI

Increasing numbers of patients with tuberculosis are receiving post sanatorium out-patient chemotherapy, extending over long periods. It is important that such patients have careful evaluation of their progress at intervals of three months or so, including x-rays, laboratory studies, and tests for the presence of tubercle bacilli. Usually the latter tests are performed by culturing the gastric washings of the patient.

When gastric washings are collected for culture for tubercle bacilli, the specimen should be immediately put into the special bottle prepared for this purpose. This bottle contains a buffering chemical which neutralizes the acid in stomach washings and inhibits the detrimental action of the gastric acidity on the tubercle bacilli. Unless this is done, the bacilli are soon killed and cultures will show no growth.

The Minnesota Department of Health furnishes these special outfits for the mailing in of gastric washings for culture and urges that the special outfit be used whenever there is to be delay between the collection of the specimen and its inoculation onto culture media in the laboratory.

FATIGUE AND HEALTH

There seems to be little doubt that physical and intellectual fatigue, as well as painful emotions, decrease resistance of the body to infection; but the physiologic mechanisms which determine these important relationships are totally unknown. Despite all this ignorance, however, a belief shines as a beacon to guide our efforts. It is that the defense mechanisms of the body which promote resistance to infection appear to be closely related to the physiologic processes which make for good health—good health being here measured in the terms broadly referred to as well-being by the layman. The inner logic of the program of antituberculosis associations should lead them to become truly health associations. For it can hardly be doubted that, in final analysis, health is a positive attribute rather than merely the absence of disease.—René J. Dubos, Ph.D., American Review of Tuberculosis, July, 1953.

MEDICAL HISTORY OF THE COUNTY OF YELLOW MEDICINE

MILDRED B. LEE
Granite Falls, Minnesota

(Continued from March, 1954, issue)

William Moffat Stratton was born in St. Lawrence County, New York, in 1844. His education began in the common schools of that state, and continued in the schools of Wisconsin when the Stratton family moved west in 1854. He received his medical training at Ann Arbor, Michigan, and at Chicago Medical College which is now a part of Northwestern University. He graduated from the latter school in 1869.

Soon after graduation Dr. Stratton began practice at Ortonville, Minnesota. In 1873 he moved to Minnesota Falls, which was a new settlement on the south bank of the Minnesota River just above Minnesota Falls in Yellow Medicine County. Here he was joined by another physician, Dr. E. N. Falk, and although the two doctors did not practice as partners they were partners in a drug business which they established and carried on for several years.

Drs. Stratton and Falk were both graduate pharmacists as well as physicians.

When Dr. Stratton came to Minnesota Falls the little town was competing for the honor of being named the county seat, and it had made astonishing progress in the short time it had been in existence, boasting a flour mill, hotel, blacksmith shop, two stores, a law office, and several saloons. A newspaper would add to the town's prestige, and Drs. Stratton and Falk became partners in establishing the first newspaper in Minnesota Falls and in the County, the *Minnesota Sentinel.* The first issue appeared October 1, 1874.

The paper was a modest effort; it was a six-column folio with the outer pages filled with local advertisements, some in Norwegian, professional cards of the doctors and lawyers, legal notices, and local news items. The few copies which are on file in the office of the clerk of court contain tax lists, and also a fairly full account of a libel action brought by Dr. N. M. Smith against Editor Hogue of the *Granite Rock.* In 1876 the *Sentinel* was sold to a Mr. Bennet who kept it in publication for another two years, after which it died from lack of support.

When Granite Falls became the county seat the population of Minnesota Falls began to move away from the town. Dr. Stratton moved to Granite Falls about 1877 or 1878, and became a member of the first town council in 1879. In 1881 he became president of the council, and later served as Health Officer of the city and as a member of the Board of Education. In the latter capacity he was one of the board members who chose the site for a school in West Granite Falls, and brought about the erection of the first school building on the present school grounds.

Dr. Stratton married Adeline Sarah Enos, whose father was a carpenter and built many houses in Granite Falls. One of these, Number 235, on Tenth Avenue, is now the home of Paul D. Stratton. Dr. Stratton himself lived in a house on south Prentice Street which is still standing, though somewhat altered, as No. 1165 Prentice.

When he first came to Granite Falls, Dr. Stratton became a partner of Dr. Cyrus P. Fuller who was the first physician to locate in Granite Falls.

Dr. and Mrs. Stratton were kindly, generous people who became popular residents of the village. In youth they were considered a rather handsome couple. Dr. Stratton was of average height, having a rather round face with large and slightly protruding eyes, a full lipped and usually smiling mouth. He walked with a slightly shuffling gait and always carried an umbrella—his own or someone else's; and it was usually in varying states of dilapidation; he liked to have something to carry as he walked along. Usually he walked with down-bent head and his manner was quiet and kindly. He had a keen sense of humor. In temperament he was easy-going; although he kept an extensive set of books he never pressed anyone for payment, and indeed seemed to be indifferent not only to obligations owed to him but to those he himself owed. He was devoted to his patients. He would answer calls at any time of day or night and give freely of his time and effort. Sometimes he suffered keenly over the death of a patient. He was a physician and surgeon of exceptional ability, and neither weariness nor other circumstance ever seemed to affect his skill and judgment in reference to medical practice. Educated in a time when the germ theory was unknown he never adapted himself to new techniques of sterilization and sanitation.

Dr. Stratton practiced under most primitive conditions. The doctor's offices had none of the conveniences essential to comfort. A wood burning stove furnished heat, water was carried in, illumination was furnished by coal oil lamps; and unless the doctor was lucky enough to be able to employ help the doctor did what janitor work was required. Like other pioneers he got around the country as best he could; by horseback, with horse and buggy, sleigh or cutter. For many years Dr. Stratton and Dr. Flynn of Redwood Falls were the best known, if not the only doctors in a very large territory, and they were on the road day and night. At one time Dr. Stratton owned seven horses and it is remembered that Mr. Charlie Rice drove for him. A driver was a necessity not only to share the labor of getting the buggy through mudholes or snow banks but to permit the doctor to rest between calls.

When the railroad came to Granite Falls in 1878 Dr. Stratton was appointed the first surgeon and physician of the road at that point, and he held this post until his death, so that he was then the oldest physician of the line in point of service. Soon he began to travel by train to the more distant points to which he was called; the *Granite Falls Journal* mentions that "Dr. Stratton is on wheels most of the time," attending to his practice, and meeting in consultation with other physicians.

The Stratton home was a favorite meeting place for the children of the neighborhood. Dr. and Mrs. Stratton had a family of their own: three sons, Frank E., William, and Paul D., and three daughters, Adeline E., Edna, and Susie. Susie loved to help her father; she is remembered as a little girl with braids down her back and glasses on her nose, using the vaccinating needle like a veteran upon the children who were candidates for this rite. But it was Edna, not Susie, who became a nurse.

The Strattons had their sorrows; the little girl, Adeline, died in 1894; the doctor's health suffered and he was away for five months for treatment and rest. Younger doctors with more modern methods and techniques settled in Granite Falls and other parts of the county which he formerly served, cutting into his practice; and in his later years the doctor had less strength for and interest in the arduous duties of the country practitioner. Throughout his career he was respected for his exceptional skill and medical knowledge, for he often succeeded where other physicians failed. His personality endeared him to all who knew him; he brought to his patients the assurance of his sincere interest and concern.

When plans were made for the erection of a tubercular sanatorium at Granite Falls for the tubercular patients of three countties, Dr. Stratton was chosen as a member of the board from Yellow Medicine County, and this position he held during the time the buildings were being erected and the first patients received at the new sanatorium. He was also a member of the Camp Release Medical Society and of the State Medical association.

Mrs. Stratton died January 11, 1912, after a rather long period of declining health. Dr. Stratton died November 10, 1916, of a stroke of apoplexy, at the age of seventy-two years. He was survived by his sons, Frank E., William, and Paul D., his daughters, Edna Maercklein, and Susie (Mrs. M. H.) Geehan. Edna Maercklein was the wife of a doctor and assisted in the operation of a hospital at Oakes, North Dakota, at one time.

Present survivors of the Stratton family are Paul D. Stratton, an attorney who has practiced at Granite Falls for about forty years; Susie, who was Mrs. M. H. Geehan of Graceville, Minnesota, was survived by a son, Robert Geehan, who is a mining engineer, and a daughter Adeline who is now employed by the city of Minneapolis.

Albert R. Torgersen

"Dr. A. Torgersen of Fargo, North Dakota, is stopping at E. P. LeSeuer's Drug Store. He has had extensive experience and will gladly attend any demands for his professional services."

This announcement in the issue of the *Canby News* of October 9, 1885, heralded the arrival of Dr. Albert R. Torgersen, who was to spend many years in the practice of medicine in Yellow Medicine County.

Albert R. Torgersen was born in Oslo, Norway, and received his education and medical training in the schools and Universities there. He came to the United States as a young man and settled first in Wisconsin. From Wisconsin he came to Minnesota, locating in Alexandria, where he met the young woman who became his wife, Miss Addie Corneliuson, who was also of Norwegian descent. From Alexandria he went to Fargo, North Dakota, leaving that city for Canby in 1885.

When Dr. Torgersen came to Canby he had two children, Rudolph Charles, who was born in Alexandria, and William Cornelius, who was born at Fargo, North Dakota. In appearance the doctor was small in stature, dark, always well groomed and giving an impression of poise and good breeding. He wore a large mustache, and in disposition was kindly, but very outspoken and so strong in his convictions that he was sometimes regarded as "contrary."

Dr. Torgersen seemed to have trouble establishing himself in Canby. A rumor to the effect that he was not qualified to practice in the state caused at least one patient to refuse to pay for his services, and Dr. Torgersen promptly sued for his fees. Thereafter he announced through the *Canby News* that he had been educated in Norway and had complied with the requirements of state law and was licensed to practice in Minnesota.

Dr. Torgersen was also a graduate pharmacist, and after three years in Clarkfield, during which time another son, Roy Jay, had been born, the doctor decided to go into the drug business in connection with his practice. In 1888 he purchased the stock of the Clarkfield Drug Store and on September 20, the *Canby News* announced that "Dr. Torgersen has taken possession of the drug store he recently purchased at Clarkfield. He will probably do well in his new venture and the outlook is flattering." As to what Clarkfield was like at this time, there is this comment:

> "Clarkfield is a new town started upon Monson's place two years ago and now numbers 300 inhabitants. It is a great wheat market for a town of its size and has taken in as high as 5,000 bushels of wheat a day. Yellow Medicine must be a wheat growing county, one 20 acre piece yielding 900 bushels of Blue Stem wheat."

The little village was in the heart of a rich farming country and its population was predominantly Norwegian. Dr. Torgersen was the first doctor to locate in Clarkfield.

The doctor's first offices in Clarkfield were in the old Myhre Building, and his family lived in the rooms adjoining them. Shortly after the arrival in Clarkfield another son, Cleve Reuben, was added to the family circle. The doctor was a Democrat and a great admirer of Grover Cleveland, and Cleve Reuben was named for him; but during Cleveland's administration the doctor suffered heavy financial losses, as did many other business and professional men, in the panic of 1893.

In 1888 Dr. Torgersen was elected county coroner; and he was very conscientious in exercising the duties of this office, as shown by the case of Peter Strand. In this case the dead man was found in a meadow where the body had evidently lain for some time undiscovered, despite the fact that one Luders had been mowing in full view of the body for some days. A quantity of blood, or evidences of blood, was found, and there were wounds in the throat. But the man was buried in spite of protests, as he was an immigrant and without friends, but believed to have had money as he contemplated buying a farm. Evidently public opinion compelled the authorities to take further steps, so the body was exhumed and an examination made. The *Canby News* reported that the doctor felt he had "been hindered in his examination," and believed that murder had been done, but the authorities refused to take action. The county attorney was A. J. Volstead.

Dr. Torgersen also served as County Physician for the Clarkfield medical district.

The fifth child born to the Torgersens was Pearl Celia, and when the little girl was still very young her mother died very suddenly.

After this bereavement the doctor was left with the responsibility of caring for the children in addition to his medical practice and drug business. Nevertheless he managed; the family lived in a home in the west part of Clarkfield where the doctor had an opportunity to indulge his hobby of gardening. During the summer his vegetable garden and gorgeous flower beds made his home a show place. He also found time for crayon work and drawing which showed his considerable talent as an artist.

The rewards of medical practice in this small community were small. The usual charge for a confinement case was $5.00; a country call was made for $2.00, and a dollar had to be paid out for a livery rig. Then often the farmer would tell some story of hard luck, and Dr. Torgersen would tell him just to forget about the fee. People respected Dr. Torgersen's ability as a doctor, and although new and younger doctors came to the community there were many people who thought no one could do quite as much for them as Dr. Torgersen.

The doctor's son Rudolph Charles answered the call for volunteers in the Spanish American War, and shortly after his discharge from service died in Seattle, Washington.

Another boy to go into the military service was Cleve Reuben, who served in World War I, and was killed in action.

In 1917 Dr. Torgersen gave up the practice of medicine in Clarkfield and went with his son Roy Jay to Hawarden, Saskatchewan, Canada, and lived there until his death.

He was survived by his son William Cornelius, who is now in business in Starbuck, Minnesota; Roy Jay who is in business in Winnipeg, Canada, and Pearl Celia (Mrs. A. G. Drysdale) who resides in Arcadia, California.

William B. Torgerson was born in Worth County, Iowa, on October 27, 1872. He was the son of Rev. Torger A. Torgerson and Dina Anderson Torgerson, and was christened Wilhelm B. Torgerson. He received his early education in the schools of Iowa, and then entered the College of Homeopathic Medicine and Surgery of the University of Minnesota. He graduated in 1897, and began his practice in Cannon Falls, Minnesota, the same year.

In November, 1908, Dr. Torgerson located in Clarkfield. The *Cannon Falls Beacon* in commenting on Dr. Torgerson's departure stated:

> "Dr. Torgerson's departure will be greatly regretted. He is a thorough gentleman and a skilled physician who has gained many friends during his stay in Cannon Falls."

In Clarkfield Dr. Torgerson occupied offices over the Clarkfield State Bank, with living quarters in connection so that he could be conveniently "found day and night" as stated in the *Clarkfield Advocate.* He was a tall, slender, dignified young man, with brown hair and eyes, a small well trimmed goatee, and is generally described as "aristocratic looking."

In 1899 Dr. Torgerson was appointed Health Officer for the village of Clarkfield.

In June, 1900, Dr. Torgerson took a brief vacation, during which, on June 14, he was married to Miss Ingeborg Johanna Ohnstad of Watsedo, Minnesota; ("the bride is one of Goodhue's fairest and most accomplished daughters") and the young couple returned from a wedding trip to take up housekeeping in the apartment formerly used as offices and living quarters, while the doctor settled in new offices over the bank. According to the *Advocate,* Dr. Torgerson "came here about two years ago, and by strict attention to his profession has built up a good practice and enjoyed a large degree of popularity in social circles."

Throughout his life Dr. Torgerson was known as William B. Torgerson rather than by his baptismal name of Wilhelm.

The first child born to the Torgersons was a girl, born May 14, 1901, and named Dora. A son, Erling A., was born October 28, 1902, Harold W., born March 27, 1905, and Theodore A., born May 22, 1907.

During the period of residence in Clarkfield the doctor and his wife were very well liked, and he enjoyed a good medical practice. He also occupied for a time the position of postmaster of Clarkfield.

In 1907 Dr. Torgerson removed to Greenbush, Minnesota, and practiced there until 1912. Another son, William B., Jr., was born there on February 12, 1909. In 1912 Dr. Torgerson moved to Oklee, Minnesota, and practiced there until his death.

Dr. Torgerson took an active part in public affairs in Oklee, and had many interests in addition to medicine. He went into banking, became a member of the school board, the secretary of the creamery at Oklee, and was active in church matters.

At the time of his death Dr. Torgerson was one of the oldest medical practitioners in the state. He was survived by his wife and five children. Mrs. Torgerson continues to live at Oklee, Minnesota; daughter, Dora, is Mrs. Robert E. Bilbrough of Overland Park, Kansas; Erling A. Torgerson lives at Thief River Falls where he is in business; Harold W. Torgerson lives in Evanston, Illinois, and Theodore is at Annapolis, Maryland, while William B. Torgerson resides in Oakland, California.

Dr. Torgerson was a member of the Evangelical Lutheran Church. A brother, Rev. Abel Anderson, lived at Montevideo and the Torgerson children were baptized by him and all became members of the "Synod" group of the Lutheran church.

Dr. Torgerson died July 7, 1952, of Parkinson's disease, for which ailment he was hospitalized for three years previous to his death.

Henry G. Townsend was born in Maine in 1823. He came west and it is believed that he lived for a time in Chippewa County, Wisconsin, for that was the place of residence of his divorced wife, Alice, and his son, Edgar H. Townsend.

Dr. Townsend came to Granite Falls in 1877, at the age of fifty-four years. He established himself in a house in East Granite Falls, Chippewa County, and inserted his professional card in the *Granite Falls Journal.* The issue of that paper on July 25, 1877, introduced him as a

> "Homeopathic Physician, Surgeon and Obstetrician. Special attention given to cronice (sic) diseases and diseases of women and children. Office and residence on east side opposite E. J. Fitch's Hotel."

This places him on the north side of what is now Baldwin Avenue near the Milwaukee depot.

Dr. Townsend was a quiet, elderly man; he was a member of the first library association of Granite Falls.

In 1882 Dr. Townsend had moved to Canby, where he had an office "1 door south of P. O." His professional card stated that "He will hold himself in readiness to attend calls at all hours night and day." Dr. Townsend is also said to have practiced in the small settlements near Canby more extensively than in the city of Canby itself.

On January 26, 1883, he was stricken with a heart attack and died at the age of sixty years. Probate proceedings of the county show that his only heir was his son, Edgar H. Townsend, sixteen years old, a resident of Chippewa County, Wisconsin.

M. N. Triplett was graduated from the University of Louisville in 1895, and was licensed in Minnesota January 11, 1898.

He was born in one of the southern states, either Tennessee or Kentucky, and educated in the schools of the South. He always kept his southern manner of speech, which was quite distinctive. He was a fine looking young man, of medium height and coloring, and good physique.

Dr. Triplett located in Hanley Falls shortly after his graduation and practiced there two years. At the end of that time he went to Dawson, where he built up a good practice and was regarded as a very good doctor. When he came to Dawson he had been married and was a widower; in Dawson he married a local girl, and they occupied living quarters adjoining his offices which were over Charles Anderson's store.

The doctor was friendly, well-mannered, and very well liked. He was extremely fond of hunting, and spent much time in the field during the season when it was permissible to hunt prairie chickens. He was a member of the Masonic Lodge.

About 1908 Dr. Triplett removed to Floodwood, where he bought some land, practiced medicine, and engaged in other activities. He was a member of and president of the Board of Education of Floodwood, and at one time was a member of the State Legislature. He had no children. He died December 8, 1922, of myocarditis.

James Alfred Watson was born September 5, 1867, in Longford, Ireland. He went to Canada with his father when he was seventeen years of age, and later his mother and three brothers followed. The family settled on a farm at Pilot Mound, Manitoba, and at first James Alfred Watson taught school and thus financed his medical education. He took his medical training at the University of Manitoba, in Winnipeg, graduating in 1894. He began his practice of medicine in Wood Lake, Minnesota, and stayed there four years. He left in 1898 to take up postgraduate work in eye, ear, nose and throat diseases in New York and Vienna between the years from 1899 to 1900. Returning to Minnesota in 1900 he married Lucia Coghlan of Wood Lake and began to practice as an ophthalmologist and otolaryngologist in Minneapolis. He practiced there until his death on June 17, 1939.

During his early years of practice in Minneapolis he was a member of the faculty of Hamline University Medical School.

Beginning with 1920, he practiced as a member of the Eye, Ear, Nose and Throat Clinic, and was associated with Doctors Eugene Strout, John Macnie, and W. E. Patterson. This clinic was located in their own building at 74 South Eleventh Street until the year 1934, and then in the Medical Arts Building.

Dr. Watson became a citizen of the United States soon after starting practice in Minneapolis. He was an active member of the Hennepin Avenue Methodist Episcopal Church. He was also a member of the Hennepin County Medical Society, Minnesota State Medical Association, American College of Surgeons, American and Minnesota Academy of Ophthalmology and Otolaryngology; and a member of the Minneapolis Athletic Club and the Lafayette Club.

Dr. Watson was survived by his widow and four children: Cecil James, Lelan Alfred, Eleanor Isabel, James Alfred, Jr., all of Minneapolis. He was known as a "sound and conservative practitioner of medicine and a true and kindly friend."

Dr. Watson was seventy-two years of age at the time of his death, and lived at 2100 Irving Avenue, Minneapolis. He was past president of the Minnesota Academy of Ophthalmology and Otolaryngology, a member of the Phi Rho Sigma Medical Fraternity and the Automobile Club.

Cause of death was coronary thrombosis; contributory causes, coronary sclerosis, hypertension (three years), apoplexy (one year). Burial was in Lakewood Cemetery.

Frederick G. Watson was born in Longford, Ireland. His father and his brother, James A. Watson, came to Canada in 1884, and shortly thereafter Frederick G. Watson, his mother, and two brothers followed. The family settled on a farm at Pilot Mound, Manitoba, and Frederick G. Watson received his elementary education in the schools of that province. For his medical training he entered Hamline University, graduating about 1895. He also graduated as a pharmacist, and about 1896 went to Wood Lake where he opened a drug business. On September 1, 1897, Dr. Watson married Isabella A. Tibbils of Wood Lake, Minnesota, and shortly thereafter moved to the neighboring town of Clarkfield and began his medical practice there.

Children born to Dr. and Mrs. Watson in the years that followed were: Horace Percy, born October 2, 1898; Leopold Frederick, born November 30, 1902; Sidney J., born August 10, 1904; Donald Goodwin, born October 13, 1907, and Anna Bernice, born August 12, 1909.

In appearance Dr. Watson was very English: tall, rather slender, very reserved and inclined to shyness. His speech was the clipped speech of the Englishman, a characteristic which set him off in a strongly Norwegian community. He had a very brilliant mind and was regarded as a very good doctor. He belonged to the Methodist church and his religious beliefs precluded membership in other organizations.

Clarkfield was a small village of about 320 when Dr. Watson began practice there, and had two Lutheran Churches and a Methodist Church. The language most commonly heard upon the streets at that time was Norwegian. About the same time Dr. Charles Watson, a brother, was practicing at Boyd, Minnesota, and James A. Watson at Wood Lake.

Dr. Watson built up a large practice in the community. He drove long distances to visit patients in the surrounding country, getting about with horse and buggy, or cutter, in all kinds of weather and over roads which were no more than tracks on the prairie sod, or at the best narrow highways which became almost impassible in wet weather. In 1906 his practice had grown so large that he felt it necessary to look for a partner, and the man he thought most suitable was Dr. M. M. Hauge. In 1907 Dr. Hauge came to Clarkfield as Dr. Watson's partner, and the two practiced together for some years. As Dr. Hauge spoke Norwegian fluently, was Norwegian, strongly Lutheran, and with gifts in music and other attributes which made him very popular in this predominantly Norwegian community, it was not long until Dr. Watson found his Norwegian patients preferring to consult Dr. Hauge. When Dr. Watson became convinced that there was no future for him in Clarkfield he left and located in Rushmore, Minnesota, where he stayed ten years. He then went to Worthington and established there a very good practice and a clinic.

Dr. Watson became ill with a heart ailment about 1930, and died January 11, 1932, of a heart attack, at Worthington, Minnesota.

Mrs. Watson survived him, and also his children Horace Percy, Leopold Frederick, Donald Goodwin and Anna Bernice. His son Sidney J. Watson became a doctor, graduating from the University of Minnesota Medical Department, and died prior to his father's death. His daughter Anna Bernice died July 21, 1932. Mrs. Watson died June 11, 1945, at Worthington, Minnesota.

Horace Percy Watson now resides at Sioux Falls, S. D., a partial invalid. Donald Goodwin operates a farm near Redfield, S. D.; Leopold Frederick is a dentist.

(To Be Concluded)

MINNESOTA STATE BOARD OF MEDICAL EXAMINERS

230 Lowry Medical Arts Bldg., Saint Paul, Minnesota

E. M. Jones, M.D., Secretary

PHYSICIANS LICENSED FEBRUARY 13, 1953

January, 1953, Examination

Name	*School*			*Address*
ALARI, Heino	Eberhard-Karls U. Tubingen, Germany	MD	1947	Northwestern Hosp., Minneapolis, Minn.
ANDERSON, Hugh Vaux	U. of Oregon	MD	1952	3425 S.W. 9th, Portland, Ore.
BEDDOW, Ralph Marlowe	U. of Oregon	MD	1950	102-110 2nd Ave. S.W., Rochester, Minn.
CRISP, Jr., Elmer Ernest	Northwestern U.	MD	1950	102-110 2nd Ave. S.W., Rochester, Minn.
DAVIS, Charles Truman	U. of Tennessee	MD	1950	102-110 2nd Ave. S.W., Rochester, Minn.
DENKER, Arthur Gilman	U. of Oregon	MD	1952	63 E. Ash, Lebanon, Ore.
DUNN, William Joseph	U. of Illinois	MD	1948	102-110 2nd Ave. S.W., Rochester, Minn.
ELSTNER, Howard L.	U. of Pittsburgh	MD	1951	102-110 2nd Ave. S.W., Rochester, Minn.
ERICKSON, Robert Dell	U. of Minnesota	MD	1953	Wykoff, Minn
FISHER, Don Harvey	Northwestern U.	MD	1951	102-110 2nd Ave. S.W., Rochester, Minn.
GALLO, Bela	Peter Pazmany U. Budapest, Hungary	MD	1945	Lake Bronson, Minn
GOSSELIN, Gilles	U. of Montreal	MD	1949	102-110 2nd Ave. S.W., Rochester, Minn.
HARDY, Paul Edward	St. Louis U.	MD	1951	Ancker Hosp., St. Paul, Minn.
HEALY, John Russell	Jefferson Med. Col.	MD	1949	102-110 2nd Ave. S.W., Rochester, Minn.
JACKS, Quentin Douglas	U. of Mantitoba	MD	1940	102-110 2nd Ave. S.W., Rochester, Minn.
JARVIS, Bruce William	U. of Minnesota	MD	1953	1869 Marshall Ave., St. Paul, Minn.
LEPPO, Niilo Erkki Albert	U. of Helsinki Helsinki, Finland Candidate of Medicine Licentiate of Medicine		 1931 1936	503 Med. Arts Bldg., Duluth, Minn.
MacMILLAN, Angus Hugh	Queens U.	MD	1950	102-110 2nd Ave. S.W., Rochester, Minn.
McPHERSON, Alan Given	U. of Manitoba	MD	1951	102-110 2nd Ave. S.W., Rochester, Minn.
MANSTEIN, George	U. of Pennsylvania	MD	1941	102-110 2nd Ave. S.W., Rochester, Minn.
MYERS, Richard Lyon	St. Louis U.	MD	1951	102-110 2nd Ave. S.W., Rochester, Minn.
OLAVS, Olga	U. of Latvia Riga, Latvia	"Physician"	1942	1009 Nicollet Ave., Minneapolis, Minn.
PRICE, Mary Lomas	U. of Aberdeen Aberdeen, Scotland	MB SB	 1947	102-110 2nd Ave. S.W., Rochester, Minn.
SATERSMOEN, Jr., Theodore	U. of Minnesota	MD	1948	Veterans Adm. Hosp., Dearborn, Mich.
SIBLEY, John Adams	U. of Chicago	MD	1946	102-110 2nd Ave. S.W., Rochester, Minn.
SKILLERN, Scott Dorsey	U. of Pennsylvania	MD	1951	102-110 2nd Ave. S.W., Rochester, Minn.
STREITZ, John McDonald	U. of Minnesota	MD	1952	Veterans Adm. Hosp., Minneapolis, Minn.
VAN PROOYEN, Cornelia Mary	Washington U., Mo.	MD	1951	Mpls. Gen. Hosp., Minneapolis, Minn.

Reciprocity Candidates

Name	*School*			*Address*
BEST, Etta Wright	U. of Louisville	MD	1949	102-110 2nd Ave. S.W., Rochester, Minn.
CARROLL, John Joseph	Creighton U.	MD	1947	117½ S. 1st St., Montevideo, Minn.
COOK, Clayton Stevens	Tulane U.	MD	1949	102-110 2nd Ave. S.W., Rochester, Minn.
FELDAKER, Mauri	Washington U., Mo.	MD	1950	102-110 2nd Ave. S.W., Rochester, Minn.
GOFF, John Robert	U. of Rochester	MD	1951	102-110 2nd Ave. S.W., Rochester, Minn.
GUNN-SMITH, Allan Murray	U. of Minnesota	MD	1950	431 10th Ave., Two Harbors, Minn.
HEIST, Erwin William	U. of Iowa	MD	1951	Jaguum Hosp., Lutheran Mission, Madang, Terr., New Guinea
HUGHES, Sidney Osborne	U. of Pennsylvania	MD	1947	172 Main St., Winona, Minn.
HYMAN, Jr., Orren Williams	U. of Tennessee	MD	1951	102-110 2nd Ave. S.W., Rochester, Minn.
JESSE, Richard Henry	U. of Nebraska	MD	1949	Truman, Minn.
KEYS, John R.	U. of Cincinnati	MD	1950	102-110 2nd Ave. S.W., Rochester, Minn.
KUNTZ, Aloysius Leo	Tulane U.	MD	1951	102-110 2nd Ave. S.W., Rochester, Minn.
LEMMON, Mark Leonard	U. of Texas	MD	1948	102-110 2nd Ave. S.W., Rochester, Minn.
O'LEARY, James Michael	Jefferson Med. Col.	MD	1946	102-110 2nd Ave. S.W., Rochester, Minn.
OPFELL, Richard William	U. of Iowa	MD	1949	102-110 2nd Ave. S.W., Rochester, Minn.
PERRY, Jr., John Francis	U. of Texas	MD	1947	U. of Minn. Hosp., Minneapolis, Minn.
SANDERS, Edward Joseph	Creighton U.	MD	1949	102-110 2nd Ave. S.W., Rochester, Minn.
SLEDGE, Claire Blount	U. of Oklahoma	MD	1948	102-110 2nd Ave. S.W., Rochester, Minn.
SPITTEL, Jr., John Allen	U. of Maryland	MD	1949	102-110 2nd Ave. S.W., Rochester, Minn.

Name	*School*		*Address*
TOON, Robert Wallace	U. of Oregon	MD 1944	1251 Med. Arts Bldg., Minneapolis, Minn.
TROUP, Richard Henry	Northwestern U.	MD 1950	102-110 2nd Ave. S.W., Rochester, Minn.

National Board Candidates

Name	*School*		*Address*
ALLEN, George Seaver	U. of Rochester	MD 1950	Mineral Spgs. San., Cannon Falls, Minn.
BAGLEY, Russell Willis	Tufts Med. College	MD 1948	871 S. Wilder St., St. Paul, Minn.
BEEMAN, Edward Arthur	Boston U.	MD 1947	102-110 2nd Ave. S.W., Rochester, Minn.
BENJAMIN, Robert Bedell	U. of Minnesota	MD 1952	18th & Taylor, Hood River, Ore.
BOUCHELLE, McLemore	Harvard U.	MD 1943	6th Ave. & 9th St. N., Virginia, Minn.
BUCKLEY, Joseph John	New York Med. Col.	MD 1946	500 Union St., Minneapolis, Minn.
CHAPIN, Lemuel Edward	Temple U.	MD 1951	102-110 2nd Ave. S.W., Rochester, Minn.
GORDON, Donald Lloyd	New York Med. Col.	MD 1947	102-110 2nd Ave. S.W., Rochester, Minn.
LIPINSKI, Stanley Wnuk	Northwestern U.	MD 1951	Hibbing, Minn.
SILVER, Lawrence	New York Univ.	MD 1950	Hosp. of Rockefeller Inst. for Med. Research, 66th St. & N. Y. Ave., New York 21, N. Y.
SVIGALS, Robert Elliott	New York Med. Col.	MD 1951	102-110 2nd Ave. S.W., Rochester, Minn.
VAN BUSKIRK, Charles	Albany Med. Col.	MD 1947	U. of Minn. Hosp., Dept. Neurology, Minneapolis, Minn.
WEBB, Jr., Wilson Davenport	New York Med. Col.	MD 1947	102-110 2nd Ave. S.W., Rochester, Minn.
YOUNG, John Van Winkle	Northwestern U.	MD 1951	102-110 2nd Ave. S.W., Rochester, Minn.

PHYSICIANS LICENSED MAY 22, 1953

April, 1953, Examination

Name	*School*		*Address*
AMBRUS, Laszlo	Eberhard-Karls U. Tubingen, Germany	MD 1948	462 S. Robert St., St. Paul, Minn.
AMBRUS, Milka	Eberhard-Karls U. Tubingen, Germany	MD 1948	Midway Hosp., St. Paul, Minn.
ANDERSON, Jack G.	Marquette U.	MD 1951	Mpls. Gen. Hosp., Minneapolis, Minn.
BALEISIS, Peter	U. of Dusseldorf	MD 1949	700 W. Broadway, Minneapolis, Minn.
BARLOW, Loren Call	Northwestern U.	MD 1951	102-110 2nd Ave. S.W., Rochester, Minn.
BOZANIC, Milos S.	Ludwig Maximilian U. Munich, Germany "Arzt"	1948	St. Joseph's Hosp., St. Paul, Minn.
CONNAUGHTON, Patrick Alex	St. Louis U., Mo.	MD 1951	Mpls. Gen. Hosp., Minneapolis, Minn.
DATESMAN, Robert Wallace	U. of Pennsylvania	MD 1951	USAH, 3444 ASU, Camp Stewart, Ga.
DE WEESE, Jr., Robert Cornell	U. of Colorado	MD 1952	St. Cloud Nat. Bk. Bldg., St. Cloud, Minn.
FEINBERG, Milton	U. of Illinois	MD 1941	2215 Plymouth Ave. N., Mpls., Minn.
FOLEY, III, Philip James	Northwestern U.	MD 1951	102-110 2nd Ave. S.W., Rochester, Minn.
GLENNY, Jr., William Robb	U. of Minnesota	MD 1953	Box 308, Osseo, Minn.
GREENWOLD, Warren Eldon	U of Chicago	MD 1946	102-110 2nd Ave. S.W., Rochester, Minn.
HEPBURN, Allan Lockwood	U. of Alberta	MD 1948	102-110 2nd Ave. S.W., Rochester, Minn.
JOHNSON, Marvin Waldemar	U. of Minnesota	MD 1953	Milaca, Minn.
KARLINSKY, William	U. of Manitoba	MD 1945	644 Niagara St., Winnipeg, Man., Can.
KARNOPP, Katherine	U. of Oregon	MD 1952	Glen Lake San., Oak Terrace, Minn.
KONIG, Theodore John	U. of Minnesota	MD 1952	Veterans Adm. Hosp., Minneapolis, Minn.
LASZEWSKI, Franz von Zelberschwecht	Westphalen Wilhelms U. Munster, Germany	MD 1929	St. Joseph's Hospital, St. Paul, Minn.
LYSYJ, Anatol	Ludwig Maximilian U. Munich, Germany	MD 1950	624 W. 54th St. S., Minneapolis, Minn.
McCULLOCH, Charles Fraser	Queens Univ.	MD 1945	102-110 2nd Ave. S.W., Rochester, Minn.
MORISSETTE, Gaston	Laval Univ.	MD 1950	102-110 2nd Ave. S.W., Rochester, Minn.
PLACE, Virgil Alan	Johns Hopkins U.	MD 1948	102-110 2nd Ave. S.W., Rochester, Minn.
PROSHEK, Lumir Charles	U. of Iowa	MD 1949	Mpls. Gen. Hosp., Minneapolis, Minn.
RICHARDS, Albert MacDonell	U. of Minnesota	MD 1948	Heart Hosp., U. of Minn., Mpls., Minn.
RYZEN, Nicholas	U. of Tennessee	MD 1951	Anoka State Hosp., Anoka, Minn.
SABANAS, Alvina Olga	U. Vytautas the Great Kaunas, Lithuania	MD 1939	102-110 2nd Ave. S.W., Rochester, Minn.
SMITH, Dale Compton	U. of Kansas	MD 1945	102-110 2nd Ave. S.W., Rochester, Minn.
SQUIRES, Franklin John	U. of Manitoba	MD 1945	102-110 2nd Ave. S.W., Rochester, Minn.
THOMPSON, Richard Eugene	Northwestern U.	MD 1951	NPBA Hosp., Glendive, Mont.
TORKELSON, Leonard Beck	U. of Minnesota	MD 1953	St. Joseph's Hosp., St. Paul, Minn.
TSAI, Shih Hao	National Med. Col. Shanghai, China	MD 1941	Glen Lake San., Oak Terrace, Minn.
VOIGHT, Allan Earl	U. of Oregon	MD 1951	Mpls. Gen. Hospital, Minneapolis, Minn.
VREELAND, Oliver Henry	U. of Oregon	MD 1952	102-110 2nd Ave. S.W., Rochester, Minn.
WESTPHAL, Paul Harry	U. of Minnesota	MD 1953	Gettysburg, S. D.
WOYDA, William Charles	U. of Minnesota	MD 1953	Box 609, Elk River, Minn.

Reciprocity Candidates

Name	*School*		*Address*
BOSSARD, John Wesley	U. of Maryland	MD 1951	102-110 2nd Ave. S.W., Rochester, Minn.
BOYD, George Knox	U. of Pittsburgh	MD 1951	102-110 2nd Ave. S.W., Rochester, Minn.
BURGERMAN, Arthur	Indiana U.	MD 1948	102-110 2nd Ave. S.W., Rochester, Minn.
BUTLER, Jr., Lewell Colbert	Louisiana State U.	MD 1946	102-110 2nd Ave. S.W., Rochester, Minn.
CAYCE, John Hill	U. of Texas	MD 1947	102-110 2nd Ave. S.W., Rochester, Minn.
CLINGER, Edwin James	Ohio State U.	MD 1946	102-110 2nd Ave. S.W., Rochester. Minn.
COOPER, Milton	Cornell U.	MD 1948	102-110 2nd Ave. S.W., Rochester, Minn.
DURAN, Robert Jackson	U. of Oklahoma	MD 1947	102-110 2nd Ave. S.W., Rochester, Minn.
FYKE, Jr., Frazier Earl	U. of Pennsylvania	MD 1947	102-110 2nd Ave. S.W., Rochester, Minn.
HUEBERT, Dan W.	U. of Kansas	MD 1946	26 Washington, Hutchinson, Minn.
JACKSON, Robert Carl	U. of Wisconsin	MD 1945	102-110 2nd Ave. S.W., Rochester, Minn.
JOHNSON, Robert Hugo	U. of Nebraska	MD 1952	210 3rd St. S.W., Little Falls, Minn.
KILMER, Warren Lee	Indiana U.	MD 1952	State Bk. Bldg., Inter. Falls, Minn.
LAW, William McConnell	Med. Col. of Va.	MD 1948	102-110 2nd Ave. S.W., Rochester, Minn.
LEE, Philip Randolph	Stanford U.	MD 1948	102-110 2nd Ave. S.W., Rochester, Minn.
LOCHRIDGE, Jr., Edwin Payne	U. of Georgia	MD 1949	102-110 2nd Ave. S.W., Rochester, Minn.
LOWE, James Cecil	U. of Tennessee	MD 1943	102-110 2nd Ave. S.W., Rochester, Minn.
MacLEAN, Donald Beacom	Wayne Univ.	MD 1950	102-110 2nd Ave. S.W., Rochester, Minn.
PRICE, Jr., William Edmund	U. of Oklahoma	MD 1946	412 Delaware St. S.E., Minneapolis, Minn.
PURO, Henry Evert	Stanford U.	MD 1949	102-110 2nd Ave. S.W., Rochester, Minn.
QUINONES, Rafael Enrique	U. of Tennessee	MD 1949	102-110 2nd Ave. S.W., Rochester, Minn.
REDELFS, John Wright	U. of Nebraska	MD 1945	102-110 2nd Ave. S.W., Rochester, Minn.
RICKMAN, James H.	U. of Louisville	MD 1946	102-110 2nd Ave. S.W., Rochester, Minn.
RYAN, Robert Frank	Stanford U.	MD 1947	102-110 2nd Ave. S.W. Rochester, Minn.
SCHWARTZ, Carl Alfred	U. of Pennsylvania	MD 1947	102-110 2nd Ave. S.W., Rochester, Minn.
SCOTT, Jr., Charles	Emory Univ.	MD 1949	102-110 2nd Ave. S.W., Rochester, Minn.
SMITH, Jr. Fred Newman	Tulane Univ.	MD 1947	102-110 2nd Ave. S.W., Rochester, Minn.
TAYLOR, Harry Edwin	Temple Univ.	MD 1949	102-110 2nd Ave. S.W., Rochester, Minn.

National Board Candidates

Name	*School*		*Address*
EDELMANN, Robert Benedict	Long Island Col. Med.	MD 1947	102-110 2nd Ave. S.W., Rochester, Minn.
GARAMELLA, Joseph John	Georgetown U.	MD 1945	Veterans Adm. Hosp., Minneapolis, Minn.
HARTRIDGE, Virginia Bishop	Woman's Med. Col. of Pa.	MD 1950	102-110 2nd Ave. S.W., Rochester, Minn.
O'NEIL, Richard Lawson	Boston Univ.	MD 1947	102-110 2nd Ave. S.W., Rochester, Minn.
WALDMANN, Edward Bernard	Creighton Univ.	MD 1948	102-110 2nd Ave. S.W., Rochester, Minn.
WANNAMAKER, Lewis William	Duke Univ.	MD 1946	Dept. Ped., U. of Minn., Hosp., Minneapolis, Minn.
WINN, Jr., William Edward	Harvard Univ.	MD 1949	102-110 2nd Ave. S.W., Rochester, Minn.

PHYSICIANS LICENSED JULY 17, 1953

June, 1953, Examination

Name	*School*		*Address*
ANDERSON, Richard William	U. of Minnesota	MD 1953	St. Joseph's Hosp., St. Paul, Minn.
ARNESON, Genevieve A.	U. of Minnesota	MD 1953	U.S.P.H.S. Hosp., New Orleans, La.
BAKKE, Arnold Clifford	U. of Minnesota	MD 1953	St. Joseph's Hosp., St. Paul, Minn.
BASINGER, Harold Preston	U. of Minnesota	MD 1953	Williamsport Hosp., Williamsport, Pa.
BEARMAN, Morton	U. of Minnesota	MD 1953	Ancker Hosp., St. Paul, Minn.
BERG, Arnold Martin	U. of Minnesota	MD 1953	Mpls. Gen. Hosp., Minneapolis, Minn.
BOWLIN, Paul Frederick	U. of Minnesota	MD 1953	Oakland Naval Hosp., Oakland, Calif.
BRETZKE, Carl Otto	U. of Minnesota	MD 1953	Greenbush, Minn.
CAMMOCK, Earl Elarth	U. of Minnesota	MD 1953	Harborview Hosp., Seattle, Wash.
CARR, William Johnson	U. of Minnesota	MD 1953	338 LaSalle Bldg., Minneapolis, Minn.
COUNTRYMAN, Ethlyn	U. of Minnesota	MD 1953	San Francisco Hosp., San Francisco, Cal.
CROUCH, Boyden L.	U. of Kansas	MD 1946	102-110 2nd Ave. S.W., Rochester, Minn.
DEMARAIS, Francis Emery	U. of Minnesota	MD 1952	2508 Colfax Ave. S., Minneapolis, Minn.
DOBSON, Mervin Wilbert	U. of Manitoba	MD 1944	Medical Center, Mankato, Minn.
DOXSEE, George Charles	U. of Iowa	MD 1952	30 N.W. 1st St., Chisholm, Minn.
DUNN, Robert Clarence	U. of Minnesota	MD 1953	St. Joseph's Hosp., St. Paul, Minn.
FILIPOVICH, Orest Nicholas	U. of Minnesota	MD 1953	Mpls. Gen. Hosp., Minneapolis, Minn.
FRENCH, Bayard Taylor	U. of Iowa	MD 1952	2229 3rd Ave. E., Hibbing, Minn.
FUSARO, Ramon Michael	U. of Minnesota	MD 1953	Mpls. Gen. Hosp., Minneapolis, Minn.
GODFREY, H. Wilson	U. of Minnesota	MD 1953	Mpls. Gen. Hosp., Minneapolis, Minn.
GOODCHILD, William Russell	U. of Minnesota	MD 1953	Mt. Sinai Hosp., Minneapolis, Minn.
GRANQUIST, Richard David	U. of Minnesota	MD 1953	St. Luke's Hosp., Duluth, Minn.
HABERLAND, Lyle Freeman	U. of Minnesota	MD 1953	Sacramento Co. Hosp., Sacramento, Cal.
HAGEDORN, Jr., Max Boykin	Loyola U.	MD 1949	Veterans Adm. Hosp., Minneapolis, Minn.
HALVERSON, William Gearey	U. of Minnesota	MD 1953	St. Mary's Hosp., Duluth. Minn.

Name	*School*		*Address*
HANSON, Daniel James	U. of Minnesota	MD 1953	Mercy Hosp., Toledo, Ohio
HANSON, Harold Walden	U. of Minnesota	MD 1953	St. Joseph's Hosp., St. Paul, Minn.
HEID, James K.	U. of Minnesota	MD 1953	Madigan Army Gen. Hosp., Fort Lewis, Wash.
HEINZERLING, Carl Robert	U. of Minnesota	MD 1953	Mt. Sinai Hosp., Minneapolis, Minn.
HOPPERSTAD, Jule Jerome	U. of Minnesota	MD 1953	St. Mary's Hosp., Minneapolis, Minn.
HOYT, Charles Sherman	U. of Minnesota	MD 1953	Valley Forge Army Hosp., Phoenixville, Pa.
HUFF, John Stephen	U. of Minnesota	MD 1953	Memorial Hosp., Phoenix, Ariz.
JOHNSON, Gordon Erik	U. of Minnesota	MD 1953	St. Luke's Hosp., Duluth, Minn.
JUDE, James Roderick	U. of Minnesota	MD 1953	Johns Hopkins Hosp., Baltimore, Md.
KADESKY, Harold Baird	U. of Minnesota	MD 1953	2300 W. 50th St., Minneapolis, Minn.
KALLENBACH, Rudolf Werner	U. of Minnesota	MD 1953	703 Armstrong Blvd. S., St. James, Minn.
KANE, John Francis	Marquette U.	MD 1943	117½ S. 1st St., Montevideo, Minn.
KAPLAN, Martin Jack	U. of Minnesota	MD 1953	Michael Reese Hosp., Chicago, Ill.
KARON, Everett Howard	U. of Minnesota	MD 1953	U. of Minn. Hosp., Minneapolis, Minn.
KATKOV, Harold	U. of Minnesota	MD 1953	Mpls. Gen. Hosp., Minneapolis, Minn.
KENT, James Roy	U. of Minnesota	MD 1953	St. Luke's Hosp., Duluth, Minn.
KETOLA, Tauno Everett	U. of Minnesota	MD 1953	Highland Alameda Co. Hosp., Oakland, Cal.
KIRSCHBAUM, Thomas Harry	U. of Minnesota	MD 1953	U. of Minn. Hosp., Minneapolis, Minn.
LARSON, Earl Russell	U. of Minnesota	MD 1953	King Co. Hosp., Seattle, Wash.
LENTON, John David	Creighton U.	MD 1953	Creighton Memorial-St. Joseph's Hosp., Omaha, Neb.
LEUZINGER, Donn Ellis	U. of Minnesota	MD 1953	U.S.P.H.S. Hosp., Seattle, Wash.
LINDALL, Dale Regnar	U. of Minnesota	MD 1953	Tripler Army Hosp., Honolulu, Hawaii
LINNELL, Leonard S.	U. of Minnesota	MD 1953	St. Luke's Hosp., Duluth, Minn.
LITIN, Robert Beryl	U. of Minnesota	MD 1953	Santa Clara Co. Hosp., San Jose, Cal.
LITTLE, Bruce Raymond	U. of Minnesota	MD 1953	Santa Monica Hosp., Santa Monica, Cal.
MAGUIRE, Gerald Eugene	U. of Minnesota	MD 1953	Sacramento Co. Hosp., Sacramento, Cal.
MARK, Aaron Louis	U. of Minnesota	MD 1953	Mpls. Gen. Hosp., Minneapolis, Minn.
MARTIN, Jr., George Burnie	U. of Illinois	MD 1950	117½ S. 1st St., Montevideo, Minn.
McFARLANE, Donald Ray	U. of Minnesota	MD 1953	Mpls. Gen. Hosp., Minneapolis, Minn.
McMAHAN, Robert Oliver	U. of Minnesota	MD 1953	U. S. Naval Hosp., Oakland, Cal.
MEAGHER, Thomas Arthur	U. of Minnesota	MD 1953	King Co. Hosp. System, Seattle, Wash.
MELBY, James Christian	U. of Minnesota	MD 1953	U. of Minn. Hosp., Minneapolis, Minn.
MELLER, Maurycy	U. of Parma, Italy	MD 1937	First Nat. Bk. Bldg., Brainerd, Minn.
MILLER, Harold Dawes	U. of Minnesota	MD 1953	Mpls. Gen. Hosp., Minneapolis, Minn.
MURN, Thomas Gregory	U. of Minnesota	MD 1953	Mpls. Gen. Hosp., Minneapolis, Minn.
NACHTIGAL, Fred C.	U. of Minnesota	MD 1953	Veterans Adm. Hosp., Long Beach, Cal.
NAHAS, Gabriel	U. of Toulouse, France	MD 1944	315 Millard Hall, U. of Minn., Minneapolis, Minn.
NASH, Eldore Bentley	U. of Minnesota	MD 1953	Memorial Hosp., Phoenix, Ariz.
NEILSON, William George	U. of Illinois	MD 1946	102-110 2nd Ave. S.W., Rochester, Minn.
NORQUIST, Joseph Louis	U. of Minnesota	MD 1953	Walter Reed Army Hosp., Washington, D. C.
ODLAND, Donald Mark	Marquette U.	MD 1952	Mpls. Gen. Hosp., Minneapolis, Minn.
OLSON, Gregory Mathias	U. of Minnesota	MD 1953	Sacramento Co. Hosp., Sacramento, Cal.
PARSONS, Roger Alan	U. of Minnesota	MD 1953	St. Joseph's Hosp., St. Paul, Minn.
PERKINS, Douglass Everal	U. of Minnesota	MD 1953	614 Broadway, Alexandria, Minn.
POPADIUK, Peter	Friedrich Alex. U. Erlangen, Germany	MD 1948	St. Mary's Hosp., Minneapolis, Minn.
PRESTON, Frank Stebbins	U. of Minnesota	MD 1953	St. Vincent's Hosp., New York, N. Y.
PUGSLEY, Robert Daniel	U. of Minnesota	MD 1953	Swedish Hosp., Seattle, Wash.
RAMLOW, Ralph Marlyn	U. of Minnesota	MD 1953	Ancker Hosp., St. Paul, Minn.
RICHMAN, Harold Gene	U. of Minnesota	MD 1953	Barnes Hosp., St. Louis, Mo.
RICHTER, David James	U. of Illinois	MD 1952	6th Ave. & 9th St. N., Virginia, Minn.
ROCKWELL, Winthrop R.	U. of Minnesota	MD 1953	Tripler Army Hosp., Honolulu, Hawaii
RUDIE, William Donald	U. of Minnesota	MD 1953	St. Mary's Hosp., Duluth, Minn.
SCHAFER, Thomas Leo	U. of Minnesota	MD 1953	Detroit Receiving Hosp., Detroit, Mich.
SCHMIDT, Jr., Erwin Rudolph	U. of Wisconsin	MD 1952	St. Luke's Hosp., Duluth, Minn.
SCHNEIDER, James A.	U. of Minnesota	MD 1953	Gorgas Hosp., Panama, Canal Zone
SCHREINER, Leon H.	Northwestern U.	MD 1949	102-110 2nd Ave. S.W., Rochester, Minn.
SETZER, Jr., Hobert Joseph	U. of Minnesota	MD 1953	Mpls. Gen. Hosp., Minneapolis, Minn.
SHIELDS, Jack William	Stanford U.	MD 1948	102-110 2nd Ave. S.W., Rochester, Minn.
SISLER, David Allen	U. of Minnesota	MD 1953	Grand Rapids, Minn.
SONTAG, David W.	U. of Minnesota	MD 1953	Veterans Adm. Hosp., Long Beach, Cal.
SUNDBERG, Ruth Dorothy	U. of Minnesota	MD 1953	U. of Minn. Hosp., Minneapolis, Minn.
TAYLOR, Robert Owen	U. of Minnesota	MD 1953	San Diego Co. Gen. Hosp., San Diego, Cal.
TEYNOR, Joseph William	U. of Minnesota	MD 1953	U. S. Naval Hosp., Great Lakes, Ill.
THAL, Sam Harold	U. of Washington	MD 1951	102-110 2nd Ave. S.W., Rochester, Minn.
TIFFANY, Francis Buchanan	U. of Minnesota	MD 1953	St. Luke's Hosp., New York, N. Y.
ULRICH, Christian Andrew	Northwestern U.	MD 1950	102-110 2nd Ave. S.W., Rochester, Minn.
VERMUND, Halvor	U. of Oslo, Norway	MD 1943	U. of Minn. Hosp., Minneapolis, Minn.
VORIS, David Clarence	U. of Illinois	MD 1951	102-110 2nd Ave. S.W., Rochester, Minn.

WALDRON, John Fossen	U. of Minnesota	MD 1953	Mpls. Gen. Hosp., Minneapolis, Minn.
WALTER, William Edward	U. of Minnesota	MD 1953	Mpls. Gen. Hosp., Minneapolis, Minn.
WATERS, Alvin W.	U. of Minnesota	MD 1953	Ancker Hosp., St. Paul, Minn.
WATSON, William Richard	U. of Minnesota	MD 1953	Fitzsimons Army Hosp., Denver, Colo.
WEGNER, Marwood Emery	U. of Minnesota	MD 1953	U.S.P.H.S. Hosp., Staten Island, N. Y.
WENZEL, James Brandrup	U. of Minnesota	MD 1953	Memorial Hosp., Phoenix, Ariz.
WILLIAMS, Paul A.	U. of Minnesota	MD 1953	Tripler Army Hosp., Honolulu, Hawaii
WILLIAMS, Richard Albert	U. of Iowa	MD 1952	Rothsay, Minn.
ZELLMER, Richard Edward	U. of Minnesota	MD 1953	U.S.P.H.S. Hosp., Baltimore, Md.
ZEMMERS, Roberts	U. of Latvia Riga, Latvia	"Physician" 1926	St. Luke's Hospital, Duluth, Minn.

Reciprocity Candidates

Name	*School*		*Address*
BEITHON, Paul Jule	U. of Nebraska	MD 1952	Miller Hosp., St. Paul, Minn.
BURLEIGH, Jr., Edward Gilbert	Louisiana U.	MD 1952	More Hosp., Eveleth, Minn.
CALVERT, Wilson Clark	Northwestern U.	MD 1951	102-110 2nd Ave. S.W., Rochester, Minn.
COLLINS, Jr., Robert Aaron	U. of Georgia	MD 1948	102-110 2nd Ave. S.W., Rochester, Minn.
DYER, John Allen	U. of Pennsylvania	MD 1947	102-110 2nd Ave. S.W., Rochester, Minn.
JOHNSON, Morris Blaine	U. of Nebraska	MD 1952	Miller Hosp., St. Paul, Minn.
LEITSCHUH, Robert Bernard	Marquette U.	MD 1946	Sleepy Eye, Minn.
LEWIS, Jr., Royce Clay	Tulane U.	MD 1946	102-110 2nd Ave. S.W., Rochester, Minn.
McCALL, J. Preston	Baylor U.	MD 1948	102-110 2nd Ave. S.W., Rochester, Minn.
McKAY, Walter Edward	Indiana U.	MD 1948	102-110 2nd Ave. S.W., Rochester, Minn.
MOBLEY, Jack Ervin	Vanderbilt U.	MD 1948	102-110 2nd Ave. S.W., Rochester, Minn.
RACE, George Alexander	Temple U.	MD 1943	102-110 2nd Ave. S.W., Rochester, Minn.
SILAS, Ralph Maurice	U. of Michigan	MD 1943	Veterans Adm. Bldg., No. 18, Ft. Snelling, St. Paul, Minn.
SIMPSON, Joseph Ralph	Louisiana State U.	MD 1947	102-110 2nd Ave. S.W., Rochester, Minn.
SMITH, Jr., Ross Haugh	U. of Pittsburgh	MD 1951	102-110 2nd Ave. S.W., Rochester, Minn.
SOMMERNESS, M. Duane	Temple U.	MD 1945	Willmar State Hosp., Willmar, Minn.
THATCHER, Wilbur Clayton	U. of Iowa	MD 1933	Physicians Bldg., Fort Dodge, Ia.
WALLACE, III, Alexander	Baylor U.	MD 1948	102-110 2nd Ave. S.W., Rochester, Minn.
WILLER, Stanley Hermann	Ohio State U.	MD 1948	102-110 2nd Ave. S.W., Rochester, Minn.

National Board Candidates

Name	*School*		*Address*
ALEXANDER, Jr., John Dewey	U. of Pennsylvania	MD 1949	102-110 2nd Ave. S.W., Rochester, Minn.
ALTHAUSEN, Jr., Theodore L.	U. of California	MD 1947	65 S. 10th St., Minneapolis, Minn.
BRIDGE, Allyn Gustave	Yale U.	MD 1948	301 Kenwood Pkwy., Minneapolis, Minn.
DE WALL, Richard Allison	U. of Minnesota	MD 1953	1833 2nd Ave., Anoka, Minn.
FARLEY, Frank Giles	Syracuse U.	MD 1945	2229 3rd Ave. E., Hibbing, Minn.
GROCH, Sigmund Noel	Tufts Col. Med. Sch.	MD 1949	102-110 2nd Ave. S.W., Rochester, Minn.
LINDNER, Janus Casimir	Yale U.	MD 1950	505 Nicollet Ave., Minneapolis, Minn.
MARR, Jr., Norval Mason	Cornell U.	MD 1948	102-110 2nd Ave. S.W., Rochester, Minn.
NICHOLSON, Richard Waldo	Harvard U.	MD 1948	Heron Lake, Minn.
RUTLEDGE, John Boyden	U. of Minnesota	MD 1952	1050 Lake Ave., Detroit Lakes, Minn.
SKRDLA, Willard Blake	U. of Nebraska	MD 1948	102-110 2nd Ave. S.W., Rochester, Minn.

PHYSICIANS LICENSED NOVEMBER 13, 1953

October, 1953, Examination

Name	*School*		*Address*
ALLBAUGH, Robert Dean	Washington U., Mo.	MD 1952	Mpls. Gen. Hosp., Minneapolis, Minn.
AMICK, Lawrence Douglas	U. of Iowa	MD 1945	Sac City, Iowa
BONNET, John David	Johns Hopkins U.	MD 1952	102-110 2nd Ave. S.W., Rochester, Minn.
BOTHAM, Richard James	U. of Wisconsin	MD 1952	102-110 2nd Ave. S.W., Rochester, Minn.
BRUNSTING, Jr., Louis Albert	U. of Michigan	MD 1952	102-110 2nd Ave. S.W., Rochester, Minn.
CAMPAGNA, Mario Joseph	U. of Oregon	MD 1952	102-110 2nd Ave. S.W., Rochester, Minn.
CARPER, John Mark	Jefferson Med. Col.	MD 1952	102-110 2nd Ave. S.W., Rochester, Minn.
CHRISTU, Chris Nicholas	Louisiana State U.	MD 1952	102-110 2nd Ave. S.W., Rochester, Minn.
COMESS, Morton Seymour	U. of Illinois	MD 1952	102-110 2nd Ave. S.W., Rochester, Minn.
CUMMISKEY, Paul Vernon	U. of Minnesota	MD 1953	104 S. 4th St., Mankato, Minn.
DEVANNEY, Louis Raynor	Northwestern U.	MD 1939	102-110 2nd Ave. S.W., Rochester, Minn.
DOAN, Robert English	U. of Minnesota	MD 1953	Mpls. Gen. Hosp., Minneapolis, Minn.
DOUGLASS, Jesse Ellsworth	U. of Minnesota	MD 1952	2325 Stinson Blvd., Minneapolis, Minn.
HENDRICKSON, Russell Robert	U. of Minnesota	MD 1953	Cook Co. Hosp., Chicago, Ill.
HIGGINS, John Alden	U. of Minnesota	MD 1953	102-110 2nd Ave. S.W., Rochester, Minn.
HOWARD, David Lloyd George	U. of Toronto	MD 1948	102-110 2nd Ave. S.W., Rochester, Minn.
HUNTER, Samuel Wynne	U. of Rochester	MD 1947	U. of Minn. Hosp., Minneapolis, Minn.
JOHNSON, David Arthur	U. of Minnesota	MD 1953	Mt. Sinai Hosp., Minneapolis, Minn.
LOUGHEED, Lawrence Edward	U. of Manitoba	MD 1951	102-110 2nd Ave. S.W., Rochester, Minn.

Name	*School*		*Address*
MAHLE, James Parker	U. of Minnesota	MD 1953	St. Joseph's Hosp., St. Paul, Minn.
McSWEENY, II, Austin J.	Loyola U.	MD 1949	102-110 2nd Ave. S.W., Rochester, Minn.
MORROW, Jr., George William	U. of Illinois	MD 1952	102-110 2nd Ave. S.W., Rochester, Minn.
MOULTON, Keith Buell	U. of Minnesota	MD 1953	St. Joseph's Hosp., St. Paul, Minn.
NORDLUND, Mildred Eva	U. of Chicago—Rush	MD 1929	Cass Lake, Minn.
NORMAN, Jr., Mark Lewis	Marquette U.	MD 1952	1764 Grand Ave., St. Paul, Minn.
OLWIN, Thomas Kent	U. of Oregon	MD 1952	Ancker Hosp., St. Paul, Minn.
OZOLINS, Marta Birzins	U. of Latvia Riga, Latvia	MD 1927	274 Iglehart Ave., St. Paul, Minn.
PEAKE, Ronald Alan	Indiana U.	MD 1952	102-110 2nd Ave. S.W., Rochester, Minn.
PITTELKOW, Robert Bernard	Marquette U.	MD 1952	102-110 2nd Ave. S.W., Rochester, Minn.
POPOVICH, Dragojla, nee'Ostojich	U. of Belgrade Belgrade, Yugoslavia	MD 1939	Northwestern Hosp., Minneapolis, Minn.
RAILE, Richard Bayles	U. of Utah	MD 1950	1354 E. Maynard Dr., St. Paul, Minn.
RENO, III, George Louis	St. Louis U.	MD 1951	102-110 2nd Ave. S.W., Rochester, Minn.
SAFIRESCU, Sorin Radu	U. of Bologna, Italy	MD 1950	Fairview Hosp., Minneapolis, Minn.
SEYBOLD, Herbert McCelvey	U. of Texas	MD 1951	102-110 2nd Ave. S.W., Rochester, Minn.
STEWART, Allan H.	Geo. Wash. U., D.C.	MD 1952	102-110 2nd Ave. S.W., Rochester, Minn.
SVARE, Gerhart Trygve	U. of Wash., Seattle	MD 1951	102-110 2nd Ave. S.W., Rochester, Minn.
TAINTOR, Ronald W.	U. of Louisville	MD 1952	Ancker Hosp., St. Paul, Minn.
TERRY, Howard Lincoln	Northwestern U.	MD 1944	Anoka State Hosp., Anoka, Minn.
THOMPSON, Murray Cornwall	U. of Western Ont.	MD 1950	102-110 2nd Ave. S.W., Rochester, Minn.
TRAYNOR, Jr., Mack Vincent	Northwestern U.	MD 1950	102-110 2nd Ave. S.W., Rochester, Minn.
VANDEVER, Harry Wallace	U. of Tennessee	MD 1948	102-110 2nd Ave. S.W., Rochester, Minn.
VEINBERGS, Arnolds	U. of Latvia Riga, Latvia	"Physician" 1940	Swedish Hosp., Minneapolis, Minn.
ZIMERING, Sabina Szwarc	Ludwig Maximilian U. Munich, Germany	MD 1950	2741 Chicago Ave., Minneapolis, Minn.

Reciprocity Candidates

Name	*School*		*Address*
BETTS, Charles Samuel	Vanderbilt U.	MD 1951	102-110 2nd Ave. S.W., Rochester, Minn.
BLATTSPIELER, Lucien H.	U. of Nebraska	MD 1946	Worthington, Minn.
CANTRELL, Glenn	U. of So. Calif.	MD-1947	102-110 2nd Ave. S.W., Rochester, Minn.
CHRISTIANSON, Herbert B.	Marquette U.	MD 1939	102-110 2nd Ave. S.W., Rochester, Minn.
CONGDON, Gordon Hall	U. of Chicago	MD 1940	102-110 2nd Ave. S.W., Rochester, Minn.
CREAMER, Dick Oliver	Baylor U.	MD 1950	102-110 2nd Ave. S.W., Rochester, Minn.
DeLAND, II, Frank Howard	U. of Louisville	MD 1952	102-110 2nd Ave. S.W., Rochester, Minn.
ELLIOTT, Dean Cook	Ohio State U.	MD 1946	102-110 2nd Ave. S.W., Rochester, Minn.
ELLWOOD, Jr., Paul Murdock	Stanford U.	MD 1953	U. of Minn. Hosp., Minneapolis, Minn.
FAUCETT, Robert Lynn	U. of Kansas	MD 1944	102-110 2nd Ave. S.W., Rochester, Minn.
FONTANA, Robert Scott	St. Louis U.	MD 1949	102-110 2nd Ave. S.W., Rochester, Minn.
FOX, LeRoy John	Northwestern U.	MD 1945	NPBA Hosp., St. Paul, Minn.
HELMHOLZ, Jr., Henry F.	Johns Hopkins U.	MD 1937	102-110 2nd Ave. S.W., Rochester, Minn.
HONATH, Donald Herbert	Wayne U.	MD 1947	111 W. Main St., Owatonna, Minn.
KIRK, Jr., Thomas Allen	U. of Virginia	MD 1949	102-110 2nd Ave. S.W., Rochester, Minn.
LEAVENS, Milam Edmund	Baylor U.	MD 1948	102-110 2nd Ave. S.W., Rochester, Minn.
LIMBECK, Donald Arthur	U. of Nebraska	MD 1950	State Hosp., Fergus Falls, Minn.
LOES, Louis Augustine	U. of Iowa	MD 1947	1225 St. Germain St., St. Cloud, Minn.
MILLER, John William	U. of Louisville	MD 1951	102-110 2nd Ave. S.W., Rochester, Minn.
MORGAN, Edward Raymond	Tulane U.	MD 1950	102-110 2nd Ave. S.W., Rochester, Minn.
MUDD, Robert Hiden	U. of Pennsylvania	MD 1951	102-110 2nd Ave. S.W., Rochester, Minn.
PIFER, Lawrence Harrison	U. of Indiana	MD 1952	1218 N. 3rd St., St. Joseph, Mo.
REIFSNYDER, III, Wm. Henry	Jefferson Med. Col.	MD 1951	102-110 2nd Ave. S.W., Rochester, Minn.
RITTER, Donald Glenn	Indiana U.	MD 1952	102-110 2nd Ave. S.W., Rochester, Minn.
TERRY, Jr., Howard Richards	State U. of N. Y.	MD 1947	102-110 2nd Ave. S.W., Rochester, Minn.
TURNER, Jr., John Cooper	Tulane U.	MD 1951	102-110 2nd Ave. S.W., Rochester, Minn.
WALKER, Jr., Harry Charles	Med. Col. of Va.	MD 1951	U. of M. Grad. Sch., Minneapolis, Minn.
WALKER, Jr., Jack Andrew	Southwestern Med. Col.	MD 1948	102-110 2nd Ave. S.W., Rochester, Minn.

National Board Candidates

Name	*School*		*Address*
BIRKHEAD, Newton Charles	U. of Pennsylvania	MD 1949	102-110 2nd Ave. S.W., Rochester, Minn.
BOTTI, John Dominic	U. of Pennsylvania	MD 1952	102-110 2nd Ave. S.W., Rochester, Minn.
BUIE, Jr., Louis Arthur	U. of Minnesota	MD 1952	102-110 2nd Ave. S.W., Rochester, Minn.
DOYLE, Jr., John B.	Geo. Wash. U.	MD 1952	102-110 2nd Ave. S.W., Rochester, Minn.
ENSRUD, Earl Richard	Northwestern U.	MD 1952	102-110 2nd Ave. S.W., Rochester, Minn.
FABI, Mario Nestor	Geo. Wash. U.	MD 1950	102-110 2nd Ave. S.W., Rochester, Minn.
FOX, Irwin J.	New York Med. Col.	MD 1951	102-110 2nd Ave. S.W., Rochester, Minn.
GLASS, Bernard	Ohio State U.	MD 1950	102-110 2nd Ave. S.W., Rochester, Minn.

Name	*School*		*Address*
GREENFIELD, Duane Leonard	U. of Maryland	MD 1946	833 S. Phillips Ave., Sioux Falls, S. D.
HADER, Marvin	New York Med. Col.	MD 1952	102-110 2nd Ave. S.W., Rochester, Minn.
HELME, William Beach	Loyola U.	MD 1951	102-110 2nd Ave. S.W., Rochester, Minn.
KOGL, Richard Conrad	U. of Minnesota	MD 1953	Veterans Adm. Hosp., Minneapolis, Minn.
LEIGH, James Arthur	Bowman Gray Sch. of Med.	MD 1952	606 N. 3rd St., East Grand Forks, Minn.
MAGNESS, John Lower	Geo. Wash. U., D. C.	MD 1950	102-110 2nd Ave. S.W., Rochester, Minn.
MISRAHY, George Albert	Harvard U.	MD 1948	State Hospital, Hastings, Minn.
O'HOLLERAN, Lloyd Stephen	Creighton U.	MD 1952	102-110 2nd Ave. S.W., Rochester, Minn.
PEMBERTON, Henry Walter	Geo. Wash. U.	MD 1952	102-110 2nd Ave. S.W., Rochester, Minn.
PETERSEN, Deane Alfred	U. of Nebraska	MD 1946	1409 Willow St., Minneapolis, Minn.
RANDALL, Alan Douglas	Tufts Col. Sch. Med.	MD 1950	102-110 2nd Ave. S.W., Rochester, Minn.
ROTHWELL, Walter Spray	Harvard U.	MD 1947	102-110 2nd Ave. S.W., Rochester, Minn.
SHRINER, John Franklin	Tulane U.	MD 1950	1826 Clearmont St., Mobile, Ala.
STEINMETZ, Rodney Dunlap	Northwestern U.	MD 1952	102-110 2nd Ave. S.W., Rochester, Minn.
TWEEDDALE, Duane Newton	Creighton U.	MD 1951	102-110 2nd Ave. S.W., Rochester, Minn.
UPDIKE, II, Edwin Hoyt	Columbia U.	MD 1949	102-110 2nd Ave. S.W., Rochester, Minn.
WEISBART, Myron Herbert	Columbia U.	MD 1950	Veterans Adm. Hosp., Minneapolis, Minn.
WILLIAMS, Winfield Tyler	U. of Minnesota	MD 1952	Veterans Adm. Hosp., Minneapolis, Minn.

CORRECTION: Owing to an accident in the printing shop, several errors occurred in the list of licentiates as published in the February number of MINNESOTA MEDICINE. The publishers therefore are reprinting the list in its entirety in this issue to rectify the errors which appeared in the previous publication.

MINNESOTA STATE BOARD OF MEDICAL EXAMINERS

230 Lowry Medical Arts Building
Saint Paul, Minnesota

J. M. Jones, M.D., Secretary

FOUR SENTENCED AT MINNEAPOLIS FOR MANSLAUGHTER FOLLOWING ABORTION

Re: State of Minnesota vs. Willard B. Peterson, Joseph E. Verhota, Vernabell Tuma and Margaret Bishop Bellanger.

On March 27, 1954, the Hon. Levi M. Hall, Judge of the District Court of Hennepin County, sentenced each of the above named defendants following their pleas of guilty, which they had previously entered, to a charge of manslaughter in the first degree, the charge arising out of the death of a 23-year-old unmarried Minneapolis woman on February 7, 1954, resulting from an abortion performed upon her on January 30, 1954, by the defendant Bellanger.

Mrs. Bellanger, 29, 1116 Como Ave. S.E., Minneapolis, an employe of a downtown bar in that city, for the past four years, who had entered a plea of guilty to the charge before Judge Hall on February 16, 1954, was sentenced to a term of not less than five years nor more than twenty years in the Women's Reformatory at Shakopee, the sentence being stayed for five years, however, and the defendant placed on probation for the same period provided she serves the first two years of her sentence in the Women's Workhouse at Parkers Lake.

Following his plea of guilty to manslaughter in the first degree on February 26, 1954, Willard B. Peterson, 37, 226 N. Van Buren, Hopkins, Minnesota, who operates a telephone answering service in Minneapolis, was sentenced by Judge Hall to a term of not less than five years nor more than twenty years at hard labor in the State Prison at Stillwater and his sentence was also stayed for a period of five years, during which time he was placed on probation provided that as one of the conditions of his probation he pays the sum of $1,000 to the Treasurer of Hennepin County within two years.

The defendant Verhota, 38, 7501 Thirteenth Ave. S., Richfield, Minnesota, assistant manager of the same Minneapolis bar referred to above, who also entered his plea of guilty to the manslaughter charge on February 26, before being sentenced by Judge Hall admitted that he had a previous felony conviction in Hennepin County District Court on December 23, 1937, for using an automobile without the permission of the owner. He was then sentenced by Judge Hall to a term of not less than ten years nor more than twenty years at hard labor in the State Prison at Stillwater with the sentence being stayed for three years and the defendant placed on probation for the same period. Vernabell Tuma, 31, 3361 Xenwood Ave., St. Louis Park, Minnesota, employed in the same bar as a hostess, was sentenced to a term of not less than five years nor more than twenty years in the Women's Reformatory at Shakopee with the sentence being stayed for a period of three years during which time she was placed on probation by Judge Hall. Both Verhota and Tuma acted as intermediaries in arranging the abortion.

Mrs. Bellanger, who admitted she had performed a prior abortion in November, 1953, has no medical training or license to practice any form of healing in Minnesota. She did the abortion on January 30 in a Minneapolis hotel room by means of injecting a solution of soapy water and turpentine with a syringe. Peterson, who paid the $500 price for the abortion, turned the money over to Verhota and he kept $150 paying the balance to Tuma. She also retained $150 and paid $200 to Mrs. Bellanger. The young woman who submitted to the abortion was hospitalized subsequently in two Minneapolis hospitals but up until the time of her death she did not furnish the names of any of the defendants to the authorities, contending that the abortion was self-induced. Nevertheless, after her death an investigation disclosed that the facts and circumstances connected with the abortion were as related above.

Woman's Auxiliary

DOCTORS' WIVES URGED TO ATTEND DULUTH MEETING

A general invitation to all doctors' wives to attend the annual convention of the Woman's Auxiliary to the Minnesota State Medical Association, June 7, 8 and 9 in Duluth, has been issued by Mrs. C. H. Christensen, president of the St. Louis County Medical Society Auxiliary, hostess auxiliary for the meeting.

The convention is scheduled to coincide, as usual, with the 101st Annual Meeting of the doctors' organization and several important program events, such as the annual banquet and Open House, will be shared by both groups.

The women's program, arranged under the capable direction of Mrs. H. E. Bakkila, with the assistance of Mrs. I. T. Clark, both of Duluth, will begin with registration on Monday morning, June 7, at the Hotel Duluth. The pre-convention board meeting and luncheon will follow at the Kitchi Gammi Club. A tea, arranged through the courtesy of the Art Department of the University of Minnesota's Duluth Branch, will be a feature of Monday afternoon. It will be held from 3 to 5 p.m. at the Tweed Art Gallery, 2531 E. Seventh Street and it will be open to all visiting women. In the evening the women are invited to join their husbands at an Open House and entertainment to be staged in the Ballroom at the Hotel Duluth.

Tuesday's program begins with the annual business meeting and luncheon of the state Auxiliary which is scheduled for the Hotel Duluth. In the evening wives will be guests of their husbands at the annual banquet of the medical organization, also at the hotel. A "round-up" breakfast at the Duluth Women's Club Wednesday morning will bring the meeting to a close.

"Those who are already members of the Auxiliary know the state organization needs their support," Mrs. Christensen said in an appeal to all doctors' wives to attend the Duluth meeting and join in the Auxiliary's work.

"Like any organization, the Auxiliary is made up of individuals—which means you and me.

"We especially wish to urge all doctors' wives who are not already members to take this opportunity to acquaint themselves with the program of the Auxiliary. We feel quite sure they will find a spot in this program in which they would like to serve."

AUXILIARY CHAIRMAN REPORTS ON MEDICAL EDUCATION FUND

Some interesting and challenging figures, covering medical education in general and its relationship to the American scene, were provided in a report submitted recently by Mrs. J. A. Cosgriff of Olivia, Auxiliary chairman for the American Medical Education Foundation, for Minnesota.

There are seventy-nine medical schools in the United States whose function it is to train doctors to take care of a population of 158 million people, Mrs. Cosgriff points out in this report. These schools produce 6,500 doctors each year at a cost of $10,000 to $12,000 for each doctor. Tuition fees pay for about one-fifth of this amount. Taxes, donations, endowments must cover the rest. Only they failed by an estimated deficit of 19 million dollars, last year, to provide the amount required.

The trouble, according to the report, is that medical training is expensive. It takes 40 per cent of the total University budgets for only 10 per cent of the total number of students. And it is constantly growing more expensive as medical science advances. At the same time, endowments are dwindling, income from endowments has steadily decreased in purchasing power while costs have steadily risen. To complicate matters still further, the population has increased by more than 20 million since 1940 and is currently growing at the rate of 2,500,000 each year.

Alternatives Are Dangerous

The result for the seventy-nine accredited medical schools is obvious and inescapable. Unless new funds are made available most of them are faced with three equally dangerous alternatives. Either they must lower teaching standards or reduce the number of doctors graduated or accept subsidies from the federal government.

Government subsidy is patently dangerous, says Mrs. Cosgriff's report. To back up the statement she quotes President Eisenhower who said in a statement to Congress that the financial crisis facing medical schools "is a dangerous threat to the national welfare which must be met. The financial problems of medical schools should be solved through private rather than governmental means. Excessive reliance on the government violates the essential principle of our free-enterprise system." Clearly, such reliance could only invite federal domination of medical education.

Compromises in teaching standards, thus brought about, would soon be felt in every community. Local hospital services would deteriorate, family medical care would decline in quality and advances in medical research would be slowed and threatened.

Solution to the whole problem of medical school financing clearly rests upon a privately raised fund on a national level. The American Medical Education Foundation was established for this purpose in 1949, upon recommendation of the medical schools, the American Medical Association and representatives of industry, agriculture and labor. Three objectives were established. They were:

1. To make known the needs of medical education to the American people.

2. To raise annually from private sources the funds necessary to support the medical schools.

3. To distribute these funds equitably to the medical schools.

(Continued on Page 314)

✦ Reports and Announcements ✦

THE AMERICAN SOCIETY OF MEDICAL TECHNOLOGISTS

The twenty-second annual National Convention of the American Society of Medical Technologists will be held at Miami Beach, Florida, June 13-17. The co-headquarters hotels are the Deland and DiLido.

CONFERENCE ON HEALTH IN COLLEGES

Forty national organizations interested in aspects of health and education are joining with the American College Health association in sponsoring the Fourth National Conference on Health in Colleges, May 5-8 in New York. They will consider ways of protecting and improving the health of college students. The theme is "Teamwork in Meeting the Health Needs of College Students."

The conference program will include sixteen working committees in which college and university presidents, deans, physicians, nurses, psychologists, specialists in physical educational and health education, student counselors, and students will pool their knowledge and experience to develop comprehensive health programs integrated with all other college functions.

Previous conferences were held in 1931, 1936 and 1947.

INDUSTRIAL HEALTH CONFERENCE

The 1954 Industrial Health Conference will be held at the Hotel Sherman, Chicago, Illinois, from April 24-30. The registration fee is $3.00. Reservations are to be made with the Industrial Health Conference Housing Bureau, Hotel Sherman.

April 26-30—Industrial Medical Association

April 25-27—American Conference of Government Industrial Hygienists

April 26-29—American Industrial Hygiene Association

April 27-29—American Association of Industrial Dentists

April 26-30—American Association of Industrial Nurses, Inc.

April 24-30—U. S. Navy I. H. Organization

EASTERN STATES HEALTH CONFERENCE

The 1954 Eastern States Health Education Conference of the New York Academy of Medicine will be held at the New York Academy of Medicine on Thursday and Friday, April 29 and 30. The program will include four sessions and an evening dinner meeting. The subject of this year's conference will be "Communication in Health Education." A workshop session will be held as part of the education conference and the concepts of communication will be applied to and tested in current health education materials and procedures.

Participation in the conference is limited. Those interested in the conference should mail their requests to Iago Galdston, M.D., The New York Academy of Medicine, 2 East 103 Street, New York 29, New York.

MEDICAL LIBRARY ASSOCIATION

The Medical Library Association will hold its fifty-third annual meeting June 15-18 in Washington, D. C. The headquarters will be the Hotel Statler, and the official host the Armed Forces Medical Library. The program will include a discussion on medical research by embassy attaches, tours of the National Institutes of Health, the National Naval Medical Center, and of the Armed Forces Medical Library. Delegates to the meeting will hear addresses by Dr. Detlev Bronk, President of the Rockefeller Institute of Medical Research, Lt. Col. Frank B. Rogers, Director of the Armed Forces Medical Library, Mr. Verner Clapp, the Acting Librarian of Congress, and Dr. Raymond Zwemer, Chief of the Library of Congress' Science Division. Further information can be obtained from Lt. Col. Frank B. Rogers, Armed Forces Medical Library, 7th Street and Independence Avenue S.W., Washington 25, D. C.

MINNESOTA SOCIETY OF NEUROLOGY AND PSYCHIATRY

A regular meeting of the Minnesota Society of Neurology and Psychiatry was held at the Town and Country Club in Saint Paul on March 9. The program consisted of "Clinical experience in the use of succinylcholine chloride (anectine) in electroshock therapy" by Dr. Burtrum Schield and Mr. Philip Margolis.

ALCOHOL PROBLEMS CONFERENCE AT UNIVERSITY APRIL 1-3

Drinking among young people and drinking and driving are two major topics which were considered at a University of Minnesota conference on the problems of alcohol April 1-3 at the University's Center for Continuation Study.

On the conference program were such outstanding authorities on the alcohol problem as Selden D. Bacon, director of the center of alcohol studies at Yale university; Dr. Paul Stevenson, psychiatrist of the National Institute of Mental Health, United States Public Health Service, Bethesda, Maryland, and the Rev. Forrest L. Richeson of Portland Avenue Christian church, Minneapolis.

The conference was planned by the University and the Minnesota Department of Health for physicians, nurses, welfare workers, judges, teachers, clergymen and other community leaders.

"Prime purpose of this conference," according to William F. Ferguson, chief of the section of mental health and consultant on alcoholism for the state health department, "was to mobilize communities to combat alcoholism. We hope the conference brought to light information about problems of alcohol in our society."

Bacon, a sociology professor who is considered the foremost authority on alcoholism, spoke on such subjects as "Drinking: Customs, Confusion and Conflict," "Nature and Extent of the Problem of Alcoholism," "Alcoholism—Therapy and Prevention" and "Mobilizing Community Resources to Combat Alcoholism." Bacon and an associate are the authors of the recently published book, "Drinking in College."

Stevenson's subjects were "Personality of the Problem Drinker" and "Alcoholism and the Family."

Rev. Richeson, who has personally counselled about 800 alcoholics in Minnesota, talked about a clergyman's contribution to an understanding of alcoholism.

HEART DISEASE COURSE AT UNIVERSITY

A course in cardiovascular diseases for general physicians was presented by the University of Minnesota and the Minnesota Heart Association March 22-24. In conjunction with the course, Dr. Samuel A. Levine, clinical professor of medicine, Harvard university, presented the annual George E. Fahr lecture. His topic was "Some Pitfalls in the Care of Cardiacs."

SCHERING AWARD SUBJECTS ANNOUNCED

To encourage scientific reporting and to stimulate medical students' interest in the ever expanding fields of therapy, the Schering Award Committee has announced the beginning of the ninth consecutive competition in 1954.

Titles of three subjects on which students in the United States and Canada are being invited to submit papers have been made known by George Babcock, Jr., M.D., chairman of the committee. They are:

(1) The Use of Androgen Therapy in the Female

(2) The Prophylactic and Therapeutic Uses of Parenteral Antihistamines

(3) Modern Treatment of Infections and Allergic Disorders of the Eye

For the best paper on each of these subjects, the committee will present one $500 first prize and a $250 second prize. Special citations and professionally useful gifts will also be awarded to all students who submit papers of merit. Outstanding medical authorities in each field will judge the papers.

Deadline for entry forms specifying the student's chosen title is July 1. All manuscripts must be mailed not later than October 1. Students may compete individually or co-operatively in research teams.

The purpose of the Schering Award is to encourage original reporting in the hope that students will later contribute to the essential communication of knowledge throughout the medical profession.

The Schering Award Committee will bring outstanding papers to the attention of editors of appropriate professional journals. Information and instructions for the competition are available from Schering Corporation, 2 Broad Street, Bloomfield, N. J.

ANATOMY BUILDING NOW JACKSON HALL

A University of Minnesota campus structure, known as the Anatomy building since it was erected forty-two years ago, has been redesignated Jackson Hall by order of the University's Regents. The hall was named for the late Dr. Clarence M. Jackson who became head of the division of anatomy in 1913 and lifted that department to a position of preeminence in the field of medical science.

Dr. Harold S. Diehl of the school of medical sciences announced the naming of Jackson Hall, April 1, at a dinner in the University's Campus club preceding the annual Clarence Jackson lecture. Dr. J. Garrott Allen, professor of surgery at the University of Chicago medical school, delivered the lecture, "Management of Upper Gastro-Intestinal Bleeding."

At the dinner, Elexious T. Bell, University professor emeritus of pathology, reviewed Dr. Jackson's service to the University.

Dr. Jackson, who died at the age of seventy-one on January 17, 1947, gained recognition as one of the first medical school leaders to launch a modern research department. He was acting dean of the graduate school in 1917-18 and again in 1925. He retired in 1941.

Dr. Jackson was editor of Morris Human Anatomy, a widely used textbook, and head of the medical division of National Research Council in Washington, D.C., in 1923-24.

WOMAN'S AUXILIARY

(Continued from Page 312)

The AMEF got off to a slow start but now shows forty-one out of the forty-eight states making substantial contributions to the fund.

The Woman's Auxiliary to the American Medical Association contributed $100,000 from its treasury, and individual state and county auxiliaries gave more than $22,000 in 1953.

1954 Contributions Requested

"We auxiliary members have pledged ourselves to help in this most worthy cause," Mrs. Cosgriff reminds the women in this report. "Let us redouble our efforts this year.

"We are still hoping," she says, "that every county auxiliary in the State of Minnesota will make a voluntary contribution to the fund before the year 1954 ends. Earmark your contribution. That is, make a notation on your check indicating whether you wish it to be used for the University of Minnesota, specifically, or if you wish it merely to be added to the general fund. Send it to the treasurer of the State Auxiliary, Mrs. Charles W. Froats, 1406 Edgcumbe Road, Saint Paul, Minnesota."

In that connection, Mrs. Cosgriff notes that "In Memoriam" cards are still available to members, and may be obtained from the state AMEF chairman.

In Memoriam

WILFRED P. FRELIGH

Dr. Wilfred P. Freligh, Albert Lea surgeon, died after a brief illness at St. Mary's Hospital in Rochester, February 28, 1954. He was sixty-six years old and had practiced at Albert Lea for the past thirty-four years.

Dr. Freligh was taken ill while he was vacationing in Phoenix, Arizona. He was flown back to Rochester with his wife the day before he died.

He was born in Stillwater and went to school there and at the University of Minnesota, where he received a B.A. degree in 1911 and his medical degree in 1913. He served internships at Abbott Hospital in Minneapolis and at Ancker Hospital in St. Paul. A fellowship followed at the Mayo Clinic in the course of which, he was Dr. Charles H. Mayo's assistant for three and one-half years.

In 1922 Dr. Freligh established himself in Albert Lea and became a member of the staff of the Naeve Hospital of which he was a past-president. He was a lieutenant in the U. S. Army Medical Corps during World War I and was active during his entire career in city, county and state medical groups. He was a member of the Freeborn County Medical Society which he served in a number of official capacities, the Minnesota State Medical Association and the American Medical Association. He was a member, also, of the Alumni Association of the Mayo Clinic, a warden of Christ Episcopal Church of Albert Lea, a past commander of Leo Carey Post of the American Legion, a past director of the Albert Lea Chamber of Commerce, a member of the Elks, of the YMCA and of the Country Club.

Surviving are his wife, the former Irene V. Comstock, and one son, Robert John, 17; also a sister, Mrs. Everett Doran, Sr., of Los Angeles, and a brother, Harold, of Omaha, Nebraska.

GERALD ARTHUR KNUTSON

Dr. Gerald A. Knutson, well-known Northern Minnesota physician, died suddenly as the result of an embolism, at his home in Hallock, Sunday, March 3, 1954. He was forty-eight years of age.

Death came as the probable consequence of an automobile accident which occured a week earlier as he and his wife were returning to Hallock from Grand Forks where he had gone for a physical examination at a clinic there. Mrs. Knutson suffered several injuries, including probable loss of an eye, and was hospitalized at the time of Dr. Knutson's death. Dr. Knutson, himself, had apparently escaped serious injury and was preparing to go to his office when he was stricken.

He was born at Buxton, North Dakota, the son of Dr. O. A. Knutson, a physician, there, and Mrs. Knutson. He attended the Buxton schools and the University of North Dakota from which he was graduated with B.A. and B.S. degrees. He received his degree in medicine from Northwestern University and served his internship at Ancker Hospital in St. Paul. He practiced medicine in Greenbush for eight years until a fire wiped out his hospital, home and office there. After that he applied for a commission in the Navy and served for three and one-half years during World War II. He held the commission of lieutenant in the Navy when he was discharged and shortly afterwards he went to Hallock to take over the practice of the late Dr. A. W. Shaleen.

Dr. Knutson was widely known in Northern Minnesota. He had recently accepted an appointment to the local board of health of Hallock and he was a member of the Lutheran Church there. He was also a member of the Red River Valley Medical Society, the Minnesota State Medical Association and the American Medical Association.

Surviving are his wife, the former Rush Aaseby, and five children including Carol, now a student at the University of Minnesota, Gerald and Aileen, high school age, Cynthia, a Hallock grade school student, and Richard, below school age. His parents, Dr. and Mrs. Knutson of Buxton survive him, also a brother, Dr. George Knutson, a physician of Negaunee, Michigan, and one sister, Mrs. G. L. Hanson of Mount Vernon, Washington.

FRED HARWOOD POWERS

Dr. Fred Harwood Powers, Rochester physician for many years, died February 27, 1954, at St. Mary's Hospital in Rochester after a long illness.

Dr. Powers was born in Bloomington, Illinois. He attended high school at Shelbyville, Illinois, and graduated in 1890 from the University of Illinois College of Pharmacy. Later he attended Rush Medical College in Chicago and received his medical degree there in 1900.

He practiced for some years in Illinois, Wisconsin and Ohio and then established himself in general practice, twenty-eight years ago, at 301½ South Broadway in Rochester. He was a member of the Olmsted-Houston-Fillmore-Dodge County Medical Society, the Minnesota State Medical Association and the American Medical Association. He became a life member of the State Association in 1952.

At one time, Dr. Powers operated a small, private hospital in Rochester. He was on the examining board for the air forces, also. He was a 32nd degree Mason, a member of the Order of the Eastern Star, the White Shrine and AOUW.

Surviving are his wife, the former Cora Sprecher, and three daughters, Mrs. Margaret Parish, Chicago; Mrs. George T. Mellinger, New Orleans, Louisiana; and Mrs. Grant H. Elford, Audubon, Iowa. There are five grandchildren.

Of General Interest

Establishment of an outpatient psychiatric clinic for adults in Saint Paul with **Dr. Clarence J. Rowe** as director of the clinic, has been announced.

The psychiatric center will provide free or part-pay treatment of the mental and emotional ills of persons who are unable to pay the cost of private psychiatric service.

The clinic will accept only patients who are referred to it by physicians, public health agencies, other outpatient clinics, the courts and social agencies.

Recommendation for the establishing of such an outpatient psychiatric clinic for adults came from a committee of doctors, social workers and lay people appointed in June, 1952, by the Saint Paul Area Public Health Council. This committee, headed by Mrs. John Driscoll, made a study of the need for psychiatric services for those persons who are unable to pay the cost of private psychiatric treatment.

The committee report and the establishment of a clinic were approved by the Public Health Council, the practicing psychiatrists in Saint Paul, the executive committee of the Ramsey County Medical Society and the planning and research council of the Saint Paul Community Chest and council.

Location of the clinic has not yet been definitely determined, but it will probably be in a downtown office building. It is hoped that operation can begin about July 1, when Dr. Rowe's appointment takes effect.

Dr. Rowe will head a professional staff that will include a clinical psychologist and three psychiatric social workers. He has been on the staff of the University of Minnesota School of Medicine since 1949, and is an assistant professor in the department of psychiatry and neurology. He has also been attending psychiatrist to the Veterans Administration Hospital for three years and psychiatric consultant for the Family Service of Saint Paul. He is a member of the American Psychiatric Association and the Minnesota Society of Neurology and Psychiatry as well as a diplomate of the American Board of Psychiatry and Neurology.

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Dr. George N. Kraemer, formerly of Mankato, has joined the staff of the Thayer-Wandke clinic. Dr. Kraemer is a graduate of the University of Minnesota, a veteran of World War II, and a resident in surgery at Minneapolis General Hospital from 1949-1953.

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Dr. Allen Hodges, Albert Lea, and **Dr. Tracy Barber,** Austin, discussed the functions of a mental health clinic and problems in mental health at Neveln PTA in Austin.

Dr. J. J. Heimark, Fairmont physician who has practiced in Tri-Mont the past year and a half, is returning to Fairmont to resume his practice in his former office there.

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Dr. Frances E. Schaar recently opened her office at sixty-seventh and Lyndale as a pediatrician. She has been with the University hospital since 1947, and is a graduate of the University of Illinois medical school.

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Doctors C. G. Wingquist and **A. R. Anderson** have moved into their newly completed office building in Crosby. The one-story structure houses nine rooms on the left of the building and five-room dental office on the right. The exterior has stuccoed sides and a brick front. The lots on either side of the building will provide parking areas for patients.

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Dr. A. I. Arneson, Morris, was the speaker for the Father-Son Banquet at the Glenwood Lutheran Church on February 15. Dr. Arneson has made a number of expeditions out of the States hunting big game and he showed one of his motion pictures taken while hunting in British Columbia.

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Dr. Theodore Bratrud, Thief River Falls, started his new duties on the staff of the department of pathology, March 1. He will complete his work for a Ph.D. degree in pathology.

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Dr. F. H. Baumgartner, Albany, was honored at a testimonial dinner for his ten years of faithful service as mayor of Albany. **Dr. J. B. Beuning,** former Albany physician and surgeon, was the principal speaker.

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Dr. W. F. Maertz, formerly of Saint Paul, has been appointed to the staff of the Anoka State Hospital.

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Dr. Harry M. Weber, Rochester, was among eighteen on a guest faculty that assisted in the annual postgraduate course in radiology at the University of Kansas medical center in Kansas City in February.

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Dr. J. K. Butler, Cloquet, was elected to the executive committee of St. Mary's hospital, Duluth, at their annual staff meeting held on February 4. Dr. Butler will be chief of the department of general medicine at St. Mary's.

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Dr. Andrew V. Grinley, Grand Rapids, was elected chief of staff at Itasaca Memorial hospital in February.

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Dr. and Mrs. C. J. Olson, Belle Plaine, left February 1 for Temple, Texas, where he took a two-week postgraduate course in heart disease at the Scott Sherwood-Brindley Foundation of the University of Texas Medical School.

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Lt. Donald S. Amatuzio, formerly of Duluth, now serving with the U. S. Navy in Japan, recently participated in a unique joint meeting of United States Medical Officers and the Yokosuka and Miura Medical society. Lt. Amatuzio spoke at the meeting attended by 50 Japanese and 100 American doctors. As part of a technical session, Lt. Amatuzio discussed the rapid intravenous dextrose tolerance test. He is a specialist in internal medicine.

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Dr. C. W. Moberg, Detroit Lakes, was guest speaker at the February meeting of the Business and Professional Women's Club. His topic was rheumatic fever.

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Dr. Ralph E. Smith, Department of Medicine at the Veterans and University Hospitals, Minneapolis, was the guest consultant discussing valvular heart disease at the monthly staff meeting of the Litchfield Hospital.

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Dr. Beryl Kennedy of the University of Minnesota Hospital staff meet with the Meeker County Hospital Medical staff at their monthly meeting on March 2. Dr. Kennedy discussed breast cancer with special emphasis on the effect of the use of hormones in its treatment. The clinical conference program throughout Minnesota is sponsored by the Minnesota Department of Health, Minnesota Medical Association, University of Minnesota, Minnesota Heart Association, and the Minnesota Cancer Society.

Volunteer leaders of the American Cancer Society's Minnesota Division, met on February 10 and 11 in Minneapolis. They planned an expanded program of cancer control, education and service. **Dr. Bernard Zimmerman**, Cancer Co-ordinator, University of Minnesota, headed a round-table discussion "The Cancer Problem." Participating in this panel were: **Dr. Marthella J. Frantz, Dr. F. John Lewis, Dr. Donn Mosser,** and **Dr. B. J. Kennedy,** all of the University School of Medicine.

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Dr. Marguerite Schwyzer, Saint Paul, suffered a fractured leg in an accident north of Pine City. En route to Miller Hospital, in an ambulance, February 22, they were involved in another collision on Ninth and Main streets in Saint Paul.

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Dr. Robert L. Pedersen, who has been associated with **Dr. W. E. Macklin** in the X-ray and radiology department at the Rice Memorial Hospital, Willmar, left for Brainerd, where he will head a similar department at the hospital there.

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Dr. Frank H. Krusen, chief of the section on physical medicine, Mayo Clinic, received the President's award for 1953 at Louisville, Kentucky, February 24, during the American Medical association's fourteenth annual Congress on Industrial Health. The award is conferred annually for outstanding contributions to the employment welfare of the handicapped. Dr. Krusen is chairman of the AMA council on physical medicine and rehabilitation.

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Dr. Martin O. Wallace, Duluth, spoke at the Duluth Health service annual luncheon meeting on February 25. His subject was "The present status of health insurance plans."

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Dr. M. Smorazczok, Monticello, spoke to the PTA in Annandale on February 15. Dr. Smorazczok is a native of Poland; he spent two years in a concentration camp. He spoke on various phases of government under the Communist rule, schools, religion, freedom and other points.

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William R. Merifield will be associated with **Dr. Marvin J. Geib**, Moorhead, in his practice of psychiatry as Clinical Psychologist. Mr. Merifield is a graduate of the College of William and Mary with a major in Clinical Psychology. His internship was completed in the Neuro-psychiatric service at Fort Lawton, Washington.

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Dr. E. J. Engberg, of the Minnesota School and Colony, Faribault, has satisfied the standards set up by the Advisory Board for Medical Specialties for inclusion in the current edition of the Directory of Medical Specialists. Dr. Engberg has qualified as a specialist in the field of psychiatry and neurology.

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Dr. and Mrs. H. L. Stemsrud, Alexandria, left February 26 for a ten-day trip to visit relatives in Milwaukee and to attend a medical conference at the Palmer House in Chicago.

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Dr. J. L. Delmore, Sr., Roseau, was honored with a membership card in the Minnesota Medical Association in the form of an inscribed gold plate in recognition of his 40 years of service in the medical profession.

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Dr. Paul G. Bauer has opened an office for the general practice of medicine in Faribault. A graduate of the University of Minnesota School of Medicine, Dr. Bauer took his internship at St. Barnabas Hospital. He served in the Army for three years. His tour of duty included 18 months in Korea as a regimental surgeon.

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Duluth physicians have accepted a goal of $200,000.00 for themselves in the $1,250,000 St. Mary's Hospital Building Fund campaign. **Dr. R. P. Buckley**, chairman of the medical division, made the announcement at the sponsor's committee dinner, February 16. He said Duluth physicians had determined the size of their objective themselves, and had approved it. Teams of doctors will solicit fellow-practitioners in an effort to reach or surpass the goal.

The Renville County Hospital at Olivia commemorated its third anniversary on February 5. In the period since its opening, the hospital has served 5,356 men, women, and children as in-patients admitted, as well as 1,238 people treated as out-patients. During the same period, 1,725 surgical operations have been done in the hospital and 931 babies have been born. **Dr. J. A. Cosgriff**, Olivia, expressed thanks to the members of the Hospital auxiliary for the service they have rendered to the hospital.

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Dr. H. P. Van Cleve, Austin, addressed the Whittier PTA on February 16. He discussed the need for a mental health clinic and the purposes of a family counseling service.

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Dr. Martin O. Wallace, Duluth, was the principal speaker at the Duluth Health service's annual dinner. He warned against health plans of too extensive a nature. Dr. Wallace asserted that in England where compulsory plans are extensive abuses have led to lower quality of medical service. "Voluntary, rather than compulsory insurance is cheaper in the long run and maintains a better doctor-patient relationship," Dr. Wallace said.

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Open house was held February 20 in the new clinic building which was opened by **Dr. W. J. Alcorn** in Wabasso. The building includes a waiting room, a room for administration of various types of treatment, the doctor's private and main office, two examining rooms, a pediatric room for care of infants, x-ray room, laboratory room and darkroom. The structure is 31 feet wide and 45 feet long. Construction was begun last summer.

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Dr. W. D. Cleaves, Sauk Centre, has moved into new offices in his recently constructed building on Third street.

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Dr. Tracy Barber, Austin, was elected president of the Austin YMCA at the kickoff banquet for the 1954 membership enrollment campaign February 17. Dr. Barber is a charter member of the YMCA and a past president of the board of directors. He has been medical director of the George A. Hormel Company since 1948.

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Problems of investigating violent deaths were discussed at the University of Minnesota's coroners' conference March 8. **Dr. Walter Jetter**, professor of legal medicine of Boston university school of medicine, spoke on poisonings, deaths in conflagrations and drownings and suspected drownings. Milton Helpern, assistant professor of forensic medicine, New York university, discussed stabbings and rape, and Briggs J. White of the Federal Bureau of Investigations talked about "trace evidence" and facilities of FBI laboratories.

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Dr. Richard A. Groat, a cosultant in the section on anatomy and associate professor of anatomy in the Mayo Foundation, has resigned from the staff of the Mayo Clinic to begin an internship at City Memorial hospital, Winston-Salem, North Carolina.

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Four Mayo Clinic surgeons participated in the scientific program of a sectional meeting of the American College of Surgeons at Omaha, Nebraska, on March 1-4. **Dr. Thomas H. Seldon** took part in a symposium on trauma; **Dr. Frederick A. Figi** participated in a discussion of cancers of the head and neck; **Dr. O. T. Clagett** took part in a symposium on esophagogastric junction; and **Dr. Howard K. Gray** discussed lesions of the pancreas.

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Dr. Robert B. Pierce is dividing his time in the practice of medicine between Renville and Olivia.

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Dr. Byrl Kennedy, Assistant Professor of Medicine, University of Minnesota, spoke at the first of four clinical conferences for the McLeod County Medical Society in Glencoe on February 25. He discussed new drugs in cancer treatment.

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Dr. Joseph S. Collins, Wabasha, is completing his fiftieth year as a physician and surgeon and was made a life member of the Minnesota State Medical Association and the Wabasha County Medical Society.

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Drs. R. P. Griffin and **N. W. Wagner**, Benson, announced the formation of a partnership which went into effect March 1. Dr. Griffin, a navy veteran, has been in practice in Benson for seven years. Dr. Wagner became associated with Dr. Griffin about five months ago; previously he had been in Graceville.

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Dr. and Mrs. C. F. Cervenka, North Branch, recently attended the Chicago Medical Society's annual clinical conference which was held at the Palmer House from March 2 to 5.

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Dr. Robert Gray, Marshall, recently spoke to the Marshall chapter of the University Women at their monthly meeting. He spoke of his experiences in Africa where he spent several years as a physician with the British and American armies and of his findings when he returned later under a Fullbright Scholarship to study native medicines. He told of the problems, politically and socially, that face Africa and those endeavoring to educate and civilize the many tribes there.

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Dr. Donald E. Dille, Litchfield, attended the University of Minnesota coroners' conference March 8-10.

The process of incorporating to bring a medical center to Hawley began early in February. Shares of stock are to be sold for $100. Committees have been appointed to inspect various medical centers.

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Dr. Paul Winchell was the guest consultant at the first of two clinical conferences for the medical staff at the Montevideo Hospital on March 8. Dr. Winchell of the Department of Medicine at the Heart Hospital on the University Campus discussed case histories on heart disease, particularly emphasizing the types of cardiac diseases that are suitable for heart surgery.

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Dr. Gaylord Anderson, director of the school of public health at the University of Minnesota, spoke to the annual mid-winter meeting of the Women's Auxiliary to the Minnesota State Medical association at the International Institute. He discussed the new polio vaccine and gave general information on the work of the physician and how doctors' wives fit into the picture.

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Dr. Julian S. Ansell, medical fellow at University hospitals and Veterans hospital, and **Dr. Frank MacDonald,** staff physician at the Veterans Hospital, were guest speakers at a one-day private duty institute of the Minnesota Nurses association February 18 and 19.

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Dr. John Tobin, pediatrician, took part in a panel discussion for parents at St. George's Episcopal Church in St. Louis Park.

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Mr. and Mrs. Ivan H. Roberts, Judson, announce the engagement of their daughter, Gloria, to **Dr. Frank J. Carthey,** New Ulm. Dr. Carthey is a graduate of the School of Medicine and the University of Minnesota and is now associated with the Seifert Clinic of New Ulm.

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Dr. Ivan G. Baronofsky, assistant professor of surgery at the University of Minnesota, discussed "Research in the Heart Field" at a luncheon meeting of the Exchange club in Saint Paul on February 2.

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Dr. Robert G. Tinkham, Minneapolis, was elected vice chairman of St. Barnabas hospital medical staff in February.

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Dr. Andrew V. Grinley, Grand Rapids was elected chief of staff at Itasca Memorial Staff in February. **Dr. John Evensta** of Grand Rapids will be assistant chief, and **Dr. R. T. Kelly** of Nashwauk was named secretary. Members of the executive committee for the medical staff are **Dr. O. C. Braun, Dr. Gordon M. Erskine** and **Dr. M. J. Schirber.**

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Dr. J. K. Butler, Cloquet, was elected to the executive committee of St. Mary's hospital, Duluth, at their annual meeting held on February 4. Dr. Butler will be chief of the department of general medicine at St. Mary's.

A conference on sterility and associated problems of reproduction in the human race was presented for physicians at the University of Minnesota center for continuation study in February. Dr. Alan F. Guttmacher, director of the department of obstetrics and gynecology, New York City, and a clinical professor at Columbia, was the guest speaker.

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Dr. Edward Allen Boyden, head of the anatomy department in the University of Minnesota medical school, received a special tribute from his friends and associates in observance of his sixty-eighth birthday March 20.

The tribute was the "Boyden Birthday Volume" of *The Anatomical Record,* scientific monthly journal published for anatomists. The special volume consist of four issues of the publication, January through April, 1954. Dr. Boyden, who has been a professor of anatomy at the University since 1931, served as managing editor of the journal for 20 years, 1928-48.

The January issue consists of a short biography of Dr. Boyden, a list of his many publications and five scientific papers contributed by eleven of his colleagues in the anatomy department. Additional papers by former associates will appear in the February, March and April issues.

Dr. Boyden received most of his training at Harvard university where he earned his doctor of philosophy degree in the medical sciences in 1916. He also studied medical anatomy for a year at the University of Freiburg, Germany. He has served on the staffs of six American universities: Harvard, Stanford, the University of California, the University of Illinois, the University of Alabama and the University of Minnesota. His publications include three books, 111 articles in scientific journals and some fifty abstracts and book reviews.

The Minnesota anatomist's studies of the kidneys, gallbladder and lungs have brought him international fame. According to one of his associates, he has made a greater number of important contributions to knowledge of the structure and functioning of the gallbladder and related tubes than any other investigator.

In 1925, he published the first x-ray picture of the normal contraction of the gallbladder in response to a meal of egg yolk. The "Boyden meal" of egg yolk is still a standard test for biliary disease.

During the last eight years, Dr. Boyden has made an intensive study of the segmental anatomy of the lungs. His observations are considered invaluable to the surgeon who wishes to remove a small, diseased part of one lobe of a lung.

His contributions in the field of x-ray diagnosis of gallbladder and lung diseases are the most important of those of any living anatomist—in the judgment of a colleague in the University medical school.

"Through the years, Dr. Boyden has brought illumination to every subject he has investigated," Dr. Charles H. Danforth, professor emeritus of anatomy, Stanford university, asserted today. "He has been, and continues to be, a potent influence in shaping the quality and the direction of the science of anatomy in America."

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Dr. Walter Judd was the principal speaker at a Ninth District Lincoln Day dinner attended by well over 200 people in Warren. **Dr. Ch. H. Holmstrom,** Warren, served as toastmaster.

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Dr. Ch. H. Holmstrom, Chairman of the Warren School Board, spoke at the opening of Warren's new gymnasium.

A substantial increase in patients treated at Warren hospital has been noted. Last year a total of 1,842 patients were admitted as compared to 1,603 in 1952, 1,736 in 1951 and 1,697 in 1950. Total number of patient days increased from 8,507 in 1952 to 9,675 last year. The daily average was 26.5 patients.

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Dr. Donald Steward, Northwestern Clinic, Crookston, spoke to the members of the Rosseau County Medical Society at the home of **Dr. and Mrs. Lloyd Klefsted,** Greenbush. Dr. Steward spoke on "Bowel obstruction." Several types of tubes for intubation were shown and demonstrated as well as x-rays of actual cases.

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Dr. O. M. Geilberg is the new president of the medical staff of the Worthington Municipal hospital. He was named at the annual election meeting of the group held recently. Other officers named include **Dr. D. J. Halpern,** Brewster, vice president, and **Dr. F. L. Shade,** secretary.

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Dr. W. F. Skaifé was elected chairman at the recent annual meeting of the Nursing Advisory Board, Little Falls. The board is aiding the nursing service to compile a list of women in the county who are willing to help care for children or old people when assistance is needed.

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Dr. John F. Kane, who has been associated with the Hartfiel Medical Center, is leaving Montevideo to become chief surgeon at the Veterans Administration hospital in Miles City, Montana. **Dr. George Martin** and **Dr. J. J. Carroll** who have also severed their connection with the Hartfiel Center are completing arrangements for practice in other cities.

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Officers were elected at the annual meeting of the Upper Mississippi Medical association in Brainerd. Ten counties were represented by 53 doctors. The new officers include **Dr. C. W. Vandersluis,** Bemidji, president; **Dr. V. E. Quanstrom,** Brainerd, first vice-president; **Dr. Luther Davis,** Wadena, second vice president; **Dr. W. Parker, Sebeka,** third vice president; **Dr. G. I. Badeaux,** Brainerd, secretary. Chosen as delegates to the state society were **Dr. Luther Davis,** Wadena, and **Dr. A. M. Watson,** Royalton. Alternates are **Dr. William Anderson,** Brainerd, and **Dr. Douglas Johnson,** Little Falls.

Failure to report several cases of less serious communicable diseases has resulted in a very healthy appearing annual health report for the city of Fairmont, **Dr. M. H. Marker,** city health officer, said as he turned over his report to the city council at its February meeting. The report shows that the city had three cases of polio, two of typhoid fever, one of tuberculosis and one of scarlet fever.

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Quarrelling in children is not all bad, **Dr. Robert Faucett,** psychiatrist and a member of the Mayo Clinic staff, told members of the parents group of the Aldrich Memorial Nursery school. He said that there are some good and constructive things about sibling rivalry in that it can teach a child how to get along in a competitive world. Dr. Faucett was moderator for a panel discussion on "Sibling Rivalry and Jealousy in Children." Also taking part was **Dr. H. F. Helmholz, Sr.,** emeritus member of the Mayo Clinic staff in the pediatrics section; he pointed out the importance of trying to see a situation from a child's point of view and emphasized that parents must avoid making children jealous by comparing their accomplishments with others.

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Dr. and Mrs. H. J. Elliott of the Hayfield Medical Center showed slides of their work as medical missionaries in Africa at a meeting of the Kasson PTA in February.

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Dr. Grace Roth of the Mayo Clinic and associate professor in physiology of the Mayo foundation spoke on "You and Your Heart" at a dessert meeting of the Scandinavian club February 22 at the YMCA in Rochester.

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A plan which would make cancer tests available to everyone in Minnesota was explained by **Dr. David P. Anderson,** Austin, president of the Minnesota Cancer Society, at a meeting of the volunteer cancer workers. In his speech Dr. Anderson said that under the program sponsored by the Minnesota State Medical Association, doctor's offices would be made cancer detection centers. Patients would undergo the tests designed to detect cancer before any symptoms appear, as a part of their yearly physical examinations.

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The Medical Staff of the Lutheran Hospital held its business meeting February 17. The following officers were elected: **Dr. Harry Palmer,** Blackduck, president; **Dr. John Griffin,** vice president, and **Dr. D. D. Whittemore,** secretary-treasurer.

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Dr. L. R. Gowan spoke to the St. Mary's Council on Volunteers, Duluth, on the subject "Psychiatry in the general hospital."

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Two University of Minnesota surgeons and a former member of the University medical school pediatrics staff have been awarded grants of $30,000 each by the John and Mary R. Markle Foundation of New York which has named them "Scholars in Medical Science." Announcement of the awards was made by John M. Russell, executive director of the foundation.

The Minnesota scholarship winners are: **Dr. Gilbert S. Campbell,** 829 25th Avenue S.E., Minneapolis, resident in surgery, and **Dr. Mitchell W. Spellman,** 2021 Commonwealth Avenue, Saint Paul, assistant professor of surgery and research fellow. Both are on the staff of Dr. Owen H. Wangensten, University surgery chief.

Also named a Markle scholar was **Dr. Robert A. Ulstrom** who left the University's pediatrics department staff last September to become an assistant professor of pediatrics at the University of California school of medicine, Los Angeles.

The Markle grants are being awarded this year to twenty-five faculty members of medical schools in the United States and Canada. Each grant is awarded at the rate of $6,000 annually for five years, according to Russell, "to offer both academic security and financial help to faculty members at the start of their careers in academic medicine. They (the twenty-five) were selected from sixty candidates nominated for the grants by deans of medical schools."

Four other University of Minnesota doctors received the scholarships in previous years. They are **Dr. George E. Moore,** 1949, then instructor in surgery and now director of Roswell Park Memorial Cancer institute at Buffalo, N. Y.; **Dr. Robert A. Good,** 1950, associate professor of pediatrics; **Dr. Leonard F. Peltier,** 1952, assistant professor of orthopedic surgery; and **Dr. William F. Scherer,** 1953, assistant professor of bacteriology.

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BLUE SHIELD-BLUE CROSS NEWS

Of increasing interest is the statistical data compiled annually which shows the utilization of Blue Shield and the number of services furnished Blue Shield subscribers by doctors of medicine. On a comparative basis, each type of service in 1953 increased numerically over the number in 1952 and the dollar payment for each service also showed an increase. Such services as in-hospital medical care, emergency medical care, obstetrical, surgical and ancillary services show a marked increase both in the number of cases in 1953 over 1952, and in the amount paid by Blue Shield for each of such services to doctors of medicine.

During the year 1952, the average payment made to doctors for in-hospital medical care was $22.80 per case. In the year 1953, this amount increased to $24.66 per service while the number of such cases increased 13 per cent over 1952. More remarkable is the increase in emergency medical care payments, and the increase in the number of such cases compared with the year 1952. Payments during the year 1952 for emergency medical care average $31.01 per case. However, in the year, 1953, this payment increased to $38.38 per case or an increase of $7.37 more than the amount paid per case in 1952. In addition, the number of such cases of this nature almost doubled.

Obstetrical services furnished Blue Shield subscribers have always been one of the more frequent of Blue Shield services, and while the cost per case in 1953 was approximately the same as the cost in 1952, the number of such cases increased 8.5 per cent over 1952. The cost per case shows the amount paid by Blue Shield to be greater than the scheduled allowance for a normal delivery, but this is clarified by the fact that included in the figures are the higher benefits for cesarean deliveries. This increased cost of obstetrical cases will further increase in 1954 since the Blue Shield allowance for routine obstetrical deliveries has been raised from $50 to $60.

While surgical services rendered Blue Shield subscribers showed an increase of 14 per cent over the number in 1952, the average service payment in 1953 remained practically the same as that of 1952.

Another classification showing marked increases are those known as ancillary services. These include anesthesia, x-ray and endoscopic services. Each increased over 1952. Anesthesia cases, for example, increased in number 31.6 per cent over 1952 and the cost of anesthesia services was 33 per cent greater than in 1952. X-ray services increased in number and the cost increased correspondingly. A greater increase was noted in the number of endoscopic services which amounted to 26 per cent above that of 1952, and the cost of endoscopic services was slightly greater per case in 1953 than in 1952.

It is apparent from these analyses that in both the number of services and the amounts paid for the different services, Blue Shield service to subscribers and doctors has grown appreciably in 1953 as compared with 1952. With the increase of fees or benefits which became effective for services rendered on and after January 1, 1954, there is a great likelihood that the increases in both volume and costs will be even greater in 1954.

Blue Cross subscribers and their dependents used more hospital care in 1953 than in any year in the history of Minnesota Blue Cross. A total of 167,484 participant subscribers . . . one out of every six . . . were hospitalized and over fifteen million dollars was provided for their care, an increase of approximately 2½ million dollars over 1952.

The record breaking number of hospital cases paid during the year (1953) represented an increase of 14 cases per year per 1,000 contracts protected over the previous year.

This increase, it was reported, occurred in the number of bed-patient cases . . . 144,006 participant subscribers received bed-patient care during 1953 compared with 142,372 participant subscribers in 1952. The average length of hospital stay for bed-patient care during 1953 was 6.5 days compared with 6.2 days in 1952.

Out-patient care during the year was given to 23,478 participant subscribers compared with 24,673 during 1952. This increase in bed-patient care and decrease in out-patient services resulted in an increase in the

(Continued on Page 322)

BOOK REVIEWS

Books listed here become the property of the Ramsey, Hennepin and St. Louis County Medical Libraries when reviewed. Members, however, are urged to write reviews of any or every recent book which may be of interest to physicians.

THE ANATOMY AND SURGERY OF HERNIA.

By Leo M. Zimmerman and Barry J. Anson. 350 pages. Illus. Price $10.00. Baltimore: Williams and Wilkins, 1953.

This new publication dealing with various types of hernia places major emphasis on the inguinal hernia. However, it deals with all types of hernia, and the discussion of each is thoroughly satisfactory. The anatomical section is thorough and meticulously carried out. The section on surgical technique covers diagnosis, preoperative preparation, choice of anaesthetic and detailed description of surgical technique. The authors' ideas in dealing with hernia differ somewhat from the accepted concepts; however, their ideas are good and are well founded. The book loses a great deal of its effectiveness because of needless repetition. The reader is belabored with details of anatomy which are presented adequately in other parts of the book.

The book is a worthwhile addition to the general surgical library.

T. M. Owens, Jr.

PLANNING GUIDE FOR RADIOLOGIC INSTALLATIONS.

By Wendell G. Scott, M.D., chairman of the committee on planning of radiologic installations of the commission on public relations of the American College of Radiology. 336 pages. Illus. Price $8.00. Chicago: The Year Book Publishers, Inc., 1953.

With the marked advances of x-ray in the fields of diagnoses and therapy, the radiologist and his associates have found a place of increased importance in the field of clinical medicine. Slowly, but inevitably, the x-ray department has emerged from its few dusty rooms in the basement to become the very center of the community hospital. Because of the broad use to which x-ray has been put and because of its extensive diagnostic and therapeutic contributions, the department of radiology has become the meeting place for physicians in the hospital; a place for them to congregate and discuss their cases with the radiologist and other colleagues. Planning and innovations in the design of radiologic installations have not kept pace with the rapid growth of this specialty. In certain geographic areas there is evidence of originality of design in the functional arrangement of the departments, but these are few and, only too frequently, the radiologist has had no part in the planning and arrangement of a department of which he is to assume direction.

In this authoritative guide, the viewpoints of radiologists, physicists, architects and hospital administrators have been assembled into one exhaustive manual for the first time. Consideration has been given to every type of installation, from a small office to a large hospital department. Adequate space is devoted to diagrams and ideas for the flow of all activities in the radiology department so as to achieve "the most effective traffic pattern for the radiologist and professional staff so that they may interview, examine and treat patients as quickly and thoroughly as possible." Of marked importance are the data for adequate protection of a radiological installation for the radiation worker as well as anyone working in adjacent areas.

This book is invaluable to those persons responsible for the installation of any type of x-ray equipment, be he a general practitioner, a dentist with a single x-ray unit or the director of a large x-ray department.

J. F.

BIBLIOGRAPHY OF OCCUPATIONAL HEALTH.

Public Health Service Publication No. 300. Washington, D. C.: Government Printing Office, 1954.

A comprehensive bibliography of Public Health Service publications on occupational health subjects issued through 1953 has just been released by the U. S. Public Health Service.

The purpose of this bibliography is to provide a complete and continuing record for reference use. It calls to the attention of the readers, particularly those new to occupational health, voluminous information on early pioneering studies and other contributions by the Public Health Service to the detection and control of occupational diseases. At the same time, by reflecting changes in subject matter from year to year, it affords a historical perspective of shifting interests in the field of occupational health and serves as an index of progress.

While the limited supply lasts, sample copies of Public Health Service Publication No. 300, "Bibliography of Occupational Health," can be made available from this Division upon request to persons and organizations concerned with health problems in industry. Copies may also be procured from the Superintendent of Documents, Government Printing Office, Washington 25, D. C., for thirty-five cents each.

BLUE SHIELD-BLUE CROSS NEWS

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average lengtth of hospital stay for all Blue Cross cases from 5.4 days in 1952 to 5.7 days in 1953.

Every twenty-four hours during 1953 Blue Cross participant subscribers used 2,612.3 days of hospital care compared with 2,472.4 days of care during 1952, 543,493.2 days of hospital care were provided throughout the year 1953 compared with 904,901.6 days of care in 1952, an increase of approximately 50,000 days of hospital care.

Every nine hours during 1953, a Blue Cross participant subscriber incurred a hospital bill totalling $1,000 or over, as compared with one every thirty-nine hours during 1952. During the last quarter of 1953, hospital bills of $1,000 or over were incurred every eight hours.

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PREVENTION AS A GOAL OF INDUSTRIAL HEALTH SERVICES

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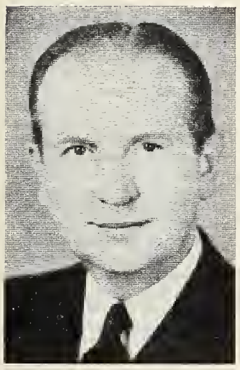

THIS CONFERENCE, it seems to me, represents a kind of partnership that has been a "going concern" for a long time—a partnership of management, labor, the health professions, and government. I would like to take just a few minutes to discuss this relationship.

We used to call this field of health work "industrial hygiene." The new term, "occupational health," characterizes it in a somewhat larger setting, including, nevertheless, the principles of industrial hygiene. The industrial hygiene movement in this country sprang from the initiative of a few progressive business men and the concern of a few physicians, toxicologists, and engineers. From the beginning, its aims were broader than the mere compliance with laws and regulations, often ineffective in bringing about improvements in the health of industrial workers. From the beginning, it was founded on the simple public health principle that "an ounce of prevention is worth a pound of cure."

Prevention—whether of accidents or occupational disease, of communicable or chronic diseases—implies a scientific understanding of causes. Prevention implies a practical means of removing, or minimizing, or controlling the risks of injury, of disease, of disability and death.

The first task of the industrial health movement was to develop the science and skills essential for the prevention of occupational morbidity and mortality. It is in this area of the development of industrial hygiene know-how that the Public Health Service entered partnership with management, labor, and the health professions just about forty years ago.

An Act signed by President William Howard Taft, August 14, 1912, gave the Service its first statutory authority to "study and investigate the diseases of man and conditions influencing the propagation and spread thereof." Prior to that time our research function had been confined to "infectious and contagious diseases." Early in 1913, on the basis of the new law, the Service set up an Office of Industrial Hygiene Investigations in the then Division of Scientific Research. Today, our Division of Occupational Health carries on a similar program of research and development—but in an entirely new environment created by four decades of technological advance and industrial expansion.

The Public Health Service came into the field of industrial hygiene at a time when industry—even large establishments—was spending very little on scientific research into the causes and prevention of occupational diseases and accidents. Then, only two or three State governments had research facilities for this purpose. At the present time, industry is spending many millions for research related to industrial health and safety.

All State governments now have some type of service related to the health and safety of in-

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Address delivered at the Upper Midwest Industrial Health Conference, Minneapolis, Minnesota, October 2, 1953.

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dustrial workers, but these services range from factory inspection concerning safety practices to comprehensive programs incorporating research and development. For the most part, however, State programs are not geared to broad occupational health research and in many instances the staff and facilities for consultant service to management and labor are meager.

Research in occupational health, however, is conducted primarily to elucidate the causes and prevention of conditions about which nothing or little is known. It incorporates basic physical and chemical laboratory studies, engineering studies, medical and epidemiologic studies. It is in this area of research that the Public Health Service has been principally active and in which we have made our major contributions.

During two world wars, the Public Health Service was given the responsibility for supervising industrial hygiene services in certain war industries owned and operated by the Federal government. In this work, we naturally investigated recognized hazards and inspected the establishments to determine whether or not industrial hygiene standards were being maintained. Also, in World War II, we loaned industrial hygienists—physicians and engineers—to the States to help them maintain their industrial hygiene service during a critical period. However, we have never operated industrial hygiene programs in private industry. When we go into an industrial establishment, it is at the request of the industry or a State agency with industry consent; and it is then solely for the purpose of conducting industrial hygiene research.

An important factor that has helped to make our contributions in this field significant is this: The Public Health Service is a "silent partner" in occupational health. That is to say, we have no voice in decisions on industrial health—either of the individual industry, or of the labor union, of the State or local government, or of the medical profession. When we get answers to a broad occupational health problem, they become public property in scientific reports. When we get answers, it is the responsibility of industry to apply the findings if it sees fit. It is the responsibility of industrial physicians and engineers to perform the direct services to men and women on the job. This has been the pattern of Public Health Service activity in occupational health since we entered the field. So I say, we are a silent partner; we have no voice in decisions —unless it be the voice of suggestion and reason.

What has been the fruit of this partnership? It is a well known fact that other industrial nations of the world regard industrial hygiene conditions in the United States and the health status of American workers as superior to their own.

Direct injuries from machinery, foreign bodies and similar hazards have been greatly reduced in most industries. Today, industrial accidents represent far less important causes of absenteeism than they once did. States have emphasized controls in this area usually through the activities of their State labor agencies.

Many of the occupational diseases that formerly took a high toll in certain industries have been reduced continuously over the years. Like many of the communicable diseases in the general population, these occupational diseases are no longer major health problems among workers because preventive measures have been widely applied. Among these one-time "black plagues" of industry, we can cite silicosis, mercurialism, lead and phosphorus poisoning.

The physicians who pioneered in industrial hygiene have seen the concept of industrial medicine broaden toward their original vision of a preventive medical service concerned with the total health of the worker. The labor unions and the individual workers no longer regard such services with fear and resistance, but with confidence and co-operation.

How have we accomplished these improvements? The Secretary of the Department of Health, Education, and Welfare gave us a challenging answer in her recent address to the American Federation of Labor. "These accomplishments have been made possible by co-operation," she said, "by working together. But we know that we still have a long way to go. For our goal goes far beyond the elimination of specific occupational diseases and hazards on the job. It looks toward the day in which the worker's total health—mental and physical, on the job and off it—reaches its maximal potential . . . It is plain that this goal can be reached . . . only by the combined efforts of labor, industry, and government."

Yes, we still have a long way to go. But the goal is worth the effort which all of us must make. It seems to me that here in Minneapolis you are involving yourselves in what we may call a way of the future in occupational health. The concern you have for health services in small firms is shared by business organizations, labor unions, medical associations, and governmental agencies up and down the country. But if that concern is to be productive of results, there must be leadership where the problem arises.

The important factor here is that the Minneapolis Chamber of Commerce and the professional associations of this area have taken the leadership to solve a problem affecting the productive capacity, the health and well-being of one of the Nation's great industrial areas. The important factor here, too, is that State and local governmental agencies are partners in this effort.

It was such multiple partnership that sustained the initial work of Dr. Leonard Arling eight years ago in setting up the Northwest Industrial Clinic to serve industries in the Twin-City Area. From the beginning, business associations and the Minnesota State departments of health and of labor and industry have co-operated to broaden the technical services available through the clinic. And today, the Minneapolis Health Department is co-operating through the work of Dr. W. E. Park.

This is the kind of co-operation that our Secretary called the motivating force in attaining the goals of occupational health. When we think of applying this partnership to the task of stimulating industrial health services in small firms, however, we realize that it is a large order.

The lack of health services for workers in small establishments is a problem throughout the country, and one that is growing and becoming more critical as small business expands. Moreover, it is a problem whether we are thinking of the repair shop or the factory, the construction firm or the bank, the public utility or the department store.

I should be less than candid if I did not tell you that—despite the growing concern, despite the pioneer efforts of management and labor, the medical profession and public health agencies—progress in the provision of occupational health services in small firms is discouragingly slow.

America is a nation of small businesses. About 99 per cent of all establishments reporting taxable wages under the Old-Age and Survivors Insurance program employ fewer than 500 persons. About 70 per cent of all employes in the United States are in these small establishments. Yet, less than 5 per cent of these employes in small firms have any type of in-plant, or on-the-job, health services. This is a conservative estimate based on limited studies by the National Association of Manufacturers, the Brookings Institution, various State agencies, and the Public Health Service.

I cannot, of course, give you comparable estimates for Minnesota and the Twin Cities area. However, it is well known that industry here follows the typical American pattern of small business. It seems to me, therefore, that you have in the Twin Cities an unusually good opportunity to demonstrate to the nation as a whole what can be accomplished in the development of constructive, economical industrial health services for small firms.

Is there a need for such services? Here again, national estimates of need are not as precise as we would like to have them. *The National Safety News* for October, 1951, cites figures, however, indicating that accidents occur in small plants about two and a half times as frequently as in medium and large size plants.

The data on sickness absenteeism are not available according to size of plant. The estimated total loss due to temporary disability from all causes is between 400 million and 500 million man-days annually. This does not include the cost to the nation of permanent disability among Americans of working age.

It would be difficult to relate the national estimate of 400 million lost man-days due to accidents and disease to the worker population of the Twin Cities. You get some idea of the magnitude of the local cost, however, by applying the national average of ten days per worker to the Twin Cities' 460,000 or more workers. For Minneapolis and Saint Paul, this means an annual loss of between 4,000,000 and 5,000,000 man-days.

Even if we assume that these losses in productivity, in purchasing power, in expenses for medical and hospital care, and in personal health, occur with equal frequency in small and large business establishments, the distribution of employment tells us that the major portion of the nation's economic loss—the Twin Cities' economic loss—occurs in small businesses.

What can we do about it? Let me say here and now that we do not know all the answers by any means. But it can be said that there have been various experiments here and there, by public and private organizations singly or in co-operation, which begin to suggest some approaches which may lead to the right answers.

In the first place, there is fairly widespread agreement on the objectives of industrial health programs. I found these objectives admirably summarized by Dr. Dale C. Cameron of our Division of Occupational Health.[1] They are:

> "1. The assessment of a worker's physical and psychological assets and liabilities in order to facilitate the selection and proper placement of employes.
>
> "2. The prevention of occupational illnesses and the detection of nonoccupational illnesses in an early stage.
>
> "3. The provision of treatment, the type and extent of which must depend on matters of policy, both local and broad.
>
> "4. The fostering of those physical, mental, and social habits and attitudes which are conducive to both productivity at work and the enjoyment of life."

These are laudable goals, but the practical means of attaining them are by no means as clearly defined as would be desirable. What about costs, for example? Does the program really pay off? Are there supplementary community health services that could be drawn upon to help achieve the goals of occupational health?

A study completed by the Public Health Service in 1952[3] brings together much of the available information on services in small businesses across the country. The striking fact revealed by this study is that there are no reliable cost estimates that could be widely used as guides to the development of such services in individual plants or areas now lacking them.

Obviously, costs are related to the size of the group receiving the service, to the nature and scope of the services, the method of providing the services, and the specific occupational health problems within the establishment. There is, however, an urgent need for well-planned cost-accounting studies that would provide practical guidelines for planning services of varying volume, scope, and content.

The same is true when we are faced with the question: Do industrial health services in small plants pay off? Here again, it is very difficult to get figures that provide clear-cut and reliable answers. In general, the difficulty stems from the fact that small firms seldom keep absenteeism records upon which to base estimates. Also, the majority of small-firm industrial health programs have been in operation for only a few years. In health work, it is necessary to follow the results of a program for a considerable time before a trend can be established. The human organism does not respond to the "play of the market" as quickly as does the sale of products. So, although one's sales chart may reveal day-by-day changes, the curve of sickness absenteeism, compensation costs, and so on will be slower.

In this area of need for sound cost-accounting and statistical studies related to occupational health services, I see an opportunity for American business to make an outstanding contribution to national health. If any institution in our national life has the special know-how, the personnel and facilities to do this job, it is American business. Government cannot and should not do it. Medical organizations cannot do it alone, but they can assist.

Business needs the co-operation of the medical and health professions in developing the kind of data requisite to studies related to occupational health, both in its medical and environmental aspects. In my opinion, we would see much more rapid progress in occupational health if business would more generally apply its special competence to the study of the economic and organizational aspects of occupational health.

The experience of individual businessmen in some small plant programs indicates the catalyzing value of management in occupational health. Their experience also shows that we can get some objective measurements of costs and results.

Mr. Ross Fraser, president of the Rome Cable Company in New York, for example, instituted a complete medical department in his medium sized plant in 1948. He has reported as follows on results in the three-year period, 1948-51: "In 1947 . . . the total man-days lost for all reasons exceeded 9,000. In the first half of 1948, this rate continued. In 1950, compared to the 1947 level, total absences have dropped by almost 50 per cent, with 10 to 15 per cent drop in man-days lost due to sickness. In 1947, we had almost 3,600 man-days lost on which we had no report as to reason, and many of which could probably have been classed as sickness. In 1950, the number (of unaccounted man-days lost) was less than one-quarter of that figure. Man-days lost for occupational injuries were cut to less than one-third of the 1947 level."[2] Mr. Fraser also cited sharp reductions in sick and accident claims and a 43 per cent drop in the cost of compensation insurance.

In a seven-year period, Mr. Ellsworth S. Grant, vice president of industrial relations, of the Allen Manufacturing Company, Hartford, Connecticut, has reported a total reduction in absenteeism from 5.8 per cent to 1.8 per cent. Compensation premium rates were reduced in five years by 24 per cent, despite changes in the Connecticut Workmen's Compensation Act which would normally have led to higher costs. The Allen Company and several other small firms with in-plant health and safety programs have hung up records of more than 2,000,000 man-hours without a single lost-time accident.

These two examples of business experience with small plant health services undoubtedly could be duplicated many times—in this area as well as in other parts of the country. There seems to be no question as to the value of such services, even when objective measures of results are not available.

Management is concerned for the "intangible" as well as the tangible results. Quite rightly, businessmen are concerned with the practical problems of cost and measurable returns. But it is most heartening to hear the repeated assertion that: "Our employes like the service," or that "they are enthusiastic," or "feel that the physicians and nurses have a real interest in their health." One official reports: "The company's health and safety programs, together with group insurance, form a keystone of employe relations; these have contributed substantially to maintaining a high degree of both employe satisfaction and day-to-day employe efficiency."

In this great industrial area, as in others, the Chamber of Commerce, in co-operation with state and local medical societies, health agencies, medical schools, and other institutions, is making concerted efforts to stimulate occupational health services in small firms. It is, as I have said, a large order—and often an uphill battle. But, just as business can and should undertake the practical study of costs, so also the small firms can and should apply their skills in co-operative planning and operation to determine the most effective ways to organize the kind of occupational health service they want. Just as small firms throughout the country have learned the advantages of pooling for the procurement of basic raw materials, so joint planning and agreements might go far to extend the "procurement" of effective health services. I believe that health conferences such as this can provide the essential leadership for such planning and action.

A year or two ago, Dr. Ordway Tead, editor and director of Harper Brothers, came to Washington to conduct a seminar on executive leadership for a group of Public Health Service administrators. I would like to pass on to you his definition of the role of leaders: "Leaders are trying always to influence people, to get something done which needs doing, because the people have come to agree that it's worth doing and that they want to play ball, so to say."

That is a statement in plain terms. It strikes home. But it is deceptively simple, as you will realize when you think it over. For, as Dr. Tead went on to say, leaders are successful in getting the job done to the extent that they are able to involve all the skills and the loyalties and enthusiasms of the folks right down the line. In the promotion of occupational health, this means that the individual workers and their labor organizations, as well as top management and supervisory personnel must be involved in the plans and operation of the program.

Dr. Tead, like many other thoughtful leaders in industry, believes that it is one of the foremost challenges to our society to involve all the folks right down the line in the pursuit of constructive goals. In business, there are various names for this process: industrial relations, employe relations, executive leadership, and so on. Basically, it amounts to the application of sound psychological principles to personal relationships. It seems to me a sign of the times that this conference has stressed this element through the contributions of Mr. Morse, Dr. Hastings, Dr. Stone, and others. For like management and labor, the fields of medicine and public health have come to realize that good personal relationships are fundamental to individual health, happiness, and productivity.

Dr. Cameron, whom I quoted earlier, has estimated on the basis of limited studies in this country and in England, that about 30 per cent

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HEALTH SERVICE IN INDUSTRY—ROCK FOUNDATIONS

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ANY BUILDING which is designed to carry a heavy weight of superstructure must have a firm foundation. There are five principal points where support must be sure, at the four corners and in the center.

In the industrial world there must be firm reasons for doing things, and foundations must be well laid. I shall mention five of the rock bottom supports upon which a stable health service may be built. Every one is essential. If any one of the five is missing, the project will not be completely successful and the structure may collapse altogether.

Management Support

First, we must have a progressive plant manager, who is completely sold on the project. He must believe that the health service is a sound business venture. In this respect he need not be in any doubt, for reports from many sources have proven the dollar value over and over again.

One of the first of these value surveys was conducted by Dr. Victor G. Heiser for the National Association of Manufacturers and reported by them in 1941.[1] In this survey all but five of 1,625 companies considered their medical programs paying propositions. The 1951 survey of the same Association gives a similar favorable report.

In 1952, Dr. Kenneth Peacock, in speaking at the Fall Personnel Conference of the American Management Association in New York City, gave before and after figures indicating significant universal profits averaging from $3.50 to $5.00 for every $1.00 spent on Medical Departments. These profits were brought about by such factors as 50 per cent reduction in compensation costs, 30 to 50 per cent reduction in turnover, 30 to 40 per cent reduction in absenteeism, 45 per cent reduction in accident frequency and 63 per cent reduction in occupational diseases.[8]

Because it pays for itself is not the only reason why management needs to give wholehearted support to the health service. There are other less tangible but equally important benefits—the impact on industrial relations;[5] the improvement in morale, company pride and loyalty; the conversion of better general health into better home and community living; to say nothing of the personal satisfaction to an employer.

It is not enough, however, for the plant manager to make funds available for development of a health service. He must be personally interested in its progress and accomplishments. He must give encouragement and inspiration, and be readily accessible, and responsive, when problems arise. He should give the medical director responsibility but not withhold the stimulus of periodic inquiry. The plant manager should acknowledge the superior judgment of the medical director in medical matters but be a tower of support to him in the area of administration.

A Place for "In-plant" Service

The second essential for a firm foundation is a suitable place to work. Every industrialist realizes the advantage of proper space, arrangement of machines and channeling of materials for production purposes, but few are competent to design a health service. Even so, how frequently the doctor or nurse is hired after the rooms have been built. I do not hesitate to say that every health service unit should be designed in consultation with an industrial physician or better still with the doctor who is going to work there. Even before the physical facilities are designed, the extent and limitations of the service should be established. Then the unit can be built to carry out those functions with due regard for later expansion of the service.

We are thinking, of course, of an "in-plant" service. That is, one in which the doctor goes to the plant on a part-time or full-time basis and does most of his industrial work there. In such a setup the physician is paid an adequate retainer fee, which is based on the time he needs to spend at the plant. He should receive no

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Dr. Park is Industrial Health Physician, Minneapolis Health Department.

additional fee, per patient, which would tend to place him on a piecework basis.

I know this is contrary to the thinking of many part-time physicians or doctors on call, who think of industrial medicine as a supplement to their private practices. However, I believe the "in-plant" medical service principle is one of the basic foundations upon which genuine industrial medicine will be built. Is it not time we in the medical profession thought of our services as an essential element of industrial production and, therefore, subject to streamlining for that purpose? It is acknowledged that industry should pay the doctor a reasonable fee or salary for making a careful physical examination of an employe, but industry should not be saddled with the additional expense associated with sending an employe to a doctor's busy downtown office during the plant's working hours. The consequent disruption of production, coupled with the payment of unearned wages is far more costly than the doctor's fee.

I shall not attempt to describe a suitable "in-plant" work place. Suffice it to say, it must be large enough, with proper placement of rooms, quiet enough and private enough to serve its purpose.

Competent Staff

The third foundation stone of an industrial health service is a proper staff. This means a doctor and nurse specially trained and competent to do the job. It also means enough nurses and enough doctor's time to accomplish the purposes and services being undertaken. It should be remembered that the more complete the service the more effective it is. Also as effectiveness is increased, the cost in terms of results goes down.

Training in occupational medicine and industrial nursing is now available as postgraduate courses in some universities. Applicants holding such university degrees have a tremendous advantage over ordinary medical and nursing school graduates. But more than academic training is needed. There is no substitute for experience in a properly conceived and well-operated health service.

One of the musts for a good health service is proper medical supervision. A fundamental concept of a nurse's training is to make her a part of a specialized team, not an individualist. Why then should we expect a nurse to work alone in industry? Every industrialist should realize that it is false economy to hire a nurse and not at least a part-time physician to work with her. And I don't mean a "doctor on call" arrangement.[3] By a "doctor on call" we mean a physician who has consented to respond in case of emergency and advise the nurse when called upon. He, of course, does not get paid for supervising the nurse. The doctor on call has no authority and little influence at the plant. He is not a part of the in-plant team. Only indirectly does he contribute anything to the preventive health program, for instance, through preplacement physical examinations.

The treatment of patients is an important and necessary function of industrial medicine which the "doctor on call" may do very well, but it should be realized that he, like other outside practitioners, stands in the salvage department and not in the production line where the industrial physician belongs.

One thing of paramount importance in an industrial physician or nurse is interest in the work. You can't make an industrial nurse out of a girl who takes a job only to carry her through a few months while she is waiting to get married. The doctor or nurse who will make a success of the industrial health field is the one who sees in it everything he wants in a full and satisfying career.

A Planned Program

The fourth necessary support for a successful industrial health service is a carefully planned program. It is not enough to hire a nurse, give her a first aid station, and tell her to go to work. If she is a nurse, right out of a training school, she won't know what to do. If you are the average employer or personnel director, you won't know what she should do either.

Planning the scope and limitations of a program calls for many hours of discussion between health service staff and other plant personnel including labor representatives. Out of these discussions will grow the medical service procedures and company policies which will govern the service. The medical director should crystalize these procedures and policies by writing them out so that they may become a part of the plant manual.[6]

Of course, the medical service is going to render first aid to the injured. That may be all management wants to undertake. If so, that is a major crippling decision. Under such a policy a nurse may make good window dressing but she is by no means necessary, because someone trained in first aid can do the job just as well. To put a nurse in such a straight jacket is to crush all her enthusiasm for industrial nursing.

A decision should be made as to where to draw the line in the treatment of non-occupational conditions. An industrial health service should not operate in competition with private practicing physicians. Nevertheless, one of the most important factors in absenteeism is non-occupational illness and no industry can afford to ignore it entirely.[4] The usual position taken, is, that the plant physician or nurse will provide dressings and treatments for non-occupational conditions of a minor nature which would not compel the patient to visit his own physician anyway. Such minor service, when available without cost at the plant, many times brings the workman to work when he would otherwise stay home. Another feature of advantage to both workmen and doctors is the early detection of more serious disease which is promptly referred to the private physician for treatment.

The purpose of physical examinations must be clarified. It should be thoroughly understood that the original physical examination will be used to determine the physical capacities of the applicants, with a view to proper placement, and not as a means of rejecting the handicapped or less fit.

How frequently should repeat physical examinations be performed? How much information will be made available to the workman's private physician? What records are necessary and where shall they be kept? How can medical confidences be maintained? How will the health service function in relation to the safety and personnel departments? How vigorously will the doctor and nurse push preventive activities and health education? Shall there be an attempt to enter into medical research or community health and recreational activities? These and many other questions enter into the planning of a health service.

One of the most useful and far-reaching functions of an industrial nurse is health counselling. But not every industrial nurse can do health counselling. It is a capacity which develops with experience and increasing maturity. It is a function which cannot be practiced at the outset but which the nurse may grow into as she becomes accepted and earns the confidence of the employes.

Planning the functions and scope of the health service, and limiting the areas of activity where necessary, is not easy, but it is an important foundation upon which to build. This is the cornerstone which adapts and individualizes the health service to the particular needs of an individual plant.

Proper Attitude

The fifth basic essential of a health service is a proper attitude. When I speak of attitude I mean the attitude of the health service, its medical director, its nursing staff, and behind that the attitude of the plant management.

The foremost proper attitude is the realization that the health service is there primarily for the benefit of the employe. It is his health that is first concern, and this should be a very personal concern. The workman who has acquired skill in the performance of his work is the company's most valuable asset and should be treated as such. Ralph M. Monk, Vice President of the Caterpillar Tractor Co., puts it this way: "The purpose of medical services in industry is not to make money but to make health. Any financial advantage is essentially a by-product and must always be recognized as such."[2]

When the health of the employe is placed first, it is only natural that the plant physician will discuss his findings with the employe and advise him to see his own private physician for treatment of correctable conditions. The employe on his part when he knows that the health service is for his benefit will feel free to use it for advice on medical matters or related problems which require a medical opinion.

It is easy to appreciate the reluctance of a workman to consult the plant physician if he thinks management will be informed of his impairments and his job security thereby threatened. This is a misconception which must be met and eliminated by honest and fair practices.

The relationship of the plant physician to his fellow private practitioners should be most cordial.[7] The plant physician should be constantly working closely with them. He should refer non-occupational conditions to the private physician,

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CARE OF HAND INJURIES

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"Next to the brain, the hand is the greatest asset to man and to it is due the development of man's handiwork."

IF ONE pauses and considers that quotation for a moment, he realizes the significance and the importance of the preservation or the restoration of the function of an injured hand.

Now, there are many types of injuries to the hand—from the burns, through the lacerations, the amputations, the crushing or squeezing injuries with or without laceration, avulsion or fractures, and finally the complicated compound injuries with lacerations involving tendons, nerves and compound fractures. Inasmuch as it would be impossible to discuss intelligently the care of all types of hand injuries in the alloted time, I am going to limit myself to the most common of all injuries, namely, the laceration, and the most common site, the palm or flexor surface. It is here where what appears to be a minor injury too often proves to be one of a major nature and there develops a loss in the function of a finger or fingers as a result of its improper recognition or handling.

The principal factor which determines the success or failure in the restoration of function of an injured hand is the degree and amount of scarring or fibrosis which results. The greater the degree of scarring and fibrosis, the more interference results in the delicate functioning mechanism of the hand and fingers, namely, the gliding of the tendons through their sheaths or tunnels, the strength of the intrinsic muscle, the flexibility of the joints or the elasticity of the joint capsules. The prevention, to as great a degree as possible, of the development of this fibrosis is one principal which is common to the care of all hand injuries. The extent or degree of development of the scarring and fibrosis is dependent upon many factors from the time of injury through the final stages of rehabilitation. The majority of these factors can be controlled by those involved in the care of the injured hand.

The amount of fibrosis development is dependent upon: first, the extent and severity of the injury; second, the degree of contamination; third, the degree of inflammatory reaction or actual infection which develops, fourth, the proper definitive surgical repair, and fifth, the proper postoperative care and rehabilitation. Obviously, the first two factors cannot be controlled except by safety measures which might prevent the more extensive injuries. The next three factors can be controlled, and their control involves all persons caring for the injured hand, from those giving first aid down through the surgeon and physical therapist.

Once the injury has occurred, the steps in the prevention of fibrosis begins, and it begins in the factory, the office, the store or home.

We will follow through the steps in the care of a laceration in the palm of the hand or finger. The first one to be involved usually will be a fellow worker or someone else in the home. The first step is to prevent further contamination of the wound. The wound is already contaminated by the cutting instrument and the dirt and bacteria on the hand. A dirty handkerchief from a pocket or some rags off a bench used in an attempt to control the bleeding or prevent blood from soiling one's clothing, the floor or rug, further contaminate the wound. Though lacerations of the hand bleed freely, they usually will not bleed to any serious extent until proper first aid can be reached in the plant or home. Let them bleed and let Nature do a little washing of its own until a proper first aid kit is brought to the patient, or the patient is brought to the first aid room. If one is near a water faucet, he should let the water run on the cut and wash away the blood from the hand. All plants or shops or offices which do not have medical departments or first aid rooms should have first aid kits. These kits should be large enough to have sufficient dressings, bandages and tape more than just three 2 x 2 inch squares of gauze, fifteen bandaids and a bottle of iodine or 2 per cent mercurochrome.

Let us assume that the patient is going to be attended by the company nurse, the first aid man or a fellow worker. The attendant may gain some information of the seriousness of the cut by

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asking the patient to move all fingers, to flex them and extend them completely, and by asking whether any of the fingers are numb. If the finger or fingers cannot be flexed or extended freely in all their joints by active voluntary movements of the patient within what limitations pain or soreness might cause, or if a finger, a part of a finger or several fingers feel numb or asleep to the patient, then we are dealing with a case which requires more definitive treatment than cleansing and bandaging at the plant by the first aid man, nurse or plant physician. Plans should be made for him to be sent to the company's surgeon or preferably his hospital. The first aid care at the plant or home for such a laceration should consist of those things which will lessen the possibility of scarring or fibrosis by preventing further contamination, the development of infection, and the adding of any further trauma, either chemical by irritating antiseptics, or mechanical by injudicious washing, scrubbing, probing or examining the wound. The wound should be covered by sterile dressing and sufficient dressing to absorb the bleeding and then wrapped with bandages snuggly, but not tightly. Usually, a good heavy dressing snuggly applied will control most hand or finger bleeding for the time being, until the patient can have more definite care by the surgeon.

I have not mentioned the use of an antiseptic, and for a purpose. Most antiseptics are fine for killing bacteria, but some of them are irritating to the tissues as well and will produce a tissue response that will increase the development of fibrosis. Especially, time-honored iodine, which even today is frequently dumped or poured on a laceration of a finger or hand, falls into this class. Therefore, if an antiseptic is to be used at all in these more serious lacerations, let it be applied to the finger or hand about the laceration, but not into it. Iodine should *not* be one of the antiseptics in the first aid kit.

The use of a tourniquet has not been mentioned, again with a purpose. A tourniquet is rarely, if ever, needed in lacerations of the hand in spite of what at first appears to be severe bleeding. In the majority of instances the tourniquet is not applied properly, so that usually the veins are shut off and prevent the return flow of the blood and the arteries are not completely shut off so that blood continues to be pumped into the hand or finger, and congestion and swelling develops with damaging of tissues which leads to further fibrosis. As long as we are talking about hands and not arms or legs, the tourniquet is not of special interest.

Now, with the first aid care completed, the hand or finger dressed with sterile and sufficient gauze and bandaged snuggly, the next step is to get the patient to the hospital without delay. An ambulance, siren, police escort or breaking of traffic laws are not necessary, but there should not be unnecessary delay as time is also a factor in our struggle to lessen the development of fibrosis. The longer the contaminants introduced into the wound are allowed to remain, the greater will be the chance of inflammatory reaction or actual infection in spite of our thorough cleansing, debridement and antibiotics.

On arrival at the hospital, the responsibility falls on the medical professional personnel, the emergency room nurse, the intern or resident, and the surgeon. Usually, much information can be gained by questioning the patient and having him move his fingers, if they are exposed, or exposing a part of them to be moved or tested for sensation without disturbing the dressing over the wound itself. If one is still not satisfied and wants to inspect the wound, the inspecting should be done with a sterile aseptic technique similar to that used in the operating room. A mask should be worn by the nurse or doctor. Sterile rubber gloves should be worn and the hand placed on a sterile towel or sheet as the dressing is opened. A sterile instrument should be used in inspecting the wound. One should avoid the excessive sponging of the wound in inspection and should attempt to control the bleeding by pressure proximal to the wound. Probing for tendon ends should not be done. Once it has been determined that tendon, nerves or other important structures have been damaged, the hand should again be dressed and bandaged and preparation made for the definitive surgical care in the operating room. If x-rays are thought advisable for foreign bodies or bony injuries they may be done through the bandaged hand.

It is now the surgeon's responsibility to see that there is no unnecessary delay in getting at the definitive surgery as the contaminants are still present in the wound. These are emergency cases and cannot be put off for hours or until the next morning. Many men feel that definite reparative surgery should not be carried out on the finger after the injury is two hours old or in the hand after four hours. It is also his responsibility to see that the factors, which within his realm, lead to fibrosis are prevented or limited to the greatest degree possible. These factors are infection, additional surgical trauma, injury to or destruction of important structures, chemical irritants which increase tissue reaction, and the too early active movements of healing tissues. His control of the infection factor comes with the thorough cleansing and preparation of the hand and an efficient and thorough debridement. This thorough cleansing and preparation of the hand should be done under a tourniquet and an anesthetic, except some of the smaller lacerations of the fingers which are not too painful or bleeding too severely. I prefer a general anesthetic, even if it entails a stomach lavage in those persons who have recently eaten or drunk. Some men prefer a nerve block. The safest tourniquet is the pneumatic tourniquet or the simple blood pressure cuff. The arm should be elevated for about three minutes before the tourniquet is applied to empty as much as possible the veins of the hand. The cleansing or preparation of the hand should be done by a thorough washing of the hand and that part of the forearm necessary in the preparation, with soap and water, or as I now prefer, pH isoderm with G-11 or so-called pH isohex. Grease or oil may be first removed with ether. Then the hand and wound should be washed with pH isohex and water until as clean as possible without scrubbing or rubbing. This may take a considerable period of time but it is as important as the surgical repair which is to follow. In the cleansing, anything which would add additional mechanical trauma should be avoided as this will cause additional tissue reaction and eventually added fibrosis. For this reason, scrubbing with brushes or rubbing vigorously with coarse gauze should be avoided. The cleansing should be dependent upon the emulsifying action of the soap or detergent and the copious washing with water. What contaminants cannot be washed away can be removed in the debridement. After the cleansing, the hand may be prepared with an antiseptic but if an irritant or alcoholic antiseptic is used it should not be introduced into the wound itself. The antiseptic of choice after a hand has been prepared with pH isohex is aqueous solution of Zephiran or Phemoral. These do not interfere with the antiseptic effect of the pH isohex and can be introduced into the wound without an irritant effect.

Debridement of the wound again involves principles which tend to lessen the development of fibrosis. All contaminating material and devitalized tissue should be removed with the preservation of all viable tissue and the avoidance of injury to the important structures of the hand, and done with the least amount of surgical trauma. This is accomplished by sharp, careful dissection and a thorough knowledge of the anatomy of the hand with ability to recognize its vital structures, especially when under tourniquet.

The next responsibility of the surgeon is surgical repair. This consists of two things: choice of surgery and technical repair. The surgeon must decide which procedure will result in the least tissue reaction in healing and thus the least fibrosis and chances of adhesion or adherence. These procedures are the primary repair of the injured structures or the simple closure of the wound with a secondary repair at a later date. This is dependent upon the type of trauma, the loss of tissue or structures and the location of the trauma. Usually, the more severe the trauma of the injury or the greater the loss of tissue, the greater will be the reaction in healing and the likelihood of scarring and adherence of the primarily repaired structures whose gliding mechanism is all-important. Nerves may be, and should be, repaired primarily, and muscle bellies may be repaired. It is my feeling that tendons severed in the fingers are best repaired secondarily either by simple repair or tendon graft. Those in the hand may be repaired primarily or secondarily depending upon the extent of trauma and damage. Those in the wrist or above can be repaired primarily. When both the profundus and superficial flexor tendons are severed in the palm of the hand or at the base of the fingers the removal of the sublimus with repair of the profundus is preferred whether done primarily or secondarily, especially where the severance is at the same site or level.

In the technical repair of the injured hand the term atraumatic technique has come into use. This implies the careful and delicate handling of the tissues, the use of proper incision and exposures and the use of proper instruments and sutures. Careful and delicate handling of the tissue needs little explanation. It implies the avoidance of unnecessary sponging or rubbing of the tissues, injudicious clamping, spreading of the tissues or continued probing for tendon ends. Incisions for exposure should parallel the flexion creases in the hand or fingers or be in the creases themselves. They should not be made to cross the flexion creases in the palm nor be in the midline of the fingers. These incisions may be made as extensions from the lacerations or be made, as oftentimes is advisable, as separate incisions away from the laceration itself. Frequently, there is less scar adhesions if the original laceration is closed primarily and the injured tendon exposed by an incision at a distance away from the tendon and by the raising of skin and subcutaneous tissue flaps. For exposure in the fingers themselves incisions should be made along the mid-lateral aspects. This position may be found by flexing the finger and placing a dot at the dorsal end of the flexion creases. On straightening the finger a longitudinal incision through these dots will be properly located and be midway between the palmar and dorsal digital vessels and nerve branches. Tendon sheaths and annular ligaments should be opened in this area along their lateral aspects close to the bone. Flaps on the fingers may be made by incisions in the flexion creases connected with the midlateral incisions.

The instruments used should fit the operation. They should be small, delicate and atraumatic instruments which will not crush or damage more tissue than is necessary. Mosquito forceps should be used for clamping; small delicate tissue forceps, small fine-pointed curved scissors with sharp and good cutting blades, small blunt or rake retractor and small sharp scalpel blades should be used.

The suture material should be that which will produce the least amount of tissue or foreign body reaction. It should be as small or fine as is consistant with what needs to be accomplished and minimum in amount. Silk, cotton, linen or wire may be used. My own preference for ligatures is silk about 4-0 to 6-0, and for the repair the technique of Bunnell with the use of fine stainless steel wire and pullout wire and approximating the tendon ends with two or three blood vessel silk right angle sutures. This technique may be used for lacerated tendons in the palm or, where I especially like it, within the tunnels of the fingers where Bunnell's technique of "suture at a distance" leaves a minimum of foreign material at the approximated tendon ends. Silk, cotton or linen may be used for the repair of the tendon but the maintaining of the repair at the approximation should not be dependent upon the strength, size or amount of material but upon the relief of tension by proper position, splinting and immobilization until sufficient healing strength has occurred. If these materials are used, they should be fine (about 6-0) and not crossed back and forth within the tendon, squeezing and devitalizing healthy tendon tissue. Each case with its individual problem dictates the suture or technique to be used. Usually, size 34 wire will suffice for finger tendons, with a slightly larger wire for the pull-out wire. I also prefer the use of wire for closure of tendon tunnels in the finger, using through-and-through mattress wire sutures tied over buttons on the dorsal aspect of the finger.

The repair, like the preparation and debridement, should be done under a tourniquet which is released just before closure so that the larger bleeding vessels may be identified and ligated. The tourniquet is then reapplied until after final closure and dressing. All severed nerves should be sought for and repaired in either the hand or fingers. After closure a sufficiently heavy dessing with snug even pressure should be applied, and the forearm, hand and fingers splinted, immobilizing the fingers and hand in the position of function. This position is similar to one the hand and fingers would assume if a baseball were held in the palm of the hand and the fingers grasped about it and with the wrist slightly dorsiflexed. The hand and forearm should be well elevated postoperatively and maintained that way for several days to reduce congestion to a minimum.

The surgeon's final responsibility is in the postoperative care and in the rehabilitation, and in this he is helped by the physical therapist and also the employer. The use of antibotics, tetanus antitoxin or toxoid is recommended postoperatively, but should never be relied upon to prevent or control infection as a substitute for the careful prevention of contamination, the thorough cleansing and debridement and meticulous aseptic surgical technique. The excellent work of M. L. Mason and H. S. Allen has shown us that in the healing of a repaired tendon the greatest strength and the least amount of fibrosis or scarring is attained when complete immobilization of the tendon is carried out for a period of three weeks. They have shown that in the healing of a tendon three tissues are involved; namely, the tendon itself, the connective tissue in and on the surface of the tendon and the connective tissue surrounding the tendon. This healing begins in the connective tissue in and about the tendon ends with an exudative reaction and fibroblast formation to form a cuff about the tendon ends which is an actual enlargement or swelling. This is especially significant when repairs have been made within tunnels or tendon sheaths where the snugness produced by this swelling or cuff may interfere with the circulation of the tendon or lead to adherence of the tendon to the sheath or tunnel.

Mason and Allen have shown that the healing process passes through three stages of tissue reaction and three phases of tensile strength. The stages of tissue reaction are those of exudation and fibroblast formation, regeneration, and finally maturation which occurs between the nineteenth and twenty-first day. The tensile strength at the approximation diminishes rapidly, becoming its weakest at about the fifth postoperative day. There is then a gradual increase up to the sixteenth day, when it levels off into a plateau until the nineteenth day when it again passes into a phase of increasing strength. Mason and Allen have shown attempted movement or function of the tendon between the fifth and fourteenth day is likely to lead to separation, as well as increased tissue reaction. Also, function or active movement of the tendon after the fourteenth day will not only increase tensile strength but also tissue reaction. After the nineteenth to the twenty-first day, function will further increase tensile strength but with much less tissue reaction, as maturation has taken place, swelling has started to subside and connective tissue elements loosen.

Complete immobilization should be carried out for three weeks. At this time, guarded active movements are begun, with hot soaking of the hand and wearing of the splint at night. At the end of four weeks, active physical therapy is begun and the splint completely discarded. Here the physical therapist takes over. He must guard against too vigorous active and passive movements and massage which will produce swelling of the finger or joints with pain and soreness, and which will retard rather than hasten the return of the functional use of the hand. Finally, the employer may play his part in the rehabilitation of this person by co-operatinng in return of the patient to some form of work of which he is capable. This should be done both for the psychological effect and the fact that there is no better physical therapist than Mother Nature herself in the functional use of the hand.

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PREVENTION AS A GOAL OF INDUSTRIAL HEALTH SERVICES

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of sickness absenteeism is due to emotional disorders. We cannot ascribe a definite proportion of this loss of efficiency to interpersonal relationships in the individual's place of employment. There is good evidence, however, that the impact of modern industrial employment, plus the tensions created by personality conflicts on the job, contributes significantly to emotional and physical disorders among employes. A well-planned medical service, integrated with a safety program and good personnel practices, can do much to ease these tensions and to lessen the impact of the industrial environment.

Sixty-three million people—more than two-fifths of the American population—are engaged in earning a living. When we consider these figures, together with those related to absenteeism, compensation costs, and wage losses due to accidents and disease, we cannot but be impressed with the opportunity which employers and employes, physicians and health agencies, have to promote occupational health—especially in small firms. In so doing, they can make a tremendous contribution to the nation's productive capacity, as well as to its health and happiness.

References

1. Cameron, Dale C.: Human Relations in Occupational Health. Pub. Health Rep., 67:7; 686-689 (July) 1952.
2. Fraser, A. D. Ross: Background and development of an industrial medical program. Tr. 16th Annual Meeting, Industrial Hygiene Foundation of America, Inc. Bull., 19:69-80, 1951.
3. Klem, Margaret C., and McKiever, Margaret F.: Small Plant Health and Medical Programs. PHS 215, Washington, 1952.

DIESEL ENGINE DERMATITIS CONTROL

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IN DISCUSSING the problems of dermatitis in industry, I believe it is well first to determine its relative position as a producer of disability to work. Perhaps a few statistics might serve to clarify this. In New York in 1950, 1½ per cent of the 112,000 cases compensated under the compensation laws of that state were dermatitis cases, and the average compensation paid was $281.00. In the same year in Illinois, less than 1 per cent of the more than 50,000 cases compensated were dermatitis cases, and the average compensation paid in each case was $147.00. The Industrial Commission of the State of Minnesota has advised me that for the years 1948, 1950, and 1952, dermatitis cases represented 2 per cent of the total compensation load.

Thus we see that in our two largest industrial states and in our own state, dermatitis is a relatively minor contributor to the total industrial disability picture. It is, however, the most disabling industrial disease, accounting for 60 per cent of the time lost from work on account of disease.

Though it is difficult to secure comprehensive figures that cover the year by year incident of industrial dermatitis, studies that have been made seem to indicate that larger industries have improved their ability to deal with this problem, with a corresponding decreasing incident, whereas in smaller industries, the incident is somewhat on the increase.

With respect to the railroad industry, from all statistics that are available, it would appear that prior to the advent of the diesel locomotive the incident of occupational dermatitis was so low as to be a negligible factor, and it was not until the diesel locomotive came into extensive use that it assumed any proportions. However, with those railroads that went into an extensive dieselization program during and immediately following the war, the number of occupational dermatitis cases climbed quite markedly. This increase was due, of course, to the tremendous increase in the use of petroleum products and derivatives incidental to the operation and maintenance of the diesel locomotive.

In co-operation with the Association of American Railroads, the petroleum producers, and the manufacturers of cleaners and solvents, the railroad industry generally launched a concerted drive to overcome and, if possible, to eliminate this problem.

At the outset of our studies certain fundamental facts became evident. These in the order of their relative importance might be listed as follows:

It became readily apparent that the petroleum products and their derivatives used to fuel and lubricate diesel locomotives, the chromate compounds used in the cooling systems, and the carbon tetrachloride solutions used in the cleaning of certain parts were all basic irritants, which would produce skin irritation in most anyone exposed to their use for any great period of time, unless proper methods of protection and personal hygiene were followed.

From the information gathered, a planned program was established and carried out. First—we attempted to eliminate from use as many as possible of the irritants; second—we undertook to provide protection in all of our diesel facilities against those irritants we could not eliminate; third—we undertook to improve the washing facilities and to provide proper cleansing agents at all facilities, and, fourth, and most important, we undertook a program of educating our employes to the practice of proper personal hygiene.

It was, of course, impossible to eliminate the use of diesel fuel and we, therefore, improved the methods of servicing the diesels so as to eliminate individual contact and provided protective gloves, aprons, salves and ointments for those most exposed to its handling. In combination with the chemical industry, extensive tests were made to determine if less irritating water solvents than the chromates then in use could be provided, with the result we have now entirely eliminated the use of chromate solutions in our water system, substituting therefor, a non-

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irritating but mechanically less effective aluminum base preparation. We have likewise eliminated entirely the use of carbon tetrachloride as a cleansing agent for parts, despite the fact extensive tests have never developed one that was mechanically as effective.

The promiscuous use of protective creams and soaps was eliminated, in our efforts to provide the best protective ointments. We have tested many of them, using one type at one shop and others at other shops, and then by questionnaire and study determined from the users themselves which afforded the best protection. These tests and studies determined there was no one protective cream or ointment that would serve all, and since, to be successful, the program had to meet the needs of each individual, we finally settled upon a standard and now provide five different kinds of protective ointment.

Perhaps the most difficult part of the program was that of educating the men themselves to the fact that carbon tetrachloride, paint thinner, or diesel oil itself, were not the proper cleansers to use after a day's work. As with the protective ointments, we placed in use for test purposes a variety of cleansers with a view to determining the most effective. Here again the study was made with the view to provide the best protection to the individual, and based upon the findings as reported by the men themselves, we are now using two standard cleansers.

All of these studies were made under the supervision of the shop nurse.

As I stated, this program was conducted on an industry-wide basis, and the information gained from the various tests and trials was freely exchanged among the railroads, all benefiting from the experience of the others.

In addition to the above general program, on the Great Northern Railroad, we undertook to make a complete investigation of each case of dermatitis with the view of determining the extent of exposure, the sensitivity of the individual involved, and the desirability of attempting to change the person's type of work. From these studies, it became apparent that in the most stubborn cases, there was very frequently an allergy factor present which was either an underlying cause, or at least a deterrent to rapid and full recovery. We are, therefore, now on the lookout in the examinations of those applying for positions in our diesel facilities for any history of allergy.

Success in a program of this type requires the co-operation of everyone in carrying out the proper procedures; use of protective equipment, the observance of personal hygiene, and a very important factor, the recognizing of early symptoms. Here the shop nurse is of great importance. Co-operation is especially needed from the supervising official, chemists, the shop and roundhouse supervisors, and the safety supervisor and the shop foremen, who must establish regular inspection procedures to determine whether the program is being enforced. Of this group the key man, I believe, is the shop foreman, for it is he who must see to it that instructions on how to wash properly are carried out, that the men change frequently into clean working clothes, that unauthorized protective creams, ointments or soaps are not used, and the proper protective equipment is worn. The best program and plan at higher levels will produce nothing if the foremen do not see to it that it is carried out.

Evidence that our program has been effective, I believe, is the fact that occupational dermatitis on railroads is decreasing. As a matter of fact, the Great Northern Railway Company, though it employs 7,500 men in its shops, roundhouses and stores, does not, at the present time, have one case of hospitalized dermatitis, and only two cases of occupational dermatitis that are keeping the men from work. We are, however, not complacent in our present situation, and we appreciate quite fully that continued success is to be maintained only through preserving and continuing to improve our program. Although we are pleased with the progress we have made, we have attempted to remain ever mindful of the fact that even one case of dermatitis represents a problem to the man afflicted, for whom it is little solace to know he may be the only case out of several thousand employes. When the employe involved is a young man with relatively little seniority, we have normally been able to change him to work that eliminates any exposure without affecting his earnings. However, with the older employes whose seniority has entitled them to preferential jobs and pay, the problem is more acute and not easily solved. We have, however, sought in each such case a satisfactory workable solution.

The job of determining whether or not the exposure was due to carelessness is that of the employer. However, only the examining dermatologist can properly state whether or not the condition is due in any degree to underlying allergy factors. I would suggest that the examining dermatologist take a careful medical history in each case with the view of assisting in determining the extent to which this last factor is present. He should also guard against a tendency to assume that employment in a diesel shop or as an engineman on a diesel locomotive automatically constitutes causating exposure, for, as a matter of fact, at the present time, such is frequently not the case. Perhaps the outstanding proof of the fact that working with petroleum products does of itself constitute dangerous exposure is found in the petroleum industry itself, for it has for many years had one of the lowest incidents of occupational dermatitis among all industrial groups. Here again a carefully taken history will assist the employer in evaluating the case.

I have stated the problem as it relates to the railroad industry because of my limited experience, rather than because of any feeling that we have discovered something new about the control of dermatitis, or that we have had unusual success. To the contrary, all of our plans have been based on work done by dermatologists and other industries years before we became concerned. One of many outstanding examples is the Allis-Chalmers Manufacturing Company of Milwaukee, Wisconsin, who, as early as 1943, undertook a concerted program of occupational dermatitis control and prevention. This program has resulted in a 74 per cent decrease in the incident of dermatitis and a reduction from 10.3 to 2.7 in the number of cases per million manhours of exposure. It is estimated that in two years alone this program effected a saving of over $18,000 in medical and compensation cost.

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of the workman's choice, as naturally as he would to another member of his own clinic. The private physician, on his part, should welcome the inquiries of the plant physician as to the progress of an employe-patient and co-operate by getting him back to work, under the watchful eye of the plant physician, as early as possible.

The attitude of the plant physician should be neutral to both management and labor on matters which are not concerned with health. Where the medical director's professional opinion is required, it should be honest, scientifically sound and based entirely on the medical merits of the case, even though it means a clash with management or labor or both.

Summary

In this discussion I have mentioned five of the foundation stones which are essential if a health service is to succeed. They are:

1. An enlightened, interested and responsive plant manager.
2. A place to work, in the plant, which has been planned and built for the purpose.
3. A qualified and enthusiastic staff, consisting of at least a part-time medical director and a nurse.
4. A well-planned program, which is tailored for the plant served, and written into the plant manual.
5. An attitude of service which places the health of the employe first and maintains a cordial and co-operative relationship with management, labor and fellow practitioners. An attitude of fairness and professional practice which upholds the best in professional ethics and the genuine application of the Golden Rule.

References

1. Heiser, Victor G.: Industrial health practices, a report of a survey of 2,064 Industrial Establishments. Nat. A. Mfrs., (Oct.) 1941.
2. Monk, Ralph M.: Medical Services in Industry, Viewpoint of Management. Indust. Med. & Surg., (June) 1952.
3. Park, W. E.: Industrial Medicine—The doctor on call. Bull. Hennepin County M. Soc., (Feb.) 1953.
4. Park, W. E.: Industrial Medicine—Industrial medicine and absentee control. Bull. Hennepin County M. Soc., (Sept.) 1953.
5. Park, W. E.: Industrial medicine—Impact on employe relations. Bull. Hennepin County M. Soc., (May) 1953.
6. Park, W. E.: Industrial Medicine—The industrial physician as administrator. Bull. Hennepin County M. Soc., (July) 1953.
7. Park, W. E.: Industrial Medicine—Professional ethics in industrial medicine. Bull. Hennepin County M. Soc., (Jan.) 1953.
8. Peacock, Kenneth C.: Costs and profits of industrial medical care. Practical problems of employe relations. Personnel Series No. 149. American Management Assn., 1952.

ABSENTEEISM

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A STORY is told about a sturdy operator of a drop forge hammer in our Lynn plant. One morning his wife appeared at the plant gate to explain her husband's rare absence. She said, in her broken English, "My husband, Joe, can't work today. He was up all night. He couldn't sleep, because, you see, I had a baby last night!"

Any study of absenteeism shows up answers just as surprising as this one. For, more often than not, the people with the best reasons for being absent find a way to get to work—and those with much less reason for absence sometimes fail to come to work. The apparently obvious answers are often not the right ones.

When I first entered the field of industrial health I found a lot of "pat" answers, especially in the field of absenteeism. Answers like—*absenteeism is caused by the absentee-prone. If you weed out the absent-prone you solve your problem.*

By "absent-prone" employe is usually meant an employe who will be among those most frequently absent regardless of the work he does. If there are such employes then it follows that similar proportions of them should be found in large work groups selected at random. It should also follow that the absenteeism statistics should be about the same in similar work groups, if they are of sufficient size to make significant comparisons.

Some of our large plants provide good groups for such a comparison. They have about 2,000 employes in each group; the groups are determined by "the luck of the draw" through a common employment office; the percentage male and female is about the same; the work is similar; *but the absenteeism* rate of one may be double that of its neighbor.

In at least one study made by another company when the so-called absent-prone were removed another group took their place. Also, most of us have seen an "absent-prone" employe change to a normal employe when his home situation was corrected, or his work was changed, or he got a new boss. So it seems unlikely that rooting out "absent-prone" employes will solve the problem.

The second "pat" answer was that *absenteeism among hourly workers doesn't cost anything—that the ones absent are the only ones to lose.*

In fact, one of our manufacturing managers told me that the ones we should go after were the salaried workers, that he had "automatic controls" on the hourly workers by which if they didn't come in they didn't get paid.

He had not faced the fact that when the hourly worker is absent the company had these losses:

1. Extra people on the payroll to take the place of those absent.
2. Idle machinery and unused investment when workers unexpectedly fail to come to work.
3. Disrupted schedules with inconvenienced customers.
4. Higher inventory caused by delay in shipment.
5. Spoilage from substitute workers having to do jobs for which they were not trained.
6. Overtime premiums paid to make up for lost time.

In the case of General Electric our absenteeism the first nine months of this year will average about 5 per cent.* This means that for our billion dollar payroll our employes will not take home approximately fifty million dollars in pay that they would have taken home had they not been absent. It is likely that if we consider the losses to the Company from these absent people that for every dollar our employes failed to take home it costs us one or two dollars additional. These are losses, then, to the employe, to the community, and to the Company.

Instead of using a large company suppose we consider a plant with 300 employes. Such a plant would have an annual payroll of approximately a million dollars a year, so 5 per cent absenteeism would mean a loss of approximately fifty thousand dollars to the employes and between fifty and one hundred thousand dollars to the company. Not all of this can be saved, but some can—enough to much more than pay for the effort.

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*Absenteeism from all causes. It is hours not worked that were planned to be worked, divided by total hours planned to be worked.

The third "pat" answer was that *the way to attack absenteeism was to find out what caused it. That is, was it sickness, personal business; accident; death in the family; or what?*

Isn't it likely that in many cases these are simply *excuses* and not the *reasons* which might be "my job bores me stiff," "I hate the sight of my boss," "I don't think my work group likes me."

One study showed that absenteeism can be broadly classified as: (1) those chargeable to industrial accident or disease: less than 10 per cent; (2) respiratory infections, colds, grip, etc.: 45 per cent; (3) all other causes including personal business, death in the family, etc.: 45 per cent. So, if it is desirable to have some kind of a breakdown of the probable causes of absenteeism, these are probably fair enough.

One of our plants studying this problem concluded that the reasons for their high absenteeism was the fact that their employes traveled an average distance of twelve and a half miles to the plant.

If this were a significant cause of absenteeism then it might be expected that the group of employes journeying over forty miles each day to come to work in this plant would have a much higher absence rate than the group of employes living within a mile of the plant. But a detailed check showed that the employes coming from a long distance had attendance records as good, or better than the employes who lived close to the plant. Absenteeism was more closely tied to motivation than to distance.

The Survey Research Center of the University of Michigan has studied for Detroit Edison Company "What Makes People Absent." They conclude that:

(a) Regardless of the kind of worker, the treatment accorded him will raise or lower his rate of absenteeism,

(b) Doubt is cast that responsible employes come to work regularly and irresponsible ones do not,

(c) Absences both among white collar and blue collar workers are primarily related to *supervision, work associates, job status, and satisfaction.*

You may be surprised to note that no mention of industrial medicine is mentioned as an absenteeism control. Which brings me to the fourth "pat" answer; that *the industrial nurse and doctor can be a prime factor in absenteeism control.*

If a fine modern industrial dispensary staffed with competent doctors and nurses can be a *prime factor* in absenteeism control, why is it that there are such large variances between different departments within the same plant? For example, in one of our typical large plants absenteeism for the third quarter of 1953 was:

	Absenteeism —Male
Department A (over 2,000 employes)	2.7
" B " " "	3.7
" C " " "	5.5
" D " " "	6.8

Each department is equally served by the medical unit; all employes are doing work of a generally similar nature; but, the absenteeism in one department is more than double that of the best of other departments. So it would seem that we should look elsewhere for the major control factor in absenteeism.

If the industrial nurse and doctor are not *prime factors* in control of absenteeism what part do they play? Perhaps one of their most important roles is to show by their treatment of employes that management has a sincere interest in the welfare of employes. This fits in with the modern concept of the industrial doctor being primarily concerned with keeping well people well, rather than simply healing the sick and injured.

There are other things, too, that can be done by the industrial doctor and nurse to lessen absenteeism, but it is doubtful if these will have sufficient influence to be classed as prime factors in control. Some of these are:

1. Good employe placement based on the pre-placement physical examination.
2. Re-location of employes based on periodic physical re-examinations.
3. Special handling of problem cases, including the alcoholic, and those troubled mentally.
4. Rehabilitation programs for employes working at less than capability.

Important as are these and other services performed in the field of industrial medicine, the big area for reducing absenteeism seems to lie more nearly within the control of the employes' own supervisor.

Other comparisons were made to see how absenteeism was related to industry, plant location, size of town, and other factors. But, in each case no simple cause was found to be the answer. One plant, for example, with several thousand employes in one of its Departments had an absenteeism in January of this year of about 8½ per cent. I called the Plant Manager and he said, "Lou, you must remember that this town has pretty rugged winters and the absenteeism figure of 8½ per cent was measured during a single week when we had one of our worst blizzards."

I agreed that that was the probable excuse given for the absenteeism but found it hard to understand why in another Department, employing 4,000 employes in the *same plant,* that during the same week their measured absenteeism was a little over 4 per cent!

What, then, are the factors that effect absenteeism? Well, these things can be said:

Absenteeism gets worse
- with the winter months
- with a higher proportion of female employes
- with high levels of employment
- with prosperous working conditions
- with conflicts in the work group

Absenteeism improves
- with good supervision
- with adequate controls
- with interesting work
- with high "sense of participation" of each employe

Oddly enough, absenteeism seems to improve with some layoffs. The layoffs may increase the turnover rate but drop the absenteeism rate. Probably the reason for this is that employes are afraid they'll be laid off so they either come to work regularly or get another job.

Here are three things that can be done to control absenteeism that are effective and low in cost.

1. *Measure absenteeism, and classify it by foremen.*

In order for management to be able to applaud a good job and criticize a bad job, yardsticks of attendance must be set up. One of the best of these is a report issued monthly, or quarterly, comparing the male and female absenteeism within each foreman's work group with that of each of the other foreman's groups. This report should list each foreman and copies of the report should be sent to every foreman so they can see how they compare with each other.

It is not uncommon to find, when such a report is started, then even though the plant absenteeism averages, say 5 per cent, that it includes foremen who have had groups running under 2 per cent for years, and perhaps one group that has been running over 15 per cent—also for years! But the foremen thought nobody cared about regular attendance; at least not enough to pat him on the back for a good job of getting his people to come to work.

When the report is issued for the first time foremen with poor absenteeism reports are likely to come anxiously into the General Foremans' office with questions about the new report. They will have comments like:

"This is the first time I ever heard that I was responsible for getting my people to come to work!" or

"My record is bad because of those two alcoholics I've got—but they are good mechanics when they're here," or

"My best drill press operator has a farm, and this is his busiest time."

Remarks made by the foremen usually emphasize the fact that they are not going to risk disfavor among their men by being critical of their attendance unless they are forced to it. They don't know how to answer replies like,

"Who's kicking—I'm the guy who don't get paid, ain't I?"

So it becomes important to get to the next step in absenteeism control.

2. *Teach the foreman how to encourage good attendance within his work group.*

One of the most effective things the foreman can do to encourage his employes to come to work regularly is to tell them "they are missed" as soon as they return from an absence. They should learn to greet the returning absentee with factual remarks like,

"Gosh, Joe, when you were out yesterday your machine stood idle all day long."

"You can't run a business with idle machinery," and

"We put Sally on your work yesterday and it was so new to her that she spoiled a lot of pieces. We certainly missed you," or

" . . . and as a result we'll be late in shipment."

In other words, *the emphasis should be put on the fact that the employe was missed, that he was needed on his job,* rather than probing for an excuse for the absence that often uncovers only a good "cock and bull" story.

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Laboratory Aids to Medical Practice

Sponsored by
The Minnesota Society of Clinical Pathologists

THE LABORATORY DIAGNOSIS OF PARATHYROID DISEASE

ELLIS S. BENSON, M.D.
Minneapolis, Minnesota

THE clinical manifestations of both hyperfunction and hypofunction of parathyroid glands are varied and frequently obscure, especially in the early phase. Since early diagnosis makes it possible to avoid debilitating and often irreversible complications, the diagnostic significance of chemical aberrations at a stage in the disease when the clinical manifestations are not outspoken is of utmost importance.

There is no uniform agreement at present regarding the site and mode of action of parathyroid hormone. Evidence suggesting action on bone and on kidney has been presented. Clearly, this hormone plays a major role in the control of calcium and phosphorus metabolism. Following the administration of purified extracts of the hormone four changes take place: (1) immediate, transitory increase in urinary excretion of phosphorus; (2) fall in the serum level of inorganic phosphorus; (3) an approximately simultaneous rise in the serum calcium level and (4) eventual increase in urinary excretion of calcium.

The biochemical abnormalities found in parathyroid disease are strictly in keeping with these hormonal effects. Thus, hyperparathyroidism causes increased calcium and decreased phosphorus in the serum and an increased urinary excretion of calcium. Conversely, hypoparathyroidism is accompanied by decreased serum calcium, increased serum inorganic phosphorus and decreased urinary calcium. No satisfactory method has been devised for the direct assay of the hormone in biologic fluids. Fortunately, then, the biochemical findings in these two conditions provide an indirect but reliable index of hormone function.

Serum Calcium

In interpreting serum calcium findings in the light of parathyroid activity one must keep several considerations in mind:

1. The patient must be *fasting* (an overnight fast is sufficient).

2. The *age* of the patient may be a factor. The normal level of serum calcium in fasting adults has been found to be 10± 1 mg. per 100 cc. of serum so consistently that this may be regarded as a physical constant. Approximately 90 per cent of normal serum calcium levels in adults fall in the range of 9.5 to 10.5 mg. In the period of growth (sixteen years of age or less), the normal serum calcium is somewhat higher, namely 10.5± 1 mg. In infants less than one year of age, a range of 11.0± 1 mg. is considered normal.

3. *Multiple determinations* may be required to establish hypercalcemia in a given instance of hyperparathyroidism because isolated determinations of serum calcium may fall within the normal range.

4. The serum calcium level depends in part on *the concentration of serum protein.* Calcium exists in serum in three physical states which are in equilibrium. Approximately 95 per cent of it is distributed equally between a nondiffusible, nonionized complex with serum protein and diffusible calcium ions. An additional 5 per cent or less exists as diffusible, nonionized calcium citrate.

It has been conventional to consider that parathyroid hormone increased the concentration of ionized calcium and thus affected the level of total serum calcium. Therefore, it would be possible to find the combination of normal total serum calcium, low serum protein and increased ionized calcium in hyperparathyroidism. On the other

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From the Laboratory Services, University Hospitals, University of Minnesota.

This is the fifteenth in a series of editorial reports sponsored by the Minnesota Society of Clinical Pathologists and designed to foster closer relationships between clinicians and pathologists.

hand, hyperproteinemic conditions may be associated with increases in total serum calcium without increase in ionized calcium. This would not be consistent with hyperparathyroidism.

Recent studies cast doubt on these conventional views and indicate that the increase in diffusible calcium in hyperparathyroidism is purely a function of the increase in total serum calcium and has no differential diagnostic significance.

Hypoproteinemia generally is associated with hypocalcemia. In hypocalcemia secondary to hypoproteinemia, the diffusible calcium is normal; tetany and hypoparathyroidism are not present.

Serum Inorganic Phosphorus

The significance of decreased serum inorganic phosphorus in the diagnosis of hyperparathyroidism formerly was not fully appreciated probably because attention was focused on the deranged calcium metabolism and skeletal manifestations. More recently it has been realized that low serum phosphorus levels are more frequently found than elevated serum calcium levels. Moreover, the combination of low serum phosphorus and high serum calcium is sufficiently unique to be of great diagnostic significance. This combination has been found rarely in metastatic carcinoma involving bone but for the most part it serves to separate hyperparathyroidism from other bone diseases. Much the same may be said for the combination of low serum calcium and high serum phosphorus in hypoparathyroidism. An elevated serum phosphorus distinguishes this condition from diseases such as rickets, osteomalacia and idiopathic steatorrhea, which often are complicated by hypocalcemia and even tetany.

In general, normal values in adults range from 2.5 to 4.0 mg. per 100 cc., with approximately 90 per cent being 3.2± 0.5 mg. Children and infants have somewhat higher levels; 5.0± 0.5 mg. and 6.0± 0.5 mg. respectively are accepted normal ranges for these two groups. The fasting state is required in this determination.

In hyperparathyroidism the average decrease may be small. Variations occur from day to day. Multiple determinations may be required and the mean of these values used.

When renal insufficiency supervenes, as it may in hyperparathyroidism, the phosphorus levels in the serum may rise, obscuring the classic changes in phosphorus and calcium.

Urinary Calcium

Determinations of urinary calcium are frequently helpful in the diagnosis of parathyroid diseases. A simple qualitative test, such as the Sulkowitch technique, may be used advantageously as a screening test for abnormal excretion of calcium. The composition of the reagent follows: oxalic acid, 2.5 gm.; ammonium oxalate, 2.5 gm.; glacial acetic acid, 5 cc. and distilled water, q.s. ad 150 cc. Five cubic centimeters of reagent is added to 5 cc. of urine in a test tube. The tube is inverted and the results read as follows: 0 = no cloud in 2 minutes; + = fine cloud in more than 30 seconds; ++ = moderate cloud in more than 30 seconds; +++ = moderate cloud in less than 30 seconds and ++++ = curdlike cloud immediately.

The findings are roughly correlated with the level of serum calcium. A negative or low result tends to exclude hyperparathyroidism, except when renal insufficiency is present. A positive result usually excludes hypoparathyroidism. This test also may be of value in regulating the treatment of patients with hypoparathyroidism. Negative results suggest inadequate treatment, while +++ or ++++ results indicate overtreatment.

In hyperparathyroidism, it is frequently necessary to determine quantitatively the amount of calcium in the urine. In this case, the patient should be placed on a low calcium diet for five days. While on this diet it is important to eliminate extraneous sources of calcium, such as tap water and certain medications. During the last three days of the diet, all urine is collected in three twenty-four-hour aliquots and its calcium content is analyzed. Consistent values of 150 mg. a day or less are considered normal. Values of over 200 mg. a day are found in hyperparathyroidism. This finding may be of special value in establishing the diagnosis in cases in which the serum values are of borderline significance. On the other hand, supervening renal insufficiency in hyperparathyroidism may result in low urine values.

Alkaline Phosphatase

An elevated alkaline phosphatase is not a sign of hyperparathyroidism per se but of bone disease. For this reason it is of limited value in establishing the diagnosis in this condition. It is elevated only in that minority of cases associated with actual skeletal involvement; a normal value most definitely does not exclude hyperparathyroidism. In hypoparathyroidism it is uniformly normal. It is well to remember that alkaline phosphatase is elevated in patients with regurgitation jaundice or hepatic metastatic tumors.

Comment

Initially, hyperparathyroidism was described almost exclusively on the basis of its skeletal manifestations. Chiefly through the efforts of Albright its renal manifestations gained emphasis. Its vague and varied symptomatology in the early stages makes it a disease in which a special search is required. The chemical findings are sufficiently characteristic to allow diagnosis in this early stage. The diagnosis should be considered and sought for in patients with:

1. Renal lithiasis. Approximately 5 per cent of patients with renal stones have hyperparathyroidism.
2. Nonspecific skeletal manifestations, such as generalized demineralization.
3. Vague gastrointestinal complaints not associated with definable gastrointestinal disease. Patients with complaints resembling those of peptic ulcer who fail to respond to or whose complaints are aggravated by conventional ulcer dietary regimens may have hyperparathyroidism.

Hypoparathyroidism also may have a vague and insidious course and be versatile in its manifestations. It should be suspected in patients with:

1. Frank tetany or its equivalents, such as paresthesias, numbness, muscle cramps and stridor.
2. Convulsive disorders.

Especially is this so if the patient has had a previous thyroidectomy or is in the first two decades of life. If in association with these neuromuscular disorders, poor dentition, cataracts or ectodermal dysplasias are found, hypoparathyroidism is especially likely. The diagnosis, once suspected, may be established with certainty through the characteristic chemical findings.

ABSENTEEISM

(Continued from Page 341)

3. *Hold the foreman accountable for the regular, on time, attendance of his employes.*

Getting people to come to work regularly is a personal thing best handled by the employe's own boss. Foremen are normally held accountable for control of cost, maintenance of quality, and adherence to production schedules. Regular, on time, attendance of each employe is essential to carry each of these responsibilities.

What is par for absenteeism? Probably about 3 per cent, made up of slightly more than 3 per cent for female and slightly less than 3 per cent for male.

Summary

Effective factors for controlling absenteeism are:

1. Supervisors who recognize their personal responsibility for regular attendance of their work groups.

2. Employes who have such a high regard for their supervisors that they are eager to give their full skill, care, and effort to their jobs.

3. Management who understand that when excess absenteeism is permitted it is like trying to carry profits in a leaking pail.

President's Letter

CHANGING TIMES

Simulating the ideal civilization, the practice of medicine is forever changing, continually improving itself, always making additions and corrections, although often cautious, which manifest themselves in benefits for all mankind.

Certain important changes and improvements have been made in the program of the annual meeting this year, which, it is hoped, will make a favorable impression on physicians attending.

Last month this letter carried a general preview of what doctors can expect to see and hear at the 101st convention in Duluth, June 7, 8, and 9. While it is true that the entire medical meeting is, in effect, a postgraduate course for doctors, there are certain sections of it which should be emphasized, for they mark important changes in the usual format of the program. I would like to tell you about three of the new features of this year's meeting.

Of special interest to the general practitioner are the two consultation booths, which will be among the scientific exhibits on the convention floor.

One, the Orthopaedic X-Ray Consultation Booth, is sponsored by the Orthopaedic Department of the Medical School at the University of Minnesota. Any problems with fractures or other orthopaedic conditions which members or guests wish to bring in and which can be illustrated by accompanying radiographs, will be discussed by the attending consultants.

The other is an Obstetric-Gynecological Consultation Clinic, sponsored by the Association's Committee on Maternal Health and the Minnesota Department of Health. Consultants will be on duty for fifteen-minute individual discussions of current obstetric and gynecologic problems. Two guest speakers, plus several Minnesota physicians, will staff the booth daily.

The other new scientific feature is scheduled for the Wednesday afternoon general session. The entire afternoon program will be devoted to "What's New?" and will present discussions of what's new in medicine, surgery, radiology, pediatrics, obstetrics and gynecology. Department heads of the University of Minnesota medical school will discuss what's new in each of the above fields of medical practice.

These new programs and policies of the 101st annual meeting indicate a growing and healthy organization—and certainly doctors should be intensely interested in keeping their own organization in the healthiest condition possible. Keeping in tune with the changing times, innovations of this type are certain to bring about a bigger and better convention for 1954.

President, Minnesota State Medical Association

Editorial

Arthur H. Wells, M.D., *Editor;* Henry G. Moehring, M.D., and John F. Briggs, M.D.

PSITTACOSIS

PSITTACOSIS as a disease in human beings is rarely diagnosed and reported. In 1953, an estimated 100 cases were reported in the entire United States, six cases in Minnesota. The clinical picture is one of fever, headache, early pneumonic involvement, moderate cough, marked prostration, delirium, slow pulse, leukopenia, and albuminuria. The cause of the disease is the psittacosis virus. A rising titer of complement-fixing antibodies to this virus during illness is significant, if accompanied by characteristic clinical symptoms. It is essential that at least two samples of serum or whole blood be submitted to the laboratory, the first during the first few days of illness, the second ten to fourteen days following the first. Early administration of antibiotics may prevent the development of complement-fixing antibodies. There are antigenic relationships between members of the lymphogranuloma and the psittacosis group of viruses.

Psittacosis undoubtedly would be discovered more frequently, if suitable laboratory studies were made more frequently. In the laboratories of the State Board of Health, blood specimens submitted from cases of virus or atypical pneumonia with the request for titration of cold hemagglutinins have been routinely tested also for the presence of psittacosis complement-fixing antibodies. A number of such specimens have been found positive for psittacosis, and clinical symptoms have supported this diagnosis.

Psittacosis became of public health importance during the depression years of the thirties when numerous cases, many fatal, occurred in persons exposed to love birds recently imported from tropical countries, and given away as prizes by carnival companies. Following this experience, practically all states adopted strict regulations forbidding any traffic in psittacine birds, and such birds were generally considered serious health hazards.

More recently the virus causing the disease has been demonstrated in a much broader host reservoir among birds, and psittacosis in man has been traced to turkeys, ducks, chickens, pigeons, and pheasants. Sea gulls and petrels have also been found to be infected. The term ornithosis, frequently used interchangeably with psittacosis, is perhaps a better descriptive title for the disease in light of these facts. In some cases of atypical pneumonia with positive laboratory findings for psittacosis, no association with birds can be determined.

The disease is commonly transmitted to man through contact with an infected bird or the bird's recent surroundings. Occasionally, contact with a human case is responsible for transmission, especially if the patient is coughing. Sick birds are highly infectious, and the birds apparently well occasionally transmit the disease. The disease is more severe in older age groups, and death seldom occurs in persons younger than thirty years. Penicillin and broad spectrum antibiotics are fairly effective in treatment of most cases.

In recent years the practice of keeping psittacine birds as household pets has increased tremendously, and people everywhere are breeding and distributing birds. The impossibility of preventing traffic in such pets, and the fact that human cases were being traced to other types of birds has led most states, including Minnesota in 1952, to rescind the regulations outlawing psittacine birds, and to put more effort into publicizing the hazards and suggesting common-sense methods of avoiding infection. Thus, there is a special danger in making house pets of recently imported psittacine birds or when there is doubt as to recent contact with other and especially with sick birds. Birds should be purchased only from reliable dealers. Shipment of birds from outside the United States is prohibited, and any birds smuggled or bootlegged into this country should be considered as a dangerous potential source of psittacosis. The common practice of permitting such pets to take food from the owner's mouth invites infection, as does any intimate handling of the birds. Pet shops and homes that have harbored infected birds should be thoroughly cleaned before any new birds are distributed from them, and it is good practice to keep new shipments of birds isolated in quarantine for thirty days to eliminate sick individuals. This does not guarantee that the remaining birds are free of infection, but it is helpful.

D. S. Fleming

MALPRACTICE INSURANCE AND ITS COST

YOUR malpractice insurance premiums have been increased. You probably wonder why. You may have a general idea, but have given no serious consideration to the problem facing the medical profession or the insurance companies. Whatever has been your attitude or your interest, the situation is extremely unfavorable and deserves your immediate attention.

Ignorance on the part of members of the medical profession about this important subject is disturbingly evident in spite of effort of insurance companies to bring the matter to their attention. It is also evident that there is a lack of understanding in what the insurance does for the profession as a whole, or how it protects against ruinous losses unless physicians, as individuals, are faced with such a claim or suit. Many have no idea how the rates (premiums) are promulgated. Few understand that the premiums must cover expenses as well as claims and, as a result, believe the insurance companies make fantastic profits. This belief is shared by the same people who believe that all doctors are rich. In the light of these misunderstandings, it is easy to understand why some people support legislative proposals to place automobile liability insurance, workmen's compensation, health insurance and other casualty insurance, under monopolistic state funds and governmental control. It also becomes easy to understand why juries hand out large awards today. Other contributing factors are: Judgments and expense items are paid in terms of inflated dollars. The juryman who pays $2,500 for a low priced automobile and $1.50 for a Blue Plate Special might not hesitate to award a large judgment in a malpractice suit.

The medical practice itself has not escaped social evolution. The personal relationship between the physician and the patient has practically disappeared. Individual practice has been replaced by group practice, clinic and partnership so that the patient does not—except on rare occasions—consult the same physician twice in succession. This may be good medicine, but it has certainly eliminated some of the warm, personal family physician relationship that once existed, plus the fact that the increased use of the hospital has taken an even larger segment of medical practice out of the home and office.

Claims originate in many and divers ways—physicians criticizing another's methods and treatment results, divulging facts about a case or ailment to others, improper diagnosis, improper treatment, and the ever-present fictitious case or hypothetical case.

The methods of correcting the situation are known. Everyone should ask these questions:

Do I follow the rules of my profession?

Do I follow honest and safe practices?

Do I realize that insurance companies are subject to the same economic pressures as my own?

Are claims morally and legally honest?

Are insurance costs in line with my own, considering current conditions, and not conditions of twenty years ago?

Have I opposed socialization of the insurance industry, as I have my own business, remembering when socialism gets its foot in the door in one important branch of the free enterprise system, it will soon be in the door altogether?

In the answer to these questions, you will find the causes for the rising insurance rates.

Don C. Hawkins
Insurance Representative

WORDS AND MEANINGS

NEW WORDS that precisely express a meaning hitherto ineffable are a delight. The exercising of such new words is healthy except when it results in painful repetition. Perhaps as pleasing as acquiring a new word for our working vocabulary is learning the original meaning of an old word or learning the meaning of a similar word in another language than English. Often such information is the means of understanding something that would otherwise have to be retained by brute memory. The abdominal, pancreatic complication of mumps ceased being mystifying, for example, after we recalled that the German word for pancreas was *Bauchspeicheldrüse,* the abdominal salivary gland. The vague significance of malaise became less vague when our friends who fretted with beginning French reminded us that *malaise* meant "bad ease" and thus resembled ill at ease.

The sobriquets applied to members of one nation by peoples of another, usually born of the mingling of nationalities during wartime, are rich in friendly banter and good fellowship, if directed toward allied forces, and as rich in sarcastic deprecation, if directed toward the enemy. Thus, in the pidgin of the Far East, the English soldiers were known as *Assays* from their frequent "I say." The same English soldiery was called *Godon* by the French and *Gadan* by the Spanish, after one of their favorite curses. The Portugese named them *Camones* and the Italians made it *Caman* from their impatient, "Come on, come on!" Italians named German soldiers *Tertòifel* and *Tartàrfel* from their use of *"der Teufel."* Years earlier, the Milanese gave Austrian troops the name *Zuruc* because of their commands to crowds to back up, *"Zurück!"* This recital must be incomplete, but the term, *Damchee,* given to American troops, and finally to Americans in general, by the Indian troops associated with the VIII Army in North Africa, has too interesting an explanation to skip: whenever American soldiers made a purchase, the low price startled them into saying, "Damn cheap," while paying.

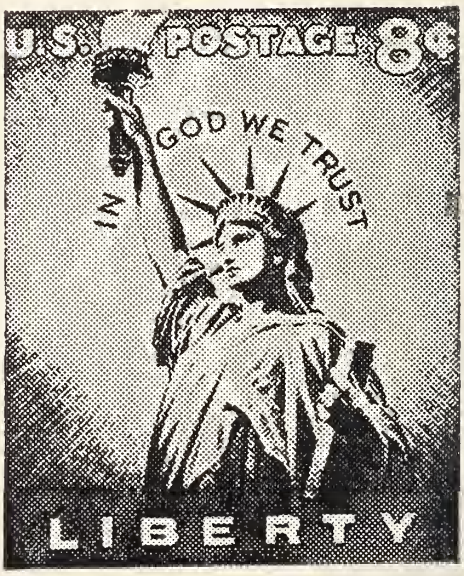
However, the imaginative, occasionally profound, insight some words and word-combinations allow give greater pleasure than these brief excursions into derivations of words. We are told about the language of the Guarani, the Phoenicians of South America, who live in the general area of Paraguay. Their language is essentially a primative one and almost solely a spoken one. It is rarely reduced to writing. The picturesque images drawn by the Guarani words brings the realization that artistry may be found in the everyday conversation of mankind, if the listener perceive the full import of the word combinations. The Guarani speaks not of a "kick" but of a *pï-voi:* the *pi* means foot, and the *vói,* fast or nimble. A kick thus becomes a fast-foot. *Tembé* means lip and *i* means water or a stream. What more logical than that *tembé-i* indicates the banks of a stream? Then in the subjective realm, *guazu* means large and *pïa,* stomach. The combination of the two, *pïa-guazu* adds up to big stomach and is used for courageous. Finally, this crowning jewel of understanding: the same *pia* (stomach) is coupled with *guapi* (seated) so that *pia-guapi* means tranquility. And for how many of our patients do we not wish tranquility via a well-seated stomach? H.G.M.

MINNESOTA MEDICINE—5c

A SURVEY of the past ten years indicates that Minnesota Medicine has cost members of the Minnesota State Medical Association less than five cents a copy during that period. Although two dollars has been allocated from annual dues as a subscription for the journal, all but about sixty cents has been returned to the treasury. The receipts from advertising, reprints, illustrations, and so forth make up the difference. In looking to the immediate future, with the increase in advertising reflected in the past nine months, it is possible that even a lower cost per copy may be achieved.

Your active support of our journal and its advertising benefits both science and organized medicine. If Minnesota Medicine is not what you need, your suggestions will receive careful consideration by the Board of Editors.

NEW STATUE OF LIBERTY STAMP



At the request of thousands of persons, the Post Office has issued a new stamp with a religious sentiment. The new stamp will be used primarily for international mail.

The Dean's Page

THE MEDICAL STUDENTS' ADVISORY COUNCIL

With increasing interest and pleasure, members of our faculty have watched the recent development of an effective, responsible *vox discipulorum,* the Medical Students' Advisory Council. The Council was organized about three years ago for the purpose of advancing "the interest and well being of all medical students and the University." It is made up of the class officers—president, vice president, and secretary—of each of the four classes in the Medical School. The Council in turn selects one member from the preceding year's Council who acts in an advisory capacity. The Dean and the Assistant Dean are also *ex officio* members of the Council.

The Council has concerned itself with a number of problems of interest to students and faculty alike. As one of its major projects, last spring it conducted a survey of student and faculty opinion concerning methods of examination of students, grading and ranking of students, periodic evaluation of the curriculum, and student-faculty relationships. The results of this survey were reported at the regular University Hospital Staff meeting on January 8 of this year and appear in some detail in the Bulletin of that date.* In brief summary, students and faculty agreed on the desirability of some form of periodic evaluation of courses, and both groups indicated that they wanted more opportunities for informal student-faculty discussions. The students were in favor of a simple "pass or fail" grading system with class rank to be determined by a special comprehensive examination and perhaps also by special "honors" work. The majority of the faculty, on the other hand. did not favor such a separation of grading and ranking systems, but most faculty members thought that the grading system could be modified and improved. The students do not like the summer session which has been in effect for the freshman sophomore, and junior classes for the past three years. Faculty reaction on this question was mixed; some faculty members are uncertain of the value of the summer session.

As a result of the survey, the Council has presented to the Medical School Administration several recommendations concerning grading systems and the curriculum. These are now under consideration. The Council has also prepared a questionnaire for the evaluation by the students of the individual courses given in the freshman and sophomore years.

Two other Council projects are worthy of special comment. Members of the Council are co-operating with the Student Counseling Bureau in a plan whereby interested junior and senior medical students will assist in the advising of pre-medical students. The Council is also planning a Pre-Medical Day for the latter part of this month for all pre-medical students at the University and neighboring colleges and for members of next fall's freshman's class. This will provide a fine opportunity for prospective medical students to become acquainted with the Medical School and its faculty.

We believe that members of the Council are to be congratulated for the careful consideration they have given to a number of important problems. I am sure that they do not expect that their suggestions will always result in immediate action. Their recommendations, however, will always be thoughtfully weighed by the administration. New ideas with merit are always welcome whether they originate with students, faculty, or persons unassociated with the Medical School.

HAROLD S. DIEHL, M.D.
Dean of the Medical Sciences
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*Proehl, Elsa; Feller, William; and Rosenholz, Mitchell: Medical Education at Minnesota. A Survey of Opinion by Medical Students in 1953. Bull. U. Minnesota Hosp. & Minn. Med. Found., 25:216, Jan. 8, 1954.

Medical Economics

Edited by the Committee on Medical Economics
of the
Minnesota State Medical Association
George Earl, M.D., Chairman

RUSSIA'S STATE MEDICINE NOTED IN ARTICLE

Surprising and interesting facts are presented on the status of medicine in the USSR in an article carried in a recent issue of the *Journal of the Indian Medical Association*. The article, entitled, "Medicine and Public Health in USSR" was written by A. C. Ukil, of Calcutta, India, and is based on a visit to Russia as a member of a Scientific Delegation from India in 1951.

The article shows, of course, a very decided pro-Russian slant, in view of the geographical and political proximity of the two countries. A sanctioning of the principle of state medicine is evidenced throughout the writing.

The article is of vital interest to American physicians, as it presents a completely pro-Russian, yet factual, view of medicine and public health in the Soviet Union. The author introduces the article with:

> "In understanding the progress in Russia, one has to remember Russia's technological backwardness at the time of the Revolution (1917). Knowing that science has developed more rapidly than society's adjustment to the new conditions created by it, the authors of the Marxist Revolution wanted to make a gigantic effort, by forced marches through successive 5-year plans since 1928, to apply the principles of science to the basic process of life, to production, distribution and consumption in the whole of the Soviet Union. They wanted to banish these by the creation of a Socialist State."

Education—A State Plan

The article describes medical education in Russia in considerable detail, noting,

> "During the first 5-year Plan (1928-32), only 14 medical colleges were opened. Now, there are 73 medical colleges turning out 18,000 medical graduates every year. There is no unemployment. As soon as they pass out, they are immediately offered posts, according to the requirements of the Master-Plan of the country. Stipend-holding medical students, when they pass out, are required to work in rural areas for 3 years. After receiving a broad-based experience there, they are transferred to urban areas or to research institutes as desired by them. The vistas of progress are open to everybody and the social conditions created encourage it."

The scientific delegation visited two medical colleges in Russia, the Second Medical Institute in Moscow and the Medical Institute at Alma Ata. According to the article, the Second Medical Institute at Moscow admits 600 students every year and the total number of students is 4,000. The institution has, attached to it, twelve hospitals with 5,000 beds. Five to seven beds are allocated to each student during "interim" training.

Health—Duty of State

In Russia, the health of the populace is the duty of the state, the article notes:

> "According to Article 120 of the constitution, citizens of the USSR have the right to maintenance in old age and also in case of sickness or disability. This right is ensured by the extensive development of social insurance of factory and office workers at State expense, free medical service for the working people and the provision of a wide network of health resorts for the use of the working people (accommodating 500,000 workers at a time during vacations, with full pay)."

The article continues:

> "The State has also taken over the production of medicaments and their supply to the population. All medical care is free and available to all, regardless of the patient's resources. The State's aim is not merely to cure disease but to prevent it, by creating such living and working conditions as would make the occurrence of disease rare."

Medical Service in Russia

In 1913, according to the same article, there were only 20,000 qualified doctors in Russia. Today, there are 220,000 doctors only under the Ministry of Health, excluding a large number in Railways, industry, et cetera.

Medical and health service throughout Russia are based on a decree issued by Lenin in 1918:

> "(1) To carry through resolutely extensive sanitary sures in the interests of the working people, such as a) improvement of health conditions in populated places (protection of soil, water and air from pollution), (b) organization of public catering on a scientific and hygienic basis, (c) launching of measures to prevent the outbreak and spread of infectious diseases, and (d) creating a code of health legislation; (2) to combat social diseases, such as tuberculosis, venereal diseases, alcoholism, etc.; (3) To make competent medical and pharmaceutical services available to all free of charge."

Distribution Noted

According to Ukil's article, there is, at present, one doctor for 800 people in urban industrial areas and one doctor for 1,000 people in rural areas. There are 10.5 beds per 1,000 people. This ratio prevails all over the union, the article states. Further:

> "With the larger output of doctors as time passes on, this ratio will be further reduced. No doctor, when he passes out, is unemployed. Private practice being practically *nil,* doctors devote their whole attention to the curative, preventive and educative work of raising the health status of the people."

Mass Production Health

In describing medical service in urban and industrial areas, a city is divided into several zones, districts or boroughs, with a district hospital serving about 40,000 to 50,000 people. The article describes the hospital set-up, and it is interesting to note how intricately organized and integrated the political and medical facets are:

> "In each hospital there is a *Board of Medical Control,* consisting of the senior staff, who analyse all cases staying in the hospital for longer than 10 days. . . . In case of urgent surgical or emergency cases the patient first informs the Polyclinic, which immediately informs the Central Emergency Station which must make arrangements to take the case to the hospital, where a bed is vacant, within 20 minutes. The specialists immediately examine the patient and, if there is nothing to the contrary, the patient, in surgical cases, is operated within 15 minutes after arrival in hospital. If there is any violation of this, it is considered as neglect and the Town Council is likely to discuss the matter and recommend necessary disciplinary or organizational steps. . . . The factory population get a health check-up twice a year, while the rest of the population get it once a year."

The article makes this conclusion:

> "Thus the people participate in the health programme from the base to the apex of the health pyramid. Diagnostic and curative doctors and health personnel cooperate at all levels."

Thorough Social Insurance

Complete social insurance is existent in Russia also. It covers the following, noted in the article: (a) medical care, (b) benefits in case of temporary disabilities (sickness, accident, quarantine, pregnancy, childbirth and nursing a sick member of the family), (c) additional benefits for infants, funerals, et cetera, (d) unemployment benefits . . ., (e) invalidity pension, (f) old age pensions, (g) pension to families in case of death of breadwinner. "Social insurance benefits extend not only to the insured workers but also to their dependents."

Other matters are covered in detail in the article, such as the organization and structure of the Health Ministry, regional distribution of medical institutions, medical and health workers' trade union, the production of drugs and instruments, and health resorts and sanatoria.

The pro-Russian bias is seen throughout the article in such statements as: "cooperate fully with the State's efforts to improve health and to prevent diseases," and "in this way, modern scientific diagnosis and treatment are taken to the whole population." Organization of health and medical services in Russia is handled from the top down to the people, and of course, the top is the State, supreme in government and in governing individual rights of medical care of all people.

GERMANY SHOWS SURPLUS OF DOCTORS

According to a recent article in the *St. Paul Pioneer Press,* there is a surplus of 30,000 doctors in Germany. In West Germany, there are 4,543 doctors unemployed or dependent on odd jobs outside the profession. 18,000 doctors have all but deserted the medical field for other careers or are waiting for a chance to set up private practice; and there are 8,000 postgraduate medical students working without pay as interns.

The reason for the whole problem is the complicated traditional German system of public health insurance, including four out of five people in the country. According to the newspaper, "public health insurance in Germany is compulsory up to a monthly income of $100. Voluntary subscription is permitted over the $100 level." Physicians cannot depend on the two out of every 10 patients who can go to private doctors. They must try to become part of the public health insurance system, which is very difficult. The newspaper states:

(Continued on Page 366)

Minnesota State Medical Association

101st Annual Meeting

Duluth, Minnesota
June 7, 8 and 9, 1954

Program

BUSINESS SESSIONS

SATURDAY, JUNE 5

Hotel Duluth

2:00 P.M.—Council.....................English Room
6:00 P.M.—Council.....................English Room

SUNDAY, JUNE 6

Hotel Duluth

8:30 A.M.—Council.....................English Room
10:00 A.M.—Reference Committees................Rooms to be assigned
2:00 P.M.—House of Delegates..............Ballroom
8:00 P.M.—House of Delegates..............Ballroom

MONDAY, JUNE 7

Hotel Duluth

8:30 A.M.—Council.....................English Room
12:15 P.M.—House of Delegates............Ballroom

TUESDAY, JUNE 8

Hotel Duluth

8:30 A.M.—Council.....................English Room
8:30 A.M.—Committees...............Ballroom Floor

WEDNESDAY, JUNE 9

Hotel Duluth

8:30 A.M.—Council.....................English Room
8:30 A.M.—Committees................Ballroom Floor

SOCIAL EVENTS

SUNDAY, JUNE 6

Buffet Supper

American Medical Women's Association, Inc., Minnesota Chapter

6:30 P.M., Home of Elizabeth Bagley, M.D. Open to all visiting women doctors. Dr. Marie Bepko will show colored slides from the Fourth Congress of the Pan-American Medical Women's meeting in New York City.

MONDAY, JUNE 7

Luncheons

Minnesota Chapter, American College of Chest Physicians

12:15 P.M., Parlor B. Hotel Duluth, tickets $2.00

Minnesota Medical Foundation

12:15 P.M., Arrowhead Room, Hotel Duluth, tickets $2.00

Speaker: Clarence Dennis, M.D., New York

Dinners

Minnesota Academy of General Practice

6:00 P.M., Ballroom, Hotel Spalding

Open House, Minnesota State Medical Association
9:00 P.M., Ballroom, Hotel Duluth

Minnesota Radiological Society

6:00 P.M., Social Hour

7:00 P.M., Dinner

Speaker: Philip J. Hodes, M.D., Philadelphia—"An Evaluation of Body Section Radiography" Northland Country Club, 39th Avenue East and Superior Street. Tickets, $5.00. Reservations should be made with Dr. John R. McNutt, 824 Medical Arts Building, Duluth.

TUESDAY, JUNE 8

Breakfast

Former St. Mary's Interns Reunion

7:30 A.M., St. Mary's Hospital

Dinners

Minnesota Society of Clinical Pathologists

6:00 P.M., Annual Dinner and Business Meeting—Home of A. H. Wells, M.D.

Annual Banquet, Minnesota State Medical Association

7:00 P.M., Ballroom, Hotel Duluth

12:15 ROUND TABLES

Duluth and Spalding Hotels

MONDAY, JUNE 7

Anesthesia as a Maternal Mortality Factor
Frederick J. Hofmeister, Milwaukee, Wisconsin

TUESDAY, JUNE 8

Use of Tracheotomy in Chest Injuries
W. P. Eder, Minneapolis

Treatment of Burns in Children
Tague C. Chisholm, Minneapolis

Emergency Treatment of Nerve Injuries
Robert L. Merrick, Saint Paul

Indications and Contraindications for the Use of the Hormones (ACTH and Cortisone) in Gastrointestinal Disorders
J. Arnold Bargen, Rochester

Skin Manifestations in Systemic Disease
John F. Schmid, Duluth

Acute Urinary Obstruction in the Male
Otto C. Phares, Saint Cloud

Drugs in Peptic Ulcer
E. E. Gambill, Rochester

The Injured Hand
J. C. Feuling, Duluth

The Management of Small Bowel Obstructions
Clarence Dennis, New York

Obstetric Hemorrhage as a Maternal Mortality Factor
Charles E. Galloway, Evanston, Illinois

WEDNESDAY, JUNE 9

Diagnostic Difficulties in Carcinomas of the Lungs
F. F. Callahan, Saint Paul

Evaluation of Operations for Peptic Ulcer
C. W. Lillehei, University of Minnesota

Relation of Blood Volume to Gastrointestinal Hemorrhage
J. E. Twomey, Minneapolis

Practical Application of Cytologic Technique for Malignancy
A. H. Wells, Duluth

Anesthesia in Tonsillectomy
F. C. Jacobson, Duluth

Functional Uterine Bleeding
R. F. Sturley, Saint Paul

Treatment of Plantar Warts
R. W. Goltz, Minneapolis

Diagnosis and Treatment of Blood Dyscrasias
Paul S. Hagen, Saint Paul

Significance and Treatment of Lumpy or Discharging Breasts
A. R. Curreri, Madison, Wisconsin

GENERAL SESSION

Duluth Armory

MONDAY, JUNE 7

A.M.

8:30 Visit Scientific and Technical Exhibits.... Armory

9:00 PANEL DISCUSSION ON THE TREATMENT OF HYPERTENSION
Moderator—R. M. Shick, Rochester
Henry A. Schroeder, Saint Louis, Missouri
A. C. Corcoran, Cleveland, Ohio
James H. Currens, Boston, Massachusetts
Robert W. Hollenhorst, Rochester
Question and Answer Period

Intermission

10:15 Visit Scientific and Technical Exhibits.... Armory

11:00 Plastic Operations for Hydronephrosis
Frederic E. B. Foley, Saint Paul
Comments
Ormond S. Culp, Rochester

11:40 Staphylococcal Sepsis; Control with Antibiotics
Robert I. Wise, Assistant Professor, Department of Medicine, University of Minnesota

P.M.

12:15 Miscellaneous Luncheons
Round-Table Luncheon

1:30 Visit Scientific and Technical Exhibits..Armory

2:00 Cancer of the Cervix
A. N. Arneson, St. Louis, Missouri

2:30 RUSSELL D. CARMAN MEMORIAL LECTURE
Pitfalls in the Diagnosis of Pulmonary Diseases
Philip J. Hodes, Professor of Radiology, School of Medicine, University of Pennsylvania, Philadelphia

Intermission

3:15 Visit Scientific and Technical Exhibits..Armory

4:00 SYMPOSIUM ON ANESTHESIOLOGY
Moderator—Ralph T. Knight, Minneapolis
Hypotension During Surgery and the Immediate Postoperative Period
Ferdinand C. Jacobson, Duluth
Cardiac Arrest During Anesthesia
James H. Matthews, Minneapolis
Will Analgesia Evolve from a Century of Anesthesia?
John S. Lundy, Rochester
Trichlorethylene—Its Possibilities for the General Practitioner
O. Sidney Orth, Professor of Pharmacology, University of Wisconsin, Madison

5:00 Visit Scientific and Technical Exhibits..Armory

6:00 Special Dinners

9:00 Open House......................Hotel Duluth

GENERAL SESSION

Duluth Armory

TUESDAY, JUNE 8

A.M.

8:30 Visit Scientific and Technical Exhibits..Armory

9:00 SYMPOSIUM ON PROBLEMS ARISING IN THE CLINICAL USE OF CORTISONE, HYDROCORTISONE AND CORTICOTROPIN
Problems Arising in the Treatment of Adrenal Insufficiency
Edmund B. Flink, Minneapolis, Moderator
Untoward Effects Upon the Gastrointestinal Tract
Eric E. Wollaeger, Rochester
The Use, Abuse and Limitations of Steroid Therapy in Dermatology
Francis W. Lynch, Saint Paul
Problems of Dosage in Treatment of Rheumatoid Arthritis
L. Emmerson Ward, Rochester

Problems Arising From Suppression of Adrenal Cortical Function by Cortisone and Hydrocortisone
Robert M. Salassa, Rochester
Pathology of the Pituitary and Adrenals
Warren A. Bennett, Rochester
Question and Answer Period

Intermission

10:15 Visit Scientific and Technical Exhibits..Armory
11:00 The Clinical Management of Fulminant Infections in Childhood
Edward B. Shaw, Clinical Professor of Pediatrics, University of California Medical School, San Francisco
11:30 ARTHUR H. SANFORD LECTURESHIP IN PATHOLOGY
The Pathologist as a Practitioner of Medicine —Fifty Years of Progress
John R. Schenken, Professor of Pathology, University of Nebraska Medical School, Omaha

P.M.
12:15 Miscellaneous Luncheons
Round-Table Luncheons
1:30 Visit Scientific and Technical Exhibits..Armory
2:00 ULCERATIVE COLITIS
Medical Aspects
J. Arnold Bargen, Rochester
Psychiatric Aspects
L. R. Gowan, Duluth
Surgical Aspects
Clarence Dennis, New York
Question and Answer Period

Intermission

3:15 Visit Scientific and Technical Exhibits..Armory
4:00 SYMPOSIUM ON FRACTURES OF THE SPINE AND LOWER EXTREMITIES
Fractures of the Spine and Pelvis
John C. Ivins, Rochester
Fractures About the Knee Joint
Harry B. Hall, Minneapolis
Fractures of the Tibia and Fibula in Adults
Malvin J. Nydahl, Minneapolis
Fractures of the Lower Extremity in Children
S. S. Houkom, Duluth
5:00 Visit Scientific and Technical Exhibits..Armory
7:00 Annual Banquet......Ballroom, Hotel Duluth

GENERAL SESSION

Duluth Armory

WEDNESDAY, JUNE 9

A.M.
8:30 Visit Scientific and Technical Exhibits..Armory
9:00 Diagnosis and Treatment of Hemolytic Anemia
Paul S. Hagen, Saint Paul
Anemias in Pregnancy
Roy G. Holly, University of Minnesota
Plasma Expanders
A. R. Curreri, Associate Professor of Surgery, University of Wisconsin Medical School, Madison
Question and Answer Period

Intermission

10:15 Visit Scientific and Technical Exhibits..Armory
11:00 The Problem of the Benign Mole and Malignant Melanoma......George T. Pack, New York

P.M.
12:15 Round-Table Luncheons
1:30 Visit Scientific and Technical Exhibits..Armory
2:00 WHAT'S NEW?
Moderator—F. J. Hirschboeck, Duluth
In Medicine
C. J. Watson, Professor and Head, Department of Medicine, Medical School, University of Minnesota
In Surgery
Owen H. Wangensteen, Professor of Surgery and Chairman, Department of Surgery, Medical School, University of Minnesota
In Radiology
Leo G. Rigler, Professor and Head, Department of Radiology, Medical School, University of Minnesota
In Pediatrics
Irvine McQuarrie, Professor and Head, Department of Pediatrics, Medical School, University of Minnesota
In Obstetrics and Gynecology
John L. McKelvey, Head, Department of Obstetrics and Gynecology, Medical School, University of Minnesota
Question and Answer Period

SPECIAL SESSIONS

MONDAY, JUNE 7

A.M.
10:00 Minnesota Academy of Ophthalmology and Otolaryngology, St. Luke's Hospital Auditorium
Hearing Loss in Children—Detection, Treatment and Rehabilitation...................
Kinsey M. Simonton, Rochester
Chronic Middle Ear Suppuration—Today......
L. R. Boies, Minneapolis
Retrolental Fibroplasia.......................
Erling Hansen, Minneapolis
Some Types of Retinal Vascular Occlusions....
Henry P. Wagener, Rochester

P.M.
1:00 Round Table Luncheon
Discussion on Vertigo.......................
Mr. Terrance Cawthorne, M.D., London, England

TUESDAY, JUNE 8

A.M.
9:00 Minnesota Society of Clinical Pathologists
Tumor Seminar..............St. Mary's Hospital
Moderator—Benjamin Castleman, Professor of Pathology, Harvard Medical School, Boston, Massachusetts
11:30 Arthur H. Sanford Lectureship in Pathology.. Duluth Armory

P.M.
12:00 Luncheon....................St. Mary's Hospital
2:00 Tumor Seminar (Continued)..................
St. Mary's Hospital
6:00 Annual Dinner and Business Meeting

History of Medicine In Minnesota

MEDICAL HISTORY OF THE COUNTY OF YELLOW MEDICINE

MILDRED B. LEE
Granite Falls, Minnesota

(Continued from April, 1954, issue)

Charles William Watson was born in Longford, Ireland, in 1863, and went to Canada with his parents at an early age. His boyhood was spent on a farm near Pilot Mound, Manitoba. After completing his elementary education he came to Minnesota to take his medical training at Hamline University, graduating in 1897.

At that time his brother Dr. James A. Watson was practicing medicine in Wood Lake, Yellow Medicine County, and another brother Frederick G. Watson was a pharmacist and proprietor of a drug store at that place. He seems to have begun his medical practice at Boyd before 1900, for the local papers of that time mention his presence in Wood Lake at various periods, taking over his brother's practice at times, and at other times working with him.

Dr. Charles Watson's service in the county was periodic and probably ended when he left the region in 1909. In 1927 he was located in Minneapolis, but was in poor health and unable to practice. He had contracted an illness which resulted in sleeping sickness.

Dr. Watson died May 15, 1930.

Florado H. Wellcome was born in Stevens Point, Wisconsin, in 1856. In 1858 the family moved to Zumbrota, Minnesota, where they lived for three years, then to Garden City, and finally to New Ulm. Dr. Wellcome was educated in the schools of these villages and took his high school course at Mankato, Minnesota. He then entered Rush Medical College in Chicago, graduating in 1879. He married Mary Taylor, of Madison, Wisconsin, and began his practice of medicine in Granite Falls, Minnesota.

Soon after his arrival in Granite Falls diphtheria became prevalent, and he was on call day and night. This was followed by the epidemic of 1881, when many fell victim to smallpox. An astute businessman, Dr. Wellcome's interests lay in finance rather than in medicine, and although he returned to Rush Medical College in 1884 for a postgraduate course in medicine he spent no small part of his time looking after business interests. About 1883 he took over the loan and collection business of Mr. Teachout, and in the fall of 1884, he announced the establishment of the Yellow Medicine County Bank with himself as president and F. W. Pearsall as Cashier. He advertised his loan business extensively in the papers, and practices in connection with it sometimes resulted in litigation in which it was found that usury was a factor.

In appearance Dr. Wellcome was a large man, of fine physique and regular, rather handsome features. A shrewd businessman, he was able to amass a considerable fortune rather early in life; a circumstance which enabled him to travel extensively in this country and abroad. His sister-in-law, Miss Laura Taylor, accompanied Dr. and Mrs. Wellcome on these journeys and wrote a most interesting account of them which was published in limited edition, beautifully bound, as *Three Westerners Abroad.* It appears that Dr. Wellcome was related to Henry Wellcome, then a member of the English chemical suppliers, or drug firm, of Burroughs and Wellcome which is still in existence. Mrs. Wellcome's forebears also being English they visited the ancestral home of the Taylors also.

Dr. Wellcome discontinued the practice of medicine in Granite Falls about 1885, to devote himself to business. In 1894 he removed to Minneapolis where he became a director of the National Bank of Commerce until its consolidation with the Northwestern National Bank. In 1903 he was one of the organizers of the Union Investment Company, a banking house interested in country banking, farm loans, and commercial business, and became president of this company and several country banks throughout the northwest.

Dr. Wellcome was a member of the Granite Falls Masonic Lodge, and of the Minneapolis, Lafayette, and Minikahda Clubs. He died at the age of sixty-two at his home on 210 Groveland Avenue, Minneapolis, on December 20, 1920. He was survived by his wife, Mary Taylor Wellcome, and three brothers. The only child of the couple, Mamie Wellcome, died at Granite Falls at the age of nine months. A memorial window bearing her name was given to the Congregational Church of Granite Falls.

John L. Wakefield was born in Winsted, Connecticut, in 1823. He received his early training there, and entered Harvard College for medical training. After graduation he began to practice at Winsted under the direction of his father. In 1854 he came west to Shakopee, Scott County, where he lived for a time, becoming one of the first officers of Shakopee Lodge No. 6, A. F. and A. M. In 1861 he was appointed government physician to the Upper Sioux Agency to succeed Dr. Jared Waldo Daniels who had resigned.

Dr. Wakefield and his family took up residence in a large, two-story brick building on the bluffs above the Minnesota River at the site of the Upper Sioux Agency. This house was of solid construction, its walls being fourteen inches thick, and it was only partially destroyed in the Indian uprising of 1862.

Dr. Wakefield's work began with new officials appointed by the Lincoln administration, who were committed to a policy of individualizing the Indians and breaking up their community system. This policy was resented and met with fierce opposition from the Indians. Resentment over the encroachments of the white men and failure of government to live up to promises made to the Indians had been building up for some time, culminating in the hostilities which began August 17, 1862.

Dr. Wakefield was away from the Agency with the Agent when news of the massacre at Acton was brought to the Upper Sioux Agency, and Mrs. Wakefield immediately took her children and set out for Redwood. On the way she was overtaken and picked up by a young man with a horse and buggy. But the attack on Redwood was already under way, and before the party reached that place they were set upon by Indians who killed the young man and took Mrs. Wakefield and her children prisoners.

Dr. Wakefield returned to the Agency later in the day, and was one of the party of twenty-two men and forty women and children who took refuge in the government warehouse. Fortunately for them, the Indians busied themselves with looting and burning the stores, and in the early morning hours John Other Day, a friendly Christian Indian, procured a team and wagon to transport the wounded and children and helped the refugees to get across the river. They had little in the way of arms, ammunition, or food, but the party made its way to Fort Ridgely, which they found under seige. They also found St. Peter under attack, but soldiers from Fort Snelling arrived shortly after the party passed and drove off the attackers. Dr. Wakefield then returned to St. Peter where he helped to care for the wounded.

At the cessation of hostilities Mrs. Wakefield and children were reunited with the doctor, and they settled in St. Peter. In 1869 Dr. Wakefield became postmaster there. He died in February, 1875, at the age of fifty-one years, from an overdose of an opiate. According to his obituary he was "well known and left many friends."

Thomas Smith Williamson was born in March, 1800, in Union District, South Carolina, the son of Rev. William Williamson and his wife Mary Smith Williamson. His grandfather was Thomas Williamson, and his grandmother's name before marriage was Anne Newton, and she was a distant relative of Sir Isaac and the Rev. John Newton. When his father was eighteen years of age he accompanied Gates in his expedition through the Carolinas, and after the war was over he resumed his studies and was graduated at Hampton Sidney College. He then studied theology, and was ordained pastor of Fair Forest Church in April, 1793. The parents and grandparents were owners of slaves, many of whom were purchased at their own request to keep them from being sold to hard masters.

The Rev. William Williamson owned slaves both before and after his marriage, but he did not approve of slavery. In 1805 he removed to Adams County, Ohio, so that he might give his slaves their freedom. Mary Smith Williamson believed that negroes should be taught, and although it was against the law she taught a number of negroes to read and write. In so doing she incurred the hostility of many slaveholders in her husband's parish. This was the chief reason for their going to Ohio. They made the journey by wagon and horseback, taking along the four-year-old Thomas and his infant sister; and behind them in single file trudged twenty-seven slaves who were to be freed upon their arrival in a free state.

Thomas Smith Williamson inherited a deeply religious nature and a sympathetic attitude toward the unfortunate. On completing his elementary education he entered Jefferson College at Cannonsburg, Pennsylvania, graduating in 1820. He then began reading medicine with his brother-in-law, Dr. William Wilson of West Union, Ohio, and after a full course of this, and some practical experience, he took a course of lectures at Cincinnati, Ohio. He then entered Yale College for further medical training and graduated with his degree in medicine in 1824.

Dr. Williamson began his medical practice at Ripley, Ohio, and there met Margaret Poage, daughter of Col. James Poage, a man of wealth and position. On April 10, 1827, Dr. Williamson and Margaret Poage were married. Of her Rev. Stephen Riggs says, "Perhaps no man was ever more blessed with a helpmate more adapted to his wants than this lovely, quiet, systematic, cheerful Christian wife, who for forty-five years of perfect harmony encouraged him in his labors."

In Ripley Dr. Williamson built up a good practice, and in the course of time three children were born. But in the years that followed he became convinced that his life should be dedicated to the service of mankind and God through missionary work. At first it seemed unwise to plan on this, for there were three small children to be cared for. But, the Rev. Riggs states: "God removed this obstacle in his own way—by taking the little ones home to himself." Accepting this hard doctrine of the time, Dr. Williamson placed himself under the supervision of the Chillicothe Presbytery and studied theology, then entered Lane Seminary. He was licensed to preach in April, 1834.

Following this event he was directed to make a survey of the missionary field in the Indian territory, and in doing this journeyed as far west as Fort Snelling. In September of that year he was ordained a missionary, and in April, 1835, he set out with his wife and infant child, Miss Sarah Poage, and the Alexander Huggins family for Fort Snelling.

Dr. Williamson's first activity in Minnesota centered around the Pond mission and he presided at the organization of the First Presbyterian Church, which was the first one established in the territory.

When at Rock Island in 1835, Dr. Williamson had met Joseph Renville, French-Indian trapper and trader and man of many talents and interests. Upon learning that Dr. Williamson was again at Fort Snelling with a missionary party for the purpose of establishing a mission, Renville invited Dr. Williamson to come to Lac qui Parle and establish his mission near the Renville trading post there. This proposal received the endorsement of the authorities, and so on June 22, 1835, the missionary party set out from Fort Snelling in a mackinaw boat loaned by the fur company, to travel up the Minnesota River to Traverse des Sioux. They reached Traverse des Sioux on June 30, and there transferred to a Red River ox cart caravan for the rest of the journey to Lac qui Parle. Dr. Williamson's group traveled by team and wagon from Traverse des Sioux to Lac qui Parle, following the ox carts. The wagon had been taken apart and carried in the mackinaw boat from Fort Snelling to Traverse des Sioux, where it was assembled and put into use. Dr. Williamson drove the team from Fort Snelling along the Minnesota shore, and met the boat at Traverse, where the team was hitched to the assembled wagon. At the time of making this long and arduous journey Margaret Williamson was pregnant. Her son, John P. Williamson, was born October 27, 1835.

For the first year the Williamsons lived at the Fort, Dr. Williamson's first task was to familiarize himself with the Dakotah language. Although languages were difficult for him to learn, he mastered the language, and within two years was preaching to the natives in their own tongue. In 1836 the missionaries chose a site on the north bank of the Minnesota at Lac qui Parle and there began the construction of a mission with the help of Joseph Renville.

The Indians in the territory were very poor and received no annuities from the government. Dr. Williamson and Alexander Huggins set to work to teach better farming methods. Dr. Williamson also practiced medicine, and "for more than a quarter of a century his medical aid went hand in hand with his preaching of the gospel." (Rev. Riggs.) The Indians called the doctor pay-zhe-hoo-ta-we-chash-ta, or Grass Root Man.

Dr. Williamson also began the work of translating the Scriptures into the Dakotah language. In this work he had the help of Joseph Renville, who had been educated in a Jesuit School. But Renville spoke principally French and Dakotah, and so it was necessary for the doctor to learn French also. This, too, he mastered. When, in 1837, Rev. Stephen R. Riggs came to the misson the work progressed more quickly. The Pond brothers had already begun the compilation of a dictionary of Dakotah words, and they had this assistance. It was a work requiring great patience and devotion.

In 1846 the chief of the Kaposia band of Sioux went to the Indian Agent at Fort Snelling and requested that a missionary be assigned to his village. He was alarmed at the deterioration of the tribe due to the liquor furnished them by the saloon keepers of Pig's Eye and other bad influences. The Agent wrote to Dr. Williamson and asked him to take charge of a mission school at Kaposia. In his report of this to the government the Agent added, "He has had charge of the mission school at Lac qui Parle for some years; is well qualified, and is an excellent physician." So in November, 1846, Dr. Williamson moved to Kaposia and took up his work there. He also took a kindly interest in the white people at Pig's Eye, where there was a little log chapel. He was the first to promote education for the half breeds. At his request Miss Harriet E. Bishop came to teach in this, the first school house in Minnesota outside the mission schools.

When the Indians were summoned to a treaty conference at Traverse des Sioux, Dr. Williamson went with them as interpreter and friend. Since the treaty provided for the establishment of a new reservation along the Minnesota River, and agencies to be established at Redwood and Pehijutazi, Dr. Williamson immediately made plans to remove to this new location. In the summer of 1852, he went to Pehijutazi, which was to be the site of the Upper Sioux Agency, and began the construction of a home for his family. In the years that followed other mission buildings were erected, and Dr. Williamson and his staff—which included his own family, and relatives who also shared the missionary spirit, labored there until compelled to leave by the Sioux uprising. At the close of hostilities he did all he could to assist the imprisoned Indians in their prisons at Mankato and Fort Snelling.

After the uprising Dr. Williamson established a home at St. Peter, but he himself devoted two years to the Dakotahs who were imprisoned at Davenport, Iowa. He ministered to their needs, both spiritual and physical, and many Indians were taught to read and write and were instructed in Christian teachings. At the end of three years of imprisonment the Indians were deported to a new reservation at Crow Creek in what is now South Dakota.

The Rev. John Poage Williamson, first of the doctor's children to be born in the Indian territory, was now an ordained minister and missionary sharing his father's work. He accompanied one boatload of 1,300 Indians on the journey to Crow Creek, at the big bend of the Missouri River. Here he stayed for three years, sharing with the Minnesota Sioux the terrible hardships of the time, during which 300 Indians died of disease and starvation. Finally the government released them to a reservation called Santee, near the mouth of the Niobrara River in Nebraska where remnants of the Minnesota Sioux remain today. After the Indians were released, Dr. Thomas S. Williamson accompanied Rev. Riggs and Rev. John B. Renville to Santee, Nebraska, stopping on the way to preach at the Indian villages.

Dr. Williamson continued his work with the Indians for more than thirty-six years. When he was no longer physically able to travel his interest continued, and he attended synods as long as he was able. He continued to work on the Bible translation to the very end. Propped up in bed, he was still at work upon it in his home in St. Peter when death ended his labors on June 24, 1879.

Of the children born to Dr. and Mrs. Williamson, three died in infancy at Ripley, Ohio. Smith Burgess Williamson, "a manly boy," died as the result of an accident while hauling wood for the mission, at the age of fourteen or fifteen years. Lizzie Williamson Hunter died "in the flower of her young womanhood"; Nancy Jane Williamson, born at Lac qui Parle, died at the age of thirty-eight, in 1878. Margaret Poage Williamson, devoted wife and mother, died about 1876.

Dr. Williamson was survived by his son, the Rev. John Poage Williamson, who spent fifty-five years of his life as a missionary in Minnesota, Nebraska, and South

Dakota, and died, still in the service, at the Yankton Indian Agency at Greenwood, South Dakota, in October, 1917. His wife, Sara Van Nice Williamson, died in 1932. John was born in a hut with a dirt floor in Renville's stockade on October 27, 1835, possibly the first white child born in western Minnesota. His knowledge of the Sioux language, learned as a boy, was unsurpassed by any white missionary. He was in constant demand by the Federal government as an interpreter during the Sioux wars.

* * *

Nicolay Hilmar Scheldrup* stems from a famous family that produced outstanding men in Scandinavia from the 16th century on. Originally, the Scheldrup family lived in Denmark, where Bishop Jens Pederson Scheldrup was born in 1524. He must have been an outstanding, brilliant man. After studying philology at the University of Copenhagen he at one time became professor of Physics at this University. Then his interest shifted to theology and in 1557 became bishop of the Bergen territory. One of his sons, Peder Jensen Scheldrup, born in 1571, studied in Denmark and with the renowned ministers school at Wittenberg, Germany. First he was pastor in Fredriksborg and in 1622 he, too, became a bishop (for the Trondheim territory). One of Peder Jensen's sons, Nils Pederson Scheldrup was assistant pastor in Trondnes when he died in 1693. Anders Nilson liked the natural resources and acquired much land outside of Trondnes; there his widow and children settled after his death and the estate remained in the Tovik-Scheldrup family. One of Pastor Anders Nilson's sons, Peder Anderson Scheldrup, was born about 1690; he became district physician in Tovik—apparently the first physician of note in the Scheldrup family tree. Another son held a similar position in Harstad and a third son, Anders Pedersen, was a writer. The latter's son, Nils Anderson Scheldrup, was a shipowner and merchant in Tovik, and his grandson, Anders Martin Scheldrup, born about 1845, was the father of Dr. N. H. Scheldrup. Anders Martin also was a shipowner and merchant. Indeed, a line of ancestors of which to be proud. His mother was Hannah Wanvik.

Dr. Scheldrup finished high school in Norway and left for America in 1891. It is therefore not likely that he graduated from the University of Christiania. He studied medicine at Rush Medical College, from which he graduated in 1897, and later specialized in surgery. He was not the only one in his family to choose this specialty, as one relative became professor of surgery at the University of Oslo.

On August 5, 1907, he married Eva Dunsmore Rose; it was the second marriage for both.

For fifty years, he spent his summers at his summer home in Ontario and his hospitality became famous. For many years, he enjoyed his fine yacht and traveled with it a great deal. His interest, aside from medicine, included ornithology, astronomy, religion and history. He was a great reader and accumulated a fine large, library.

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*See "Medical History of the County of Yellow Medicine" by Mildred B. Lee. MINNESOTA MEDICINE, pp. 224-226, March, 1954.

Most of the information given herewith is taken from a clipping from an unidentified Norwegian newspaper which contained an article by Knute Haddeland: "Berømt Norsk Amerikaner 80 Aar."

Bibliography

1. Christianson, Theodore: Minnesota, the Land of Sky Tinted Waters, a History of the State and Its People. 5 volumes. Chicago and New York: American Historical Society, Inc., 1935.
2. Compendium of History and Biography of Central and Northern Minnesota, Containing a History of the State of Minnesota. Chicago: George A. Ogl, 1904.
3. Dirnberger, Mrs. Al: Echoes of '52.
4. Folwell, William Watts: History of Minnesota. 4 volumes. St. Paul: Minnesota Historical Society, 1921, 1924, 1926, 1930.
5. Helland, Andreas: Missionary John O. Dyrnes, M.D., p. 98.
6. History of the Minnesota Valley (Including the Explorers and Pioneers of Minnsota by Edward D. Neill, and History of the Sioux Massacre by Charles S. Bryant). Minneapolis: North Star Co., 1882.
7. Leonard, Joseph: Olmsted County History. P. 121. Chicago: Goodspeed Historical Association, 1910.
8. Minnesota Board of Commissioners on Publishing of Histories of Minnesota in the Civil and Indian Wars: Minnesota in the Civil and Indian Wars, 1861-1865. 2nd Edition. St. Paul: Pioneer Press Co., 1891.
9. Minnesota Historical Society Collections.
10. Riggs, Stephen R.: Mary and I. Forty Years With the Sioux. Chicago: W. G. Holmes, 1880.
11. Rose, Arthur: An Illustrated History of Yellow Medicine County. Marshall, Minnesota: Northern History Co., 1914.
12. Yellow Medicine County: Records; Statistics; Minutes of the County Board.

HEBREW MEDICAL JOURNAL ISSUES TWO TWENTY-SIXTH ANNIVERSARY NUMBERS

The *Hebrew Medical Journal* completed its twenty-sixth year by issuing two volumes in 1953 under the editorship of Moses Einhorn, M.D., of New York. Written in Hebrew, with English summaries, the journal has played an important part in the creation of a medical literature and terminology in the language of the Bible. Such a terminology represents an important linguistic and scientific achievement and materially accelerated the rise of a medical literature in Hebrew. To this literature, The *Hebrew Medical Journal* is also making substantial contributions.

Among the articles in the *Spring issue,* Volume 1, 1953, is the section on "Israel and Health." Dr. Meir Yoeli of the Hebrew University in Jerusalem presents a treatise on "Tropical Diseases in Israel." In his article, Dr. Yoeli emphasizes the growing trend of tropical diseases to spread to non-tropical countries as a result of population transfers. He also gives statistical studies on the recent incidence of tropical diseases in Israel, describes the special position of the country as being "on the cross roads of infection," and concludes with a report on recent progress in controlling those diseases in Israel. Another contribution of interest is the manuscript of a medieval Hebrew treatise on "Melancholy" (12th Century), with valuable notes and commentary, by Zussmann Muntner, M.D., of Jerusalem.

In the *Fall issue,* Volume 2, 1953, is the paper by Dr. Edward Tolstoi on "Treatment of Diabetes Mellitus by the Clinical Approach." In this number is included a symposium on "Artificial Insemination." Dr. Abner I. Weisman writes on "Some Consideration on the Status of Artificial Insemination in the United States of America," and Dr. Akiba Joel discusses "Artificial Insemination in Israel." Rabbi I. Jakobovits, Chief Rabbi of Ireland, gives a clarification on the attitude of Judaism, Catholicism and Protestantism towards the subject of "Artificial Insemination, Birth-Control and Abortion."

In the section called "Israel and Health," Dr. A. A. Schwarzbart deals with the increase in the number of tuberculosis cases, which followed the huge influx of newcomers from culturally and economically backward countries after the establishment of the State of Israel, and reports on the success achieved by the treatment of the malady with antibiotics.

✦ Reports and Announcements ✦

FAMILY DOCTORS' DAYS

at the

University of Minnesota Hospitals

All physicians are cordially invited to attend the following Family Doctors' Days which will be presented under the direction of the department indicated.

June 2—Dept. of Pathology

July 14—Ear, Nose, and Throat

Activities begin with luncheon at 12:15 noon with the staff of the department in the Hospital Dining Room and will continue throughout the afternoon.

For further information, write the head of the department concerned or the Director, Department of Continuation Medical Education, University of Minnesota Hospitals.

AMERICAN CONGRESS ON OBSTETRICS AND GYNECOLOGY

The sixth American Congress on Obstetrics and Gynecology will be held at the Palmer House, Chicago, December 13-17, 1954.

The five-day meeting is sponsored by the American Committee on Maternal Welfare, Inc., and the American Academy of Obstetrics and Gynecology. It will bring together the four major groups concerned in the provision of better care for mothers and babies—medicine, nursing, public health and hospital administration.

The program will include twenty-seven formal papers, twenty-two symposia and panels, luncheon discussion groups, and several hundred round-table discussions, covering every phase of maternal and newborn care. Scientific and technical exhibits are also being planned.

Information about the meeting may be obtained by writing to the Sixth American Congress on Obstetrics and Gynecology, 116 South Michigan Avenue, Chicago 3, Illinois.

AMERICAN CONGRESS OF PHYSICAL MEDICINE AND REHABILITATION

The 32nd annual scientific and clinical session of the American Congress of Physical Medicine and Rehabilitation will be held September 6-11, 1954, inclusive, at the Hotel Statler, Washington, D. C.

Scientific and clinical sessions will be given September 7, 8, 9, 10 and 11. All sessions will be open to members of the medical profession in good standing with the American Medical Association.

In addition to the scientific sessions, annual instruction seminars will be held. These lectures will be open to physicians as well as to therapists, who are registered with the American Registry of Physical Therapists or the American Occupational Therapy Association.

Full information may be obtained by writing to the executive offices, American Congress of Physical Medicine and Rehabilitation, 30 North Michigan Avenue, Chicago 2, Illinois.

Annual Essay Award

To stimulate interest in the field of physical medicine and rehabilitation, the American Congress of Physical Medicine and Rehabilitation will award annually a prize for an essay on any subject relating to physical medicine and rehabilitation. The contest, while open to anyone, is primarily directed to medical students, interns, residents, graduate students in the pre-clinical sciences and graduate students in physical medicine and rehabilitation.

The following rules and regulations apply to the contest:

1. Any subject of interest or pertaining to the field of physical medicine and rehabilitation may be submitted.
2. Manuscripts MUST BE in the office of the American Congress of Physical Medicine and Rehabilitation, 30 N. Michigan Ave., Chicago 2, not later than June 1, 1954.
3. Contributions will be accepted from medical students, interns, residents, graduate students in the pre-clinical sciences, and graduate students in physical medicine and rehabilitation.
4. The essay must not have been published previously.
5. The American Congress of Physical Medicine and Rehabilitation shall have the exclusive right to publish the winning essay in its official journal, the *Archives of Physical Medicine and Rehabilitation.*
6. Manuscripts must not exceed 5,000 words (exclusive of headings, references, legends for cuts, tables, etc.), and the number of words should be stated on the title page. An original and one carbon copy of the manuscript must be submitted.
7. The winner shall receive a cash award of $200, a gold medal properly engraved, a certificate of award and an invitation to present the contribution at the 32nd Annual Session of the American Congress of Physical Medicine and Rehabilitation at the Hotel Statler, Washington, D. C., September 6-11, 1954.
8. The winner shall be determined by the Annual Awards Committee composed of four members of the American Congress of Physical Medicine and Rehabilitation.
9. All manuscripts will be returned as soon as possible after the name of the winner is announced.
10. The American Congress of Physical Medicine and Rehabilitation reserves the right to make no award if, in the judgment of the Annual Awards Committee, no contribution is acceptable. The Congress may also award certificates of merit to contributors whose essays may be considered second and third best submitted. Announcement of the winner will be made after the annual meeting. Officers and members of the American Congress and the American Society of Physical Medicine and Rehabilitation are not eligible for this award.

SUMMER CAMP FOR DIABETIC CHILDREN

A summer camp for diabetic children will be opened for the sixth season under the auspices of The Chicago Diabetes Association, Inc., from July 18, 1954 to August 8, 1954 at Holiday Home, Lake Geneva, Wisconsin.

Physicians are requested to notify parents of diabetic children and to supply the names of children who would like to attend camp. Applications may be obtained from, and inquiries should be addressed to: The Chicago Diabetes Association, 5 South Wabash Avenue, Chicago 3, Illinois. Telephone: Andover 3-1861.

NATIONAL SOCIETY FOR CRIPPLED CHILDREN AND ADULTS

The 1954 convention of the National Society for Crippled Children and Adults, the Easter Seal Society, will be held November 3-5, at the Statler Hotel, Boston, Massachusetts.

Convention sessions now being planned, will be geared to the over-all theme of rehabilitation, with general sessions, institutes, seminars, workshops and roundtables on various specialized subjects.

In this thirty-first national convention of the Easter Seal Society, authorities in every area related to helping the crippled will participate. The 1954 convention will highlight the latest thinking and the newest practical developments in the various fields of work with the crippled. A traditional feature will be the Handicapped Panel, comprising persons who have successfully overcome major handicaps to lead useful lives.

The Easter Seal Society, the oldest and most comprehensive organization of its kind, extends 874 specific services and facilities to thousands of crippled persons each year. It does this through the efforts of 1,253 affiliates in the 48 states, the District of Columbia, Alaska, Hawaii and Puerto Rico.

COURSE IN CARDIOVASCULAR DISEASES

A course in cardiovascular diseases for general physicians was presented by the University of Minnesota and the Minnesota Heart association, March 22 to 24, in the University's Center for Continuation Study.

In conjunction with the course, Dr. Samuel A. Levine, clinical professor of medicine, Harvard university, presented the annual George E. Fahr lecture March 23, in Owre amphitheater. His topic was "Some Pitfalls in the Care of Cardiacs."

Director of the course was Dr. C. J. Watson, head of the University's department of medicine. On the faculty were staff members of the University medical school and the Mayo Foundation at Rochester.

Divisions of the course were "Examination of the Cardiovascular System," "Therapy of Congestive Heart Failure" and "Selection of Patients for Cardiac Surgery."

MINNESOTA SOCIETY FOR THE MENTALLY RETARDED

The fourth annual convention of the Minnesota Society for the Mentally Retarded will be held Friday and Saturday, June 4 and 5, at the Hotel Nicollet in Minneapolis. Chief speaker for the convention will be Dr. Salvatore Dimichael, newly appointed executive director of the National Association for Retarded Children, who will fly from New York to speak at the Saturday noon banquet. Dr. Dimichael's topic will be: "The Present and Future in a National Program for the Mentally Retarded." Before joining the NARC early this year, Dr. Dimichael was with the Federal Office of Vocational Rehabilitation in Washington, D. C.

Registration for the convention begins at noon, Friday, June 4. The afternoon will be spent in visits to various schools, workshops and clinics which work with the retarded. A reception from six to seven p.m. will give conventioneers a chance to meet with one another. From seven to nine p.m. there will be workshops in the areas of education for the retarded, publicity and public relations, legislation, fund-raising, and expanding horizons through new group organization. Business sessions will occur Saturday morning from nine to twelve.

Of especial interest to professional persons, as well as to parent-members of the Minnesota Society, will be three forums from 2:30-4:30 Saturday afternoon. These forums will consider: (1) the education of the retarded child, (2) institutional care, and (3) care of the retarded child in the home. Prominent persons from the fields of education and welfare, as well as parents, will analyze the problems in these three areas and discuss solutions, after which discussion from the floor will follow.

Mr. Arthur Gunderson, 821 Summit, Minneapolis, 1953-1954 president of the Minnesota Society for the Mentally Retarded, extends a cordial invitation to all medical, welfare and institutional personnel, teachers, future teachers and all friends of the retarded to attend the convention.

MINNESOTA ACADEMY OF OCCUPATIONAL MEDICINE AND SURGERY

The annual meeting of the Minnesota Academy of Occupational Medicine and Surgery will be held at 6:30 P.M. on May 26, at the Minneapolis Athletic Club.

MINNESOTA SOCIETY OF NEUROLOGY AND PSYCHIATRY

The Minnesota Society of Neurology and Psychiatry held its March meeting at the Town and Country Club in Saint Paul. Dr. Burtrum C. Schiele, Minneapolis, and Dr. Philip M. Margolis, Saint Paul, spoke on "Clinical Experience in the Use of Succinylcholine Chloride in Electroshock Therapy."

In Memoriam

VERNE SEYMOUR CABOT

Dr. Verne S. Cabot, Minneapolis surgeon, died at the age of sixty-four, March 9, 1954, at his home in Minneapolis. He had been absent from his office in the LaSalle Building since last August, but had planned to return to active practice soon.

Dr. Cabot had practiced medicine in Minneapolis since 1913. He was born at Faribault and received his medical degree from the University of Minnesota. He served his internship at Minneapolis General Hospital and took postgraduate training in surgery at the North Chicago Hospital, as assistant to Dr. Carl Beck, and at Cook County Hospital in Chicago. He served for two years during World War I in the Surgical Service of the United States Army Medical Corps in France. He was a member of the staffs of Deaconess, Swedish, and St. Barnabas Hospitals in Minneapolis, and served as Chief-of-Staff of St. Barnabas.

Among the professional organizations of which Dr. Cabot was a long-time member are the Hennepin County Medical Society, the Minnesota State Medical Association and the American Medical Association. He was also a member of the American College of Surgeons, of which he was a Fellow, and of the Minneapolis Surgical Society, the Professional Men's Club and the Business and Professional Men's American Legion Post.

He was one of three brothers, all of whom were physicians and one of whom, Dr. Clyde M. Cabot, survives him. A brother-in-law, Dr. Arthur A. Wohlrabe of Minneapolis, is also a physician.

Other survivors beside Dr. Clyde M. Cabot, are his wife, the former Emma M. Miner; a daughter, Mrs. Charles D. Hellberg; a son, John S.; three grandchildren; and a sister, Mrs. Arthur A. Wohlrabe.

THEODORE A. ESTREM

Dr. Theodore A. Estrem, seventy, died April 8, 1954, after a long illness at his home in Hibbing. He went to Hibbing thirty-five years ago as member of the staff of Rood Hospital. Later he became affiliated with the Mesaba Clinic, specializing in urology and in obstetrics and gynecology.

Dr. Estrem was born at Petersburg, Nebraska, and received his premedical education at the Jewell Lutheran College and at the Red Wing College and Seminary. He was graduated in medicine from the University of Wisconsin and Rush Medical College and served his internship at St. Luke's Hospital in Chicago.

He served a term as chief of staff of the Hibbing General Hospital and he was a member of the St. Louis County Medical Society, the Minnesota State Medical Association and the American Medical Association. He was also a past president of the Hibbing Chamber of Commerce and the Rotary Club and a member of Mesaba Lodge 255 A.F. and A.M., the Algonquin and Hibbing Curling clubs and of Our Savior Lutheran Church of Hibbing.

Surviving are his wife, Violet; a son, Theodore A., Jr., Minneapolis; a daughter, Mrs. W. F. Braasch, also of Minneapolis; a brother, Herman, of Albion, Nebraska; six sisters including Mrs. S. Watland, San Francisco; Mrs. C. Radcliff, Olive View, California; Mrs. John Levos, Norfolk, Nebraska; Mrs. R. Levos and Mrs. O. Limbo, both of Oakdale, Nebraska, and Mrs. George Karabel, Tilden, Nebraska; and two grandchildren.

ALCIBIADES A. GIROUX

Dr. Alcibiades A. Giroux, North Mankato pediatrician, died March 11, 1954, at St. Joseph's Hospital in Mankato at the age of sixty-nine.

Dr. Giroux had never recovered completely from an automobile accident, which occurred several years ago, and had been forced to retire from practice as a result of the shock produced by the floods of 1951 which did serious damage to his home in North Mankato. He had practiced in Mankato since 1944 and, before that, in Duluth for many years and, for a short period, at Moose Lake Hospital.

Dr. Giroux was born at Granby in Canada. He received his pre-medical training at St. Joseph's College in Montreal, and his medical education at the University of Montreal Medical Department. He served his internship at St. Mary's Hospital in Duluth and took his postgraduate training in pediatrics at the University of Minnesota Medical School.

He was a member of the Nicollet-LeSueur County Medical Society, the Minnesota State Medical Association, and the American Medical Association.

Surviving are his wife, Katheryn, North Mankato; one daughter, Mrs. Lee Mockrud, Chicago; three sons, Glen, Donald and Richard, and four grandchildren, all of Mankato.

LOUIS WILLIAM KATZBERG

Dr. Louis William Katzberg, fifty, died at Rice Hospital in Willmar, Minnesota, March 17, 1954. He had been staff physician at Willmar State Hospital since 1951.

Dr. Katzberg was born in Adams County, Nebraska, received his pre-medical education at Hastings College, Hastings, Nebraska, and his medical training at the University of Nebraska College of Medicine in Omaha. After a year's internship at Bishop Clarkson Memorial Hospital in Omaha and a short period in practice at Pine Island, Nebraska, Dr. Katzberg moved to Minnesota and became staff physician at the Fergus Falls State Hospital from 1935 to 1939. From 1939 until he went to the Willmar institution in 1951, he practiced at Spring Valley, Minnesota.

Dr. Katzberg was a member of the Minnesota State Medical Association, the American Medical Association and the Kandiyohi-Swift-Meeker County Medical Society.

Survivors include his wife, the former Mabel Richards of Omaha; three daughters, Ann, fifteen, Carol, fourteen, and Sue, twelve; his parents, Mr. and Mrs. Gus Katzberg of Prosser, Nebraska; and a brother, Ralph, also of Prosser.

JOHN RICHARD PETERSEN

Dr. John Richard Petersen, for thirty-five years medical director of the Lutheran Brotherhood Insurance Company, died in Minneapolis, April 4, 1954. He was eighty-two years old.

Dr. Petersen was born in Minneapolis and graduated from Luther College at Decorah, Iowa, and the University of Minnesota Medical School. He had practiced at Willmar and at Renville before joining the insurance company.

He was a life member of the Hennepin County Medical Society, the Minnesota State Medical Association and the American Medical Association. He was associated, also, with the Athletic and Old Guard clubs.

Surviving are his wife, Thea; a son, Richard L. of Park Rapids; a daughter, Mrs. L. S. Carroll, Minneapolis; a sister, Mrs. Clara Ulvilden, Minot, North Dakota; and one granddaughter.

FRANK THOMAS RODWELL

Dr. Thomas Frank Rodwell died at his home in Mahnomen, April 22, 1954, at the age of ninety-three. He was a member of the Fifty Club of the Minnesota State Medical Association and had been honored last year by townspeople as the oldest living Legionnaire in Minnesota.

Dr. Rodwell served for twenty-eight years in the U. S. Indian Service as physician and superintendent at Walker and at White Earth. He moved to Mahnomen to engage in private practice in 1930. A reference to him in Polk's Medical Directory of 1898 indicates that he had been established there before. He continued to practice in Mahnomen on a limited basis until a few years before his death.

Dr. Rodwell was born at Hamilton, Ontario, and received his preliminary education at the Collegiate Institute at Hamilton and at Michigan College (now Wayne University), Detroit, from which he received his medical degree in 1886. He served his internship at Harper Hospital in Detroit and took seven years of postgraduate training at the Hospital of Lumber Casualties at Saginaw, Michigan. He came to Minnesota in 1893.

He was a member of the Red River Valley Medical Society until the time of his retirement, as well as the Minnesota State Medical Association and the American Medical Association. He served in the Army Medical Corps during World War I.

Survivors include his wife, the former Mayme Holden of Brainerd, whom he married in 1904 and two sons, Thomas, of Granite Falls, and Harold, of Mahnomen.

ANTON G. WETHALL

Dr. Anton G. Wethall, Minneapolis urologist, died April 4, 1954, at the age of eighty-two. He had practiced in Minneapolis for more than fifty years and had maintained offices in the LaSalle Building there until a year ago.

Dr. Wethall was born in Norway but came to America with his parents seventy-six years ago, settling in Stoughton, Wisconsin. He received his pre-medical education and a B.S. degree from Valparaiso University in Indiana and his medical training at the University of Minnesota. He interned at Deaconess Hospital and has been on the staff there ever since. He was also a member of the staffs of St. Andrews and Mt. Sinai Hospitals. He took postgraduate work in urology at the New York Post-Graduate School in 1913 and again in 1919, and at the University of Vienna in Austria in 1927.

From 1911 to 1917, Dr. Wethall was clinical assistant in urology at the University of Minnesota. He served as staff instructor from 1917 to 1928 and as assistant professor from 1928 to 1941.

He was a life member of the Hennepin County Medical Society, the Minnesota State Medical Association and the American Medical Association; also he was a member of the American Urology Association, Sons of Norway and the Central Lutheran Church.

Surviving are his wife, Amanda J., and two daughters, Mrs. Clarence A. Kuntz and Mrs. John G. Gustafson, both of Minneapolis.

ARCHA EDWARD WILCOX

Dr. Archa E. Wilcox, surgeon and leader in industrial medicine, died at his home in Minneapolis, April 6, 1954. He was seventy-eight years old.

For thirty-nine years until his retirement in 1941 he was a prominent figure in civic and welfare affairs in Minneapolis, as well as in the practice of medicine. As medical director of the Washburn-Crosby Company, he established the first industrial medical department in this area. He was for some years a member of the Minneapolis Board of Charities and of the Welfare Board. For a short time, also, he was superintendent of Minneapolis General Hospital and he served as chief of the surgical service and head of the medical staff for ten years.

Dr. Wilcox was born in Minneapolis and attended East High School there. He received pre-medical training at the University of Buffalo and his medical education at the University of Pennsylvania. Following internship at the German Hospital in Philadelphia, he took postgraduate training in surgery under the late Dr. John B. Deaver and in Europe. He established himself in medical practice in Minneapolis in 1902, and taught surgery, first at Hamline University and then at the University of Minnesota, where he was associate professor emeritus of surgery at the time of his death.

Dr. Wilcox served as chairman of the Committee on Industrial Health of the Minnesota State Medical Association and he was also a member of the medical panel for occupational diseases for the State of Minnesota.

He was one of the organizers of the former Hillcrest Hospital and he served as chief of staff at both the Abbott and Eitel Hospitals. He was elected president of the Minnesota Academy of Medicine in 1934 and 1935. He was a fellow of the American College of Surgeons and member of the Minneapolis Surgical Society, the Southern Minnesota Surgical Society and the Western Surgical Society. He was a life member, also, of the Hennepin County Medical Society, the Minnesota State Medical Association and the American Medical Association. He belonged to the Minneapolis Club and to the Minneapolis Institute of Arts. During World War I, he was in the reserve officers corps. During World War II, he served on the selective service appeal board.

Music was one of his hobbies. He was a skilled pianist and entertained his friends often at his summer residence, Rippleside, on Elm Creek near Anoka, during his more active days.

Surviving are his wife, the former Mary Hays Hunter; two children, Archa Edward Wilcox, Jr., of Minneapolis, and Mrs. G. Chester P. Gifford, Houston, Texas; two sisters, Mrs. Myrtice Joslin and Mrs. Beatrice Keator, both of Excelsior, and two granddaughters.

GERMANY SHOWS SURPLUS OF DOCTORS

(Continued from Page 351)

"First, the public health insurance law allots one physician to 600 patients under public insurance, and one dentist to 1,250 patients. This allocation of doctors and dentists automatically restricts the number who may practice in the public health insurance field. Further, permission to treat public health insurance patients is not given to all physicians admitted to practice.

"Finally, because of the 'splitting-the-melon' type of payment to the doctors participating in the public insurance scheme, the doctors in the plan are encouraged to oppose any increase in the numbers dividing the lump sum of payments."

In the insurance plan, half of the premium payment is taken out of the wage-earner's pay, the other half is paid by the employer. In addition, a patient pays about 10 cents for a "sick ticket" when he wishes to see a doctor. The article states:

"This means that eight of every 10 patients enter doctors' offices with 'tickets.' The remaining two patients visit private practitioners. However, private practitioners cannot count on drawing two of every 10 patients. Since these latter are presumably well off, at least one of them will take his medical business to a 'professor,' not to a mere 'private practitioner.' The 'professor,' peculiar to German medical practice, is a specialist who, besides drawing a fixed income at the university and possibly as head of a hospital as well, will put his outstanding knowledge and ability at the disposition of a few private patients . . ."

The situation is aggravated by the fact that there are many refugee physicians from German territory behind the Iron Curtain. The refugee medical men also demand—and rightly so, the West German government feels—a chance to establish a private medical practice.

MINNESOTA STATE BOARD OF MEDICAL EXAMINERS

230 Lowry Medical Arts Building
Saint Paul, Minnesota

J. M. Jones, M.D., Secretary

BLOOMINGTON WOMAN GIVEN THREE YEARS PROBATION ON ABORTION CHARGE

Re: State of Minnesota vs. Carolyn Lillian Lilledahl

On March 25, 1954, Mrs. Carolyn Lillian Lilledahl, 37, 9949 Maple Ave., Bloomington, Minnesota, was sentenced by the Hon. Levi H. Hall, Judge of the District Court of Hennepin County, to a term of not to exceed four years in the Women's Reformatory at Shakopee, following her plea of guilty on February 25, 1954, to an information charging her with the crime of abortion. However, Judge Hall stayed the execution of the sentence for a period of three years and placed the defendant on probation during that time.

The defendant Lilledahl did not actually perform the abortion but received $35 of the $200 that was paid for it for acting as the contact between an alleged nurse and the twenty-nine-year-old unmarried Minneapolis woman who submitted to the illegal operation on February 18 and was hospitalized in Minneapolis two days later due to the after-effects of the abortion. Although the abortionist has not been identified, she has been described as being a registered nurse, single, rather attractive, 27 years of age, 5 feet in height, 120 pounds, and having a medium complexion and reddish brown hair, her first name being Marie.

When Judge Hall sentenced Mrs. Lilledahl, who has no license to practice any form of healing in Minnesota, he pointed out that there was no excuse for her becoming involved in an abortion in any manner whatsoever since she knew that her mother was presently serving a sentence for the same offense. Lillian Mullane, the defendant's mother, was sentenced by Judge Hall on October 2, 1953, to a term not to exceed two years in the Women's Reformatory at Shakopee on an abortion charge. While the defendant Lilledahl claims that she does not know the identity of the abortionist her story in that respect is not believed by the authorities.

◆ Of General Interest ◆

Dr. H. A. Wilson, Veterans' Administration Hospital, has been released from the hospital in Spokane, Washington, where he has been recovering from a heart attack suffered while on a hunting expedition. He will be confined to his home for several more months before he will be able to take up his professional duties again.

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The "natural increase"—gain in births over deaths—has been 150,776 since 1950-51, **Dr. A. J. Chesley,** head of the state health department, reported. Minnesota is well past the 3 million population mark, with a gain of more than 100,000 the last three years, raising the total to 3,089,904, according to state department of health figures.

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Dr. J. Garott Allen, professor of surgery at the University of Chicago School of Medicine, spoke at a course in emergency surgery for general physicians at the University of Minnesota April 1-3. Emergency management of various types of wounds and injuries and acute abdominal conditions were stressed. **Dr. O. H. Wangensteen,** chairman of surgery at the University and director of the course, and **Dr. Leo G. Rigler,** head of radiology at the university, led a symposium on intestinal obstruction, April 3. Dr. Allen also delivered the annual Clarence M. Jackson lecture April 1 on "The Management of Acute Upper Gastrointestinal Hemorrhage."

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Dr. C. W. Truesdale, Glencoe, attended the New Orleans Graduate Medical Assembly in New Orleans, Louisiana, in March.

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Dr. Roy H. Good became associated with Dr. Paul Swedenburg, Glenwood, in medical practice on March 1, 1954. Dr. Good is a recent graduate of the University of Minnesota Medical School and previously practiced in southern Minnesota. He is a veteran of three years' service with the United States Navy.

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Dr. Roy Burt, Anoka, spoke to the members of the Cambridge P.T.A. on March 15. His topic was "Mental Health." Dr. Burt is a member of the executive board of the Minnesota congress of parents and teachers, and is chairman of its human relations and citizenship committee.

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Dr. Robert N. Barr, deputy executive health officer of the Minnesota health department, spoke on "Rehabilitation" at the Midway Lions club meeting on March 15.

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Dr. Donald B. Simonson was elected chief of Fairview hospital medical staff at the March staff meeting. Other officers elected were: **Dr. Rudolph W. Koucky,** vice chief; **Dr. Harry B. Hall,** secretary; and **Dr. Maurice G. Gilbert,** treasurer. The executive committee includes the officers and **Doctors Donald B. Frand, Richard C. Gaard, Harold R. Leland, Glenn L. Peterson** and **Ragnar T. Soderlind.**

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Dr. Robert N. Barr, University state health department, was the guest speaker at the Women's section of the Industrial Safety school in Duluth.

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Dr. Warren L. Kilmer, International Falls, left the first part of March to accept a surgical residence fellowship in a Chicago hospital. The fellowship is for a four-year period, and in addition to his residency he will be employed as a physician by the United States Steel Corporation in Gary, Indiana.

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Dr. C. W. Rumpf, Faribault, recently returned from Korea, where he was Deputy Surgeon of the Eighth Army, was the guest speaker at the annual Father and Son banquet at the St. Luke Evangelical and Reformed Church on February 22.

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The story of Swift county's tuberculosis accreditation was related in the March issue of *Everybody's Health*, state Christmas Seal magazine. Pictured in the magazine were residents of Swift County who participated in the ceremony. Speaker at the accreditation, **Dr. L. S. Jordan,** superintendent of Riverside Sanatorium, Granite Falls, made special mention of the tuberculosis control work done by the late Dr. C. L. Scofield of Benson, a pioneer in tuberculosis work in Minnesota.

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Dr. Carl J. Potthoff, Rochester, was one of the featured speakers at the seventh annual interim session of the Montana Medical Association in Helena, March 4 and 5. Dr. Pothoff spoke on "Recent Developments Toward a Vaccine Against Poliomyelitis."

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On Tuesday, February 16, **Dr. R. V. Williams,** Rushford, marked the fiftieth anniversary of medical practice. Dr. Williams is a graduate of Northwestern University, Evanston, Illinois. He spent his internship at Cook County Hospital, Chicago. In February, 1904, he went into practice in Rushford.

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Dr. Agnes Marie Hoeger, medical missionary to New Guinea, now studying public health at the University of Minnesota, addressed the members of Alpha Epsilon Iota, medical sorority at a noon meeting on March 9.

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Inability of state hospitals to hire physicians and psychiatrists because of a shortage of doctors was one of the problems that confronted legislators, hospital officials and others that participated in a mental health conference in Minneapolis, on March 18. The conference was called by **Dr. Ralph Rossen**, superintendent of Hastings state hospital and Minnesota's first mental health commissioner.

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Dr. W. F. Maetz has been appointed to the medical staff of the Anoka State Hospital; he assumed his duties March 1. He practiced in New Prague until about ten years ago when he moved to Saint Paul.

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Dr. Kristofer Hagen, who has been associated with Dr. Theodore Greenfield at the Cokato Hospital since September, 1952, left in March to open a practice in Richfield. Dr. Hagen went to Cokato after serving as a medical missionary in India. Previously he had served with the army in Europe.

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Dr. Richard A. DeWall, Anoka, has left Mork's Clinic to do research work at the University of Minnesota hospital.

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The Minnesota Heart association presented a course in cardiovascular disease in the University of Minnesota Center for Continuation Study, March 22-24. **Dr. Samuel A. Levine,** clinical professor of medicine, Harvard University, presented the annual George E. Fahr lectures. His subject was "Some Pitfalls in the Care of Cardiacs."

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Dr. Paul M. Smith, Lake Crystal, enrolled in a four weeks' course in surgery at the Cook County Medical School in March.

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Dr. O. M. Odlund, Granite Falls, will spend Tuesday and Friday afternoons at the Maynard Clinic, according to arrangements completed between the Maynard Clinic Board and members of the Granite Falls Clinic staff.

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Dr. R. E. Risch has opened a new office building at 2915 Johnson St., N.E., Minneapolis. Having been in practice at 2854 Johnson St., N.E., since 1936, he now has an associate with him, **Dr. Paul V. Cummiskey,** Mankato. Dr. Cummiskey was graduated from the University of Minnesota. He practiced in Fresno, California and in Graceville, Minnesota, before joining Dr. Risch.

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Dr. J. S. Blumenthal spoke to the dietitians at the Continuation Center, Friday, March 5, on "Diets in Allergic Diseases."

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Dr. Merrill D. Chesler, Minneapolis, has announced the removal of his offices to 600 Physicians and Surgeons Building. His practice is limited to Plastic and Reconstructive Surgery.

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The management of common medical problems involving the eyes, ears and upper respiratory tract were stressed at the course for general physicians at the Continuation Center. The course was under the direction of **Dr. L. R. Boies,** head of the department of ophthalmology and otolaryngology, and **Dr. Erling W. Hansen,** director of the division of ophthalmology.

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Dr. Howard M. Frykman, Minneapolis, spoke on the topic "Office Proctology" at the monthly meeting of the Chippewa County Medical Society held at Chippewa Falls, Wisconsin, on March 9.

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Dr. J. R. Balach has been named chairman for the business division for West Duluth for the American Red Cross Drive.

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Dr. William B. Martin, Rochester, addressed the St. Louis County Medical Society. He spoke on atrio-septal defects.

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Veterans administration officials have asserted emphatically that the Fort Snelling, St. Cloud, Fargo and Minot hospitals, among other VA hospitals, are on the national accredited list. The statement was made after the VA hospitals were omitted from the annual published listing. Phil Fordyce, VA director of public relations at Fort Snelling, telephoned Dr. Edwin Crosby, Chicago, director for the joint commission of accreditation, regarding the omission. Dr. Crosby explained that the VA hospitals are listed separately in the back of the accreditation book and were thus inadvertently overlooked. The explanation came after VA hospital doctors protested the omission.

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Dr. C. G. Sheppard and **Dr. Elmer W. Lippmann,** Hutchinson, left by plane March 21 for Cleveland, where they attended the annual meeting of the American Academy of General Practice.

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The Rochester state hospital announced in March that it had received a $31,000 grant from the Louis W. and Maud Hill Family foundation, Saint Paul, for a statewide two-year study on the prediction and control of Huntington's chorea. The study will begin July 1, under the direction of **John S. Pearson,** the hospital's chief clinical superintendent, and **Dr. Jorde A. Lazarte,** assistant superintendent. Faculty members of the University of Minnesota and the Mayo foundation will assist.

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Dr. William P. Shepard, vice president in charge of health and welfare for the Metropolitan Life Insurance Company, will speak at the Middle States Public Health association meeting in the Hotel Saint Paul, June 2-4.

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The alumni of the Cyrus Fishermen's College, of which **Dr. Herman Linde** is the dean, held its sixteenth annual banquet at Cyrus, on April 30. Dr. O. J. Jerde, St. Cloud State Teachers College, was the guest speaker.

Drs. Walter H. Ude, C. N. Borman and **Eugene E. Ahern,** Diplomates of the American Board of Radiology, announce the opening of enlarged new offices at 250 Medical Arts Building, Minneapolis. They will continue to maintain the offices at 701 LaSalle Building.

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Dr. H. O. McPheeters, Minneapolis, recently spent the month of March in the Hawaiian Islands and reported a very fine vacation. While there he spoke before the Honolulu County Medical Society in Honolulu and the Maui County Medical Society on the Island of Maui, on the treatment of varicose veins.

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Dr. Alan Challman, Minneapolis, has been selected by the Surgeon General, U. S. Army, for a thirty-day tour in the Far East, as a civilian Consultant in Psychiatry. He left April 6 and will return about the middle of this month. The tour includes visits to military installations in Japan, Korea, Okinawa, and Hawaii, for the purpose of improving the quality of medical care and instruction.

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Dr. John W. Schumacher, Minneapolis, spoke to an adult education class at the Y.W.C.A. in Minneapolis, on "Common Misconception in Psychiatry" on March 15. Following the talk there was a question-and-answer period, at which time the various members asked questions about Psychiatry and its relationship to medicine.

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Dr. Michael Danyluk, on April 1, 1954, became associated in the general practice of medicine at 1518 East Lake Street, Minneapolis, with **Dr. S. M. Lagaard.** On July 1, 1954, Dr. Danyluk will take over the entire practice since Dr. Lagaard will then specialize in the field of orthopedic surgery.

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Dr. H. S. Diehl, dean of the medical sciences at the University of Minnesota, has been selected as one of three United States delegates to the annual assembly of the World Health Organization to be held in Geneva, Switzerland, May 4-22. Accompanied by Mrs. Diehl, Dean Diehl will fly to Geneva. The other two delegates to the assembly will be Dr. Leonard A. Scheele, United States surgeon general, and Dr. Chester S. Keefer, assistant secretary for health and medical affairs of the United States Department of Health, Education and Welfare.

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Dr. and **Mrs. H. W. Meyerding,** Rochester, left March 29 for a two-month trip to South America. They will visit many of the places they toured in 1950. En route they attended the dinner of the International College of Surgeons in New York. Their first stop was Trinidad, then the Isle of Barbados, Bahai, Rio de Janeiro, Santos, Sao Paulo, Montevideo and Buenos Aires will be visited before returning to Santos and Sao Paulo where Dr. Meyerding will speak before the Brazilian and Argentine Orthopedic Society of which he is a member.

Dr. Albert V. Stoesser, Minneapolis, spoke on "Eczema and Urticaria" and on "Incidence and Treatment of Headaches Due to Allergy in Children" at the meeting of the American College of Allergists held in Miami Beach in April. He also took part in a round-table discussion on "Pitfalls in the Diagnosis and Management of Asthma."

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Dr. and **Mrs. Titus C. Kreuzer,** Marshall, recently returned from a six weeks' vacation in Mexico. While in Mexico City Dr. Kreuzer attended the Pan American Congress of Otorhinolaryngology and Bronchoesophagology. They also visited Cuernavaca and Guadalajara. Dr. Kreuzer had previously become acquainted with Drs. Luis Martinez, Jr. and Jose Sanchez Cortes, heads of the departments of Ophthalmology and Otorhinolaryngology of the Guadalajara University School of Medicine. They requested Dr. Kreuzer to demonstrate his bloodless tonsillectomy. He performed six tonsillectomies before the Academy of Ophthalmology and Otorhinolaryngology and for ten days instructed the residents in the technique.

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Dr. Owen H. Wangensteen, recognized as one of the world's outstanding leaders in the field of cancer surgery and the early diagnosis of cancer, addressed a dinner meeting in Winona on April 12, in observance of Cancer Crusade Month. His topic was "In This Great and Urgent Cause."

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Dr. Russell L. Cox has recently been added to the staff of the Jackson Medical Center, Jackson. Dr. Cox is a radiologist and makes his home in Estherville, Iowa. He will be at the Medical Center in Jackson on Tuesdays and Fridays.

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Drs. E. J. Baldes and **Jan Tillisch,** Rochester, attended the meeting of the Aero Medical association in Washington, D. C. Dr. Baldes is a member of the executive council and Dr. Tillisch is on the committee on aviation medical training.

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Dr. M. B. Dockerty, Rochester, was on a tour of duty as resident consultant for the Armed Forces Institute of Pathology in Washington, D. C. He gave a demonstration and a lecture at the institute and also gave a demonstration at the Walter Reed General Hospital. During his stay in Washington Dr. Dockerty was the guest speaker at a meeting of former fellows of the Mayo Foundation. He spoke on New Developments at the Mayo Clinic.

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On April 1, 1954, **Dr. J. P. Grahek,** Ely, purchased the partnership interest of **Dr. H. N. Sutherland** in the Shipman Hospital and Clinic. Simultaneously Dr. Grahek announced that two doctors have accepted positions with the hospital staff. It is anticipated that two more doctors will join the staff about July 1, bringing the total to five, Dr. Grahek stated. First of the new staff members, is Dr.

Frederick Colosey, Hartford, Connecticut. He received his pre-medical training at Trinity College in Hartford and then continued his training at Temple University in Philadelphia, where he received his medical degree. He served his internship in Frankfort Hospital in Philadelphia. The second new staff member is **Dr. Edward W. Ciriacy,** Philadelphia, Pennsylvania, who also received his medical degree from Temple University. He later completed a post-graduate course in surgery techniques and procedures at Frankfort and Temple Hospitals. Following his internship at Frankfort Hospital, he served on the staff of the Chestnut Hill Hospital which also is located in Philadelphia.

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Dr. William A. Jordan, Minnesota Department of Health, spoke on the new fluoride treatment for the prevention of tooth decay and showed pictures to illustrate this work at the Parent Teachers Association meeting in Lafayette in March.

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Research in the field of heart trouble was outlined to members of the Exchange Club at their weekly meeting in Hotel Saint Paul by **Dr. Ivan G. Baronofsky,** associate professor of surgery at the University of Minnesota. Dr. Baronofsky, also director of post-graduate surgical education at Ancker Hospital, reviewed what has been done to date on the problem of heart disease and latest surgical technique.

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Dr. Ralph E. Smith, Department of Medicine at the Veterans and University Hospitals, Minneapolis, was the guest consultant discussing valvular heart disease at the March meeting of the McLeod County Medical Society in Glencoe.

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Dr. Arthur N. Antonow has joined the staff of the East Range Clinic as obstetrician and gynecologist. He and his family have moved to Virginia from Highland Park, Illinois, where he was associated with Dr. A. F. Lash in the practice of obstetrics and gynecology. He was trained at the Cook County Hospital and Chicago Maternity Center and was also a member of the staff at Michael Reese Hospital in Chicago.

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Dr. Robert W. Bachmeyer, Canton, Ohio, hospital administrator, is the choice of St. Barnabas hospital board to succeed **Dr. Karl S. Klicka,** who has resigned to become administrator of Presbyterian Hospital, Chicago. Dr. Bachmeyer has been administrator of Aultman Hospital in Canton since 1947. He served his residency in hospital administration at St. Luke's Hospital, New York, and then was assistant director at the Hospital for Special Surgery, New York, until 1942, when he entered military work with the United States public health service. Following his discharge from the army, he became assistant director of Children's Hospital in Boston, before going to Aultman.

Thirty-four fellows, including twelve from foreign countries, began studies with the Mayo Foundation, Rochester, for the spring quarter.

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Dr. J. Burns Amberson, New York, world authority on chest diseases, delivered the annual John W. Bell tuberculosis lecture in Minneapolis April 5. **Dr. John H. Moe,** Hopkins, president of the Hennepin County Medical Society, presided over the meeting. The John W. Bell lecture is co-sponsored by the Hennepin County Medical Society and the Hennepin County Tuberculosis Association. The Bell lecture brings outstanding tuberculosis authorities to Hennepin County.

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Dr. William F. Maloney, acting assistant dean of the Medical School at the University of Minnesota, was guest consultant at the first of two scheduled clinical conferences held for the medical staff of the Itasca County Memorial Hospital in Grand Rapids on April 14. Dr. Maloney presented case histories of patients with heart diseases. He also discussed with the medical staff some of the recent techniques and methods being employed in the diagnosis and treatment of cardiac disorders.

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Lieutenant (jg) John E. Indihar, son of **Dr.** and **Mrs. J. E. Indihar,** Chisholm, has been assigned to the Oakland, California, Naval Supply Center. The twenty-eight-year-old physician was called to active duty in the Navy last September. He had been serving at the Naval Medical Center, Bethesda, Maryland, until his assignment to Oakland. Dr. Indihar was engaged in general practice in Ely and Virginia before he entered the service.

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Dr. Harold J. Walder, Duluth clinic urologist, spoke on the increase in certain types of cancer in the Duluth area at the noon meeting of the Rotary Club, April 8.

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Dr. Clyde A. Undine, Minneapolis, attended the National convention of the American College of Physicians held in Chicago, April 5-9.

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Dr. Paul Winchell, department of medicine at the Variety Club Heart Hospital, was guest consultant at the first of two clinical conferences held for the medical staff of St. Mary's Hospital in Detroit Lakes, on April 14. Dr. Winchell is doing research on various disease conditions of the heart and is well qualified to evaluate some of the techniques and methods now being used in diagnosis and treatment of heart diseases.

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Minneapolis Veterans Administration Hospital will take part in a co-operative research program with other VA hospitals to provide more accurate standards for diagnosing and treating heart and lung diseases. **Dr. John A. Seaberg,** hospital manager, said measurement of heart and lung functions has been carried on at the hospital for several years. Studies at the Minneapolis hospital are being conducted by **Dr. Ralph Smith**, cardiology service, and **Dr. Frank Martin**, tuberculosis service.

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Dr. Rudi Schmid, University of Minnesota instructor in medicine, has been awarded a special $6,000 research fellowship by the National Institutes of Health. The one-year fellowship begins July 1. Dr. Schmid will study at Columbia University. Dr. Schmid, a native of Switzerland, spent his residency at University Hospitals in 1948. He has been an instructor of internal medicine since 1952.

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Dr. Nicholas H. Zeller has joined the staff of the Worthington Clinic as a specialist in internal medicine. Dr. Zeller comes to Worthington from the Veterans Administration Hospital in Saint Louis, Missouri, where he held the position of assistant chief of medicine. He graduated from the University of Minnesota in 1946 and since that time he has been taking specialty training in internal medicine at the Saint Louis Hospital.

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Dr. William J. Muesing joined the Seifert Clinic in New Ulm early in April. He recently completed a four-year residency in general surgery with the Youngstown, Ohio, Hospital Association. Dr. Muesing attended St. Olaf College and the University of Minnesota, graduating from the University Medical School in 1946. He took his internship at the San Diego, California, Naval Hospital.

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Dr. Alan Hodges, head of the Southern Minnesota Mental Health Clinic at Albert Lea, discussed his work with the mentally retarded at the meeting of the Austin Association for the Mentally Retarded in April. In an informal question-answer session, Dr. Hodges outlined the services offered and the mental illnesses treated at the clinic.

MINNESOTA BLUE SHIELD-BLUE CROSS

Already, in the February monthly report of Blue Shield affairs, it is evident that more of the Blue Shield subscriber's dollar is being spent for claims. This was the Board of Directors' purpose in increasing the benefits or fees which became effective for services rendered subscribers on and after January 1, 1954. Up to the present time, a significant proportion of the claims processed are for services rendered prior to the first of this year. Consequently, the full effect of the increased fees is not readily predictable even though the trend of the increase in claims cost is apparent.

During 1953, 76.8 per cent of the cash earned from subscribers was devoted to the cost of claims. This was a distinct increase over 1952 when 70.2 per cent of this income was disbursed for claims. For January of this year, this percentage increased to 77.2 and again in February it increased to 77.9. Even though these increases are slight, it is assumed they indicate a trend. The latter figure is more impressive considering the fact that it is a short month and, also, since a not inconsiderable proportion of the claims involved services uninfluenced by the increases of fees which became effective the first of this year.

Other features of the report for February emphasize the fact that while the trends shown are apparent, the time has been too short to make broad conclusions. For instance, the proportions of claims involving surgery and obstetrics are practically the same as last year, whereas, there is a definite increase in claims involving medical care, x-rays and anesthesia. If the present trend continues throughout the year, medical care services will experience about a ten per cent increase in general and emergency medical care services almost a 50 per cent increase. Based on data in the February report, claims for anesthesia may show a final ten per cent increase and x-ray claims about five per cent.

Increased cost of claims is important in its relationship to the organization's reserves for future medical and surgical care and contingencies. In fact, as soon as such reserve funds began to approach the level which has been recommended for soundness and security, more money was allocated by the Board of Directors to increased benefits. It was the result of this policy that many of the fees were increased the first of January, 1954. In the light of these facts, it is important that while for the year 1953, 14.1 per cent of gross income was transferred to unassigned surplus to meet contingencies, only 10.7 per cent has been allocated to such reserves during the first two months of 1954. That this percentage is likely to decrease throughout the year is evident from the fact that in February it was only 9.2 per cent.

From the facts presented, it is obvious that the increased benefits or fees are already requiring an increase in the percentage of income spent from medical-surgical-obstetrical claims, and that a decreasing percentage is available for reserves. However, these developments are in keeping with the non-profit motive of Blue Shield and also with the Board of Directors' policy to return as much of the income to subscribers in the form of benefits for various types of doctors' services as is financially sound.

HOSPITAL NEWS

Following is a list of the officers of the staff of Hibbing General Hospital and department heads for the year 1954:

Officers	
Chief of Staff	Dr. B. F. Flynn
Vice president	Dr. J. L. Arko
Secretary	Dr. G. Doxsee
Department Heads	
Medicine	Dr. W. G. Tomhave
Surgery	Dr. C. Jacobson
Otolaryngology	Dr. A. Sinamark
Ophthalmology	Dr. T. R. Schweiger
Contagion	Dr. C. A. Tingdale
Pediatrics	Dr. C. N. Harris
Radiology	Dr. B. Halper
Pathology	Dr. H. R. Irwin
Obstetrics	Dr. T. A. Estrem
Physiotherapy	Dr. S. Lipinski

BOOK REVIEWS

Books listed here become the property of the Ramsey, Hennepin and St. Louis County Medical Libraries when reviewed. Members, however, are urged to write reviews of any or every recent book which may be of interest to physicians.

TUBERCULOSIS IN CHILDHOOD AND ADOLESCENCE. By F. J. Bentley, M.D., F.R.C.P., D.P.H., Senior Physician, and S. Grzybowski, M.D., M.R.C.P., Senior Registrar, High Wood Hospital for Children, Brentwood, Essex; and B. Benjamin, B.Sc., F.I.A., formerly Statistician, London County Council. Preface by Sir Robert Young, C.B.E., M.D., F.R.C.P. Foreword by Wilfrid Sheldon, C.V.O., M.D., F.R.C.P. 272 pages. Illus. Price $5.00. London: National Association for Prevention of Tuberculosis, 1954.

This must surely be the first major project of clinical research arising from the National Health Service since the new scheme for decentralized research was set out in 1953. In point of fact, the present study was commenced shortly after the National Health Service was inaugurated, although final arrangements for decentralized research have only recently been promulgated.

The work is based on an experience of over 1,000 children treated in sanatorium and followed up for an average period of six years. A special investigation into the clinical and pathological details surrounding the deaths from tuberculosis of 100 unselected London children is also included, and this reveals the extent to which death from this disease in childhood may be preventable.

The practical management of tuberculosis in children has so far presented a confused and uncertain picture and this has probably arisen from an acceptance of the pattern of the disease as it has been so fully studied and understood in adults. It is the authors' contention that, apart from those rare instances of the adult type of the disease, pulmonary tuberculosis in childhood presents an entirely different clinical picture. Data are provided, for example, which demonstrate that the extent of the radiological shadows in primary disease is often of very little prognostic significance.

The various manifestations of the disease in childhood and adolescence are considered from the aspect of incidence, diagnosis, prognosis and treatment in such a way that the publication can be used as a text-book.

An original picture of the possible connections between primary tuberculosis and the adult type of disease is outlined in a chapter on pathogenesis. Although some of these views may be found controversial, they should certainly stimulate further thought and research on this important subject which, for so long, has been dominated by theories formulated in the early years of the present century.

As Dr. Sheldon states in his Foreword, High Wood Hospital—the largest hospital for pulmonary tuberculosis in children in the country—was an ideal field for observation. In addition to the prolonged period of residential treatment, over 90 per cent of the cases were followed up for many years. No other centre could have supplied so much information and particularly is this the case in the section dealing with chronic pulmonary tuberculosis (the adult type of the disease)—a section described by Dr. Sheldon as unique.

The authors' findings demonstrate that our methods of dealing with the disease in children have become unsatisfactory and call for an overhaul of both clinical and administrative arrangements.

The book will also be of great value in those parts of the world where the main battle against tuberculosis has still to be fought, as the fundamental pattern of tuberculosis shows striking similarities although its tempo may vary from country to country.

RECENT ADVANCES IN CARDIOVASCULAR PHYSIOLOGY AND SURGERY. A symposium presented by the Minnesota Heart Association and the University of Minnesota at the University of Minnesota in September, 1953. Edited by Grace M. Roth, Ph.D., Rochester, Minnesota, Chairman of the Committee for the Symposium. 132 Pages. Illus. Paper binding, $1.00. Saint Paul: Minnesota Heart Association, 532 Endicott-on-Fourth, 1954.

This symposium brings together the important contributions of a representative group of investigators from various parts of the United States, Canada, and Europe, and was designed to include discussion of the various aspects of new knowledge in cardiovascular physiology and surgery.

Subjects covered in the three-day symposium are: Heart Muscle Contraction, Regulation of the Circulation, Measurements of Pressure and Flow, Differential Diagnosis of Various Defects by Physiologic Means, Recent Advances in Surgical Treatment of Heart Disease. The George Fahr Lecture on the Physiology of Congestive Failure of the Circulation, presented at an evening session, adds to the comprehensive scope of the presentation.

Contributors to the symposium include:

Wilfried F. H. M. Mommaerts, Western Reserve University School of Medicine, Cleveland; Olle Snellman, University of Uppsala, Uppsala, Sweden; Torgny Sjostrand, Karolinska Hospital, Stockholm, Sweden; Otto H. Gauer, Aero Medical Laboratory, Wright Air Development Center, Dayton. Robert F. Rushmer, University of Washington School of Medicine, Seattle; Richard W. Stow, Ohio State University College of Medicine, Columbus; Erik Wetterer, University of Munich, Western Germany; Earl H. Wood, Mayo Foundation, Rochester; C. W. Sheppard, Oak Ridge National Laboratory, Oak Ridge; John W. Remington, Medical College of Georgia, Augusta; Homer R. Warner, University of Utah College of Medicine, Salt Lake City; Lewis Dexter, Harvard University Medical School, Boston; H. J. S. Swan, Mayo Foundation, Rochester; Charles T. Dotter, University of Oregon Medical School, Portland; Robert P. Glover, Hahnemann Medical College and Hospital, Philadelphia; John H. Gibbon, Jefferson Medical College, Philadelphia; W. C. Bigelow, University of Toronto, Toronto, Canada; Henry Swan, University of Colorado School of Medicine, Denver; Willis J. Potts, Northwestern University Medical School, Chicago; Dwight F. Harken, Harvard University Medical School, Boston; William F. Hamilton, Medical College of Georgia, Augusta.

This is a valuable addition to any physician's or surgeon's library and should be of particular interest to those specializing in cardiology.

Minnesota State Medical Association Roster for 1954

Officers

Justus Ohage, M.D.	*President*	Saint Paul
Paul C. Leck, M.D.	*First Vice President*	Austin
Vernon D. E. Smith, M.D.	*Second Vice President*	Saint Paul
B. B. Souster, M.D.	*Secretary*	Saint Paul
W. H. Condit, M.D.	*Treasurer*	Minneapolis
C. G. Sheppard, M.D.	*Speaker, House of Delegates*	Hutchinson
H. M. Carryer, M.D.	*Vice Speaker*	Rochester
R. R. Rosell	*Executive Secretary*	Saint Paul

Councilors

(Terms expire December 31 of year indicated)

First District

J. Minott Stickney, M.D. (1956)	Rochester

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H. A. Roust	Montevideo
M. A. Burns	Milan
L. A. Krystosek	Clara City

CHISAGO COUNTY

J. E. Halpin	Rush City
A. L. Holmes	Rush City
R. G. Swenson	North Branch

CLAY COUNTY

V. D. Thysell	Hawley
Carl Simison	Barnesville
H. G. Rice	Moorhead

CLEARWATER COUNTY

L. J. Larson	Bagley
W. E. Anderson	Clearbrook

COOK COUNTY

W. R. Smith	Grand Marais

COTTONWOOD COUNTY

H. C. Stratte	Windom
E. S. Schutz	Mountain Lake
J. V. Carlson	Westbrook

CROW WING COUNTY

V. E. Quanstrom	Brainerd
G. I. Badeaux	Brainerd
J. B. Nixon	Crosby

DAKOTA COUNTY

Bernice Thoreson	South St. Paul
H. J. Just	Hastings
A. H. Field	Farmington

DODGE COUNTY

C. E. Bigelow	Dodge Center
D. E. Affeldt	Kasson
G. E. Olson	West Concord

DOUGLAS COUNTY

G. W. Clifford	Alexandria
E. E. Emerson	Osakis
E. R. Sather	Alexandria

FARIBAULT COUNTY

W. C. Chambers	Blue Earth
M. D. Cooper	Winnebago
Richard Virnig	Wells

FILLMORE COUNTY

H. M. Skaug	Chatfield
L. W. Clark	Spring Valley
J. P. Nehring	Preston

FREEBORN COUNTY

O. A. Erdal	Albert Lea
J. P. Person	Albert Lea
A. G. Sherman	Albert Lea
S. G. Egge	Albert Lea

GOODHUE COUNTY

W. W. Liffrig	Red Wing
R. B. Graves	Red Wing
G. F. Hartnagel	Red Wing

GRANT COUNTY

L. R. Parson	Elbow Lake
J. H. Cain	Hoffman

HENNEPIN COUNTY (RURAL)

W. W. Rieke	Wayzata
H. E. Drill	Hopkins
E. J. Lillehei	Robbinsdale

HOUSTON COUNTY

N. T. Norris	Caledonia
L. K. Onsgard	Houston
L. A. Knutson	Spring Grove

HUBBARD COUNTY

Donald Houston	Park Rapids
W. W. Higgs	Park Rapids
John Eiler	Park Rapids

ISANTI COUNTY

W. T. Nygren	Braham
R. J. Gully	Cambridge
G. E. Larson	Cambridge

ITASCA COUNTY

E. K. Rowles	Coleraine
G. M. Erskine	Grand Rapids
M. J. McKenna	Grand Rapids

JACKSON COUNTY

J. T. Rose	Lakefield
H. A. Christensen	Jackson
W. H. Halloran	Jackson

KANABEC COUNTY

C. S. Bossert	Mora
W. F. Nordman	Mora
Harry L. Berge	Mora

KANDIYOHI COUNTY

H. G. Bosland	Willmar
Walter Hinz	Willmar
J. C. Jacobs	Willmar

KITTSON COUNTY

A. S. Berlin	Hallock
R. B. Skogerboe	Karlstad

KOOCHICHING COUNTY

R. A. MacDonald	Littlefork
R. D. Hanover	International Falls
C. C. Craig	International Falls

LAC QUI PARLE COUNTY

George Boody, Jr.	Dawson
Chester Anderson	Madison

LAKE COUNTY

Ralph Papermaster	Two Harbors

LAKE-OF-THE-WOODS COUNTY

A. A. Brink	Baudette

LINCOLN COUNTY

C. P. Johnson	Tyler
P. E. Hermanson	Hendricks
L. J. Monson	Canby

LYON COUNTY

B. C. Ford	Marshall
R. R. Remsberg	Tracy
W. W. Yaeger	Marshall

MAHNOMEN COUNTY

K. W. Covey	Mahnomen
J. M. Gacusana	Mahnomen

MARSHALL COUNTY

C. H. Holmstrom	Warren
E. E. Pumala	Warren

MARTIN COUNTY

R. C. Hunt	Fairmont
O. E. Wandke	Fairmont
C. W. Krause	Fairmont
H. A. Williamson	Heron Lake

MCLEOD COUNTY

A. M. Jensen	Brownton
E. W. Lippman	Hutchinson
Joseph Selmo	Norwood

MEEKER COUNTY

C. A. Wilmot	Litchfield
Lennox Danielson	Litchfield
David D. Allison	Litchfield

MILLE LACS COUNTY

W. R. Blomberg	Princeton
A. T. Kapsner	Princeton
N. B. Metcalf	Onamia

MORRISON COUNTY

D. L. Johnson	Little Falls
A. M. Watson	Royalton
E. G. Knight	Swanville

MOWER COUNTY

R. R. Wright	Austin
S. C. Peterson	Austin

MURRAY COUNTY

B. M. Stevenson	Fulda
R. F. Pierson	Slayton
H. D. Patterson	Slayton

NICOLLET-LE SUEUR COUNTY

R. A. Curtis	Le Center
H. C. Johnson	North Mankato
M. E. Lenander	Saint Peter

NOBLES COUNTY

E. W. Arnold	Adrian
E. A. Kilbride	Worthington
C. R. Stanley	Worthington

NORMAN COUNTY

Eskil Erickson	Halstad
Theodore Loken	Ada
B. R. Kinkade	Ada

OLMSTED COUNTY

H. F. Polley	Rochester
T. O. Wellner	Rochester
C. B. McKaig	Pine Island

OTTER TAIL COUNTY

Charles Lewis	Henning
Frank Naegeli	Fergus Falls
H. K. Helseth	Pelican Rapids

PENNINGTON COUNTY

M. D. Starekow	Thief River Falls
Philip G. Parker	Thief River Falls
William M. Feigal	Thief River Falls

PINE COUNTY

A. K. Stratte	Pine City
E. G. Hubin	Sandstone
H. P. Dredge	Sandstone

PIPESTONE COUNTY

W. G. Benjamin	Pipestone
J. G. Lohmann	Pipestone
G. Beckering	Edgerton

POLK COUNTY

George Sather	Fosston
O. K. Behr	Crookston
W. F. Mercil	Crookston

POPE COUNTY

Paul Swedenberg	Glenwood
A. F. Giesen	Starbuck

RAMSEY COUNTY

J. F. Borg	Saint Paul
T. S. McClanahan	White Bear Lake
Herman J. Wolffe	Saint Paul

RED LAKE COUNTY

L. N. Dale	Red Lake Falls
A. M. McKaig	Red Lake Falls

REDWOOD COUNTY

Wm. Alcorn	Wabasso
R. J. Cairns	Redwood Falls
A. W. Diessner	Redwood Falls

RENVILLE COUNTY

J. A. Cosgriff, Sr.	Olivia
John Dordal	Sacred Heart
A. M. Fawcett	Renville

RICE COUNTY

Bernard Street	Northfield
G. L. Kennedy	Faribault
A. M. Nielsen	Northfield

ROCK COUNTY

C. L. Sherman	Luverne
A. C. Martin	Luverne
F. W. Bofenkamp	Luverne

ROSEAU COUNTY

J. L. Delmore, Jr.	Roseau
D. O. Berge	Roseau
K. L. Nelson	Warroad
L. Klefstad	Greenbush

ST. LOUIS COUNTY

P. F. Eckman	Duluth
L. R. Gowan	Duluth
D. W. Wheeler	Duluth

SCOTT COUNTY

H. M. Juergens	Belle Plaine
B. F. Pearson	Shakopee

SHERBURNE COUNTY

A. B. Roehlke	Elk River
G. H. Tesch	Elk River

SIBLEY COUNTY

Rolf Hovde	Winthrop
R. H. Kath	Arlington
D. C. Olson	Gaylord

STEARNS-BENTON COUNTY

G. F. Brigham, Jr.	St. Cloud
John B. Beuning	St. Cloud
R. N. Jones	St. Cloud
N. F. Musachio	Foley
K. A. Walfred	St. Cloud

STEELE COUNTY

D. H. Dewey	Owatonna
Theo. Stransky	Owatonna

STEVENS COUNTY

M. L. Ransom	Hancock
R. A. Rossberg	Morris
A. I. Arneson	Morris

SWIFT COUNTY

E. J. Kaufman	Appleton
D. F. Holm	Benson
B. C. Ostling	Kerkhoven

TODD COUNTY

M. E. Mosby	Long Prairie
J. M. Cook	Staples
C. B. Will	Bertha

TRAVERSE COUNTY

A. L. Lindberg	Wheaton
A. E. Magnuson	Wheaton
W. F. Muir	Browns Valley

WABASHA COUNTY

L. M. Ekstrand	Wabasha
W. P. Gjerde	Lake City
E. W. Ellis	Elgin

WADENA COUNTY

L. T. Davis	Wadena
C. H. Pierce	Wadena
W. E. Parker	Sebeka

WASECA COUNTY

G. H. Olds	New Richland
H. M. McIntire	Waseca
B. J. Gallagher	Waseca

WASHINGTON COUNTY

W. R. Humphrey	Stillwater
Russell E. Carlson	Stillwater

WATONWAN COUNTY

O. B. Bergman	Saint James
Herbert Boysen	Madelia

WILKIN COUNTY

C. W. Jacobson	Breckenridge
L. T. O'Brien	Breckenridge

WINONA COUNTY

R. H. Wilson	Winona
R. B. Tweedy	Winona
Herbert Heise	Winona

WRIGHT COUNTY

John J. Catlin	Buffalo
C. L. Roholt	McIntosh

YELLOW MEDICINE COUNTY

Carl Lundell	Granite Falls
M. W. Plucker	Clarkfield
G. Charles Wilcox	Wood Lake

Councilor Districts

FIRST DISTRICT

J. M. Stickney, Jr., M.D. .... Rochester
Counties—Dodge, Fillmore, Goodhue, Houston, Mower, Olmsted, Rice, Steele, Wabasha, Winona

SECOND DISTRICT

R. C. Hunt, Sr., M.D. .... Fairmont
Counties—Cottonwood, Faribault, Freeborn, Jackson, Martin, Murray, Nobles, Pipestone, Rock, Watonwan

THIRD DISTRICT

L. G. Smith, M.D. .... Montevideo
Counties—Big Stone, Chippewa, Kandiyohi, Lac Qui Parle, Lincoln, Lyon, Meeker, Pope, Redwood, Renville, Stevens, Swift, Traverse, Yellow Medicine

FOURTH DISTRICT

H. J. Nilson, M.D. .... North Mankato
Counties—Blue Earth, Brown, Carver, LeSueur, McLeod, Nicollet, Scott, Sibley, Waseca

FIFTH DISTRICT

L. R. Critchfield, M.D. .... Saint Paul
Counties—Anoka, Chisago, Dakota, Isanti, Kanabec, Mille Lacs, Pine, Ramsey, Sherburne, Washington

SIXTH DISTRICT

H. B. Sweetser, Jr., M.D. .... Minneapolis
Counties—Hennepin, Wright

SEVENTH DISTRICT

W. W. Will, M.D. .... Bertha
Counties—Aitkin, Beltrami, Benton, Cass, Clearwater, Crow Wing, Hubbard, Koochiching, Morrison, Stearns, Todd, Wadena

EIGHTH DISTRICT

C. L. Oppegaard, M.D. .... Crookston
Counties—Becker, Clay, Douglas, Grant, Kittson, Lake of the Woods, Mahnomen, Marshall, Norman, Otter Tail, Pennington, Polk, Red Lake, Roseau, Wilkin

NINTH DISTRICT

A. O. Swenson, M.D. .... Duluth
Counties—Carlton, Cook, Itasca, Lake, St. Louis

WOMAN'S AUXILIARY to the MINNESOTA STATE MEDICAL ASSOCIATION

Officers

Mrs. Peter S. Rudie	*President*	Duluth
Mrs Henry W. Quist, Sr.	*Past President*	Minneapolis

Officers to be Elected June, 1954

President-Elect
First Vice President
Second Vice President
Third Vice President
Fourth Vice President
Recording Secretary
Treasurer
Auditor

Officers Appointed

Mrs. S. N. Litman	*Corresponding Secretary*	Duluth
Mrs. A. J. Bianco	*Historian*	Duluth
Mrs. Leo Fink	*Parliamentarian*	Minneapolis

Regional Advisors

(To be selected)	*First District*	
Mrs. O. M. Heiberg	*Second District*	Worthington
Mrs. E. R. Hudec	*Third District*	Echo
Mrs. T. R. Fritsche	*Fourth District*	New Ulm
Mrs. O. I. Sohlberg	*Fifth District*	Saint Paul
Mrs. Ralph Creighton	*Sixth District*	Minneapolis
Mrs. J. C. Buscher	*Seventh District*	Saint Cloud
Mrs. C. W. Moberg	*Eighth District*	Detroit Lakes
Mrs. J. L. McLeod	*Ninth District*	Grand Rapids

Chairmen of Committees

Advisory—Mrs. Henry W. Quist, Sr. .... Minneapolis
Allied Medical Careers—Mrs. Stanley Peterson .... Austin
American Medical Education Foundation Fund—Mrs. J. A. Cosgriff .... Olivia
Archives—Mrs. J. L. Benepe .... Saint Paul
Bulletin—Mrs. C. E. Carlson .... Alexandria
Cancer—Mrs. W. G. Benjamin .... Pipestone
Civilian Defense—Mrs. D. G. Mahle .... Plainview
Editor (Minnesota Medicine)—Mrs. L. Raymond Scherer .... Minneapolis
Finance—Mrs. Mark Ryan .... Saint Paul
Health Days—Mrs. Harold F. Wahlquist .... Minneapolis
In Memoriam—Mrs. John J. Ryan .... Saint Paul
Legislation—Mrs. Philip K. Arzt .... Saint Paul
Medical and Surgical Relief—Mrs. W. T. Greenfield .... Cokato
Mental Health—Mrs. A. E. Lindblom .... Mankato
News Letter—Mrs. M. F. Fellows .... Duluth
Organization—(To be selected)
Press and Publicity—Mrs. A. B. Rosenfield .... Minneapolis
Printing—Mrs. William Gjerde .... Lake City
Program and Health Education—Mrs. C. E. Merkert .... Minneapolis
Public Relations—(To be selected)
Resolutions—Mrs. P. J. Pankratz .... Mountain Lake
Revisions—Mrs. D. V. Boardman .... Winona
Roster—Mrs. R. F. Erickson .... Minneapolis
School of Instruction—Mrs. Leonard Arling .... Minneapolis
Today's Health—Mrs. M. I. Hauge .... Clarkfield

County Society Roster

Key to Symbols: *Deceased; †Affiliate, Associate or Life Members; ‡In Service; §Wife is Member of Woman's Auxiliary.

BLUE EARTH COUNTY MEDICAL SOCIETY

Regular meetings, last Monday of each month
Annual meeting, December
Number of Members—56

President
Kearney, R. Wynn........Mankato

Secretary
Chalgren, William S........Mankato

§ Anderson, James J........Mankato
Anderson, Margaret........Mankato
§ Andrews, Roy N........Mankato
§ Baird, Raymond L........Lake Crystal
§ Batdorf, B. Niles........Good Thunder
§ Butzer, John A........Mankato
Chalgren, William S........Mankato
§ Conley, Robert H........Mankato
*§†Dahl, G. A........Mankato
‡ Dahlstet, John P........San Diego, Calif.
Dobson, M. W........Mankato
§ Engstrom, Robert........Mankato
§ Eustermann, John J........Mankato
†§Franchere, F. W........Lake Crystal
§ Fugina, G. R........Mankato
§ Geurs, Benjamin R........Mankato
§ Haes, Julius E........Mankato
§ Hammar, Lawrence M........Mankato
Hankerson, Robert G........Minnesota Lake
† Hassett, Roger G........Mankato
§ Hoeper, Philip G........Mankato
§ Heller, Edgar E........Mankato
§ Howard, Marshall I........Mankato
† Huffington, H. L........Mankato
Huffington, Herbert L........Waterville
Jones, Orville H........Mankato
† Juliar, R. O........Glendale, Calif.
§ Kaufman, Walter B........Mankato
§ Kearney, R. Wynn........Mankato
§ Kemp, Alphonse F........Mankato
§ Koenigsberger, Charles........Mankato
§ Langhoff, A. H........Mankato
† Liedloff, A. G........Mankato
§ Lindblom, A. E........No. Mankato
† Luck, Hilda........Mankato
McNear, George R., Jr........Mankato
§ Mickelson, John C........Mankato
§†Miller, Victor I........Mankato
§ Morgan, Hugh O........Amboy
§ Penn, George E........Mankato
§ Roth, Fred K........Mankato
§ Sanford, Raymond........Mankato
§ Samuelson, L. Gordon........Mankato
† Schmidt, Paul A........Aurora, Ill.
§ Schmitz, Anthony A........Mankato
§ Sjoding, J. Donald........Mankato
§ Smith, Harry J........Lake Crystal
§ Smith, Paul M........Lake Crystal
† Sohmer, Alphonse E........Mankato
§ Stillwell, Walter C........Mankato
§ Thiem, Chester E........Mankato
§ Troost, H. Bradley........Mankato
§ Vezina, John C........Mapleton
§ Von Drasek, Joseph........Mankato
§†Wentworth, Albert J........Mankato
Williams, H. O........Lake Crystal
§ Wohlrabe, John C........St. Clair

BLUE EARTH VALLEY MEDICAL SOCIETY

Regular meetings, third Thursday of each month
Annual meeting 3rd Thursday in November
Number of Members—40

President
Bratrude, Earl J........St. James

Secretary
Boysen, Herbert........Madelia

Armstrong, Ralph S........Winnebago
Bergman, Oscar B........St. James
§ Blumberg, Henry B........Fairmont
§ Boysen, Herbert........Madelia
§ Bratrude, Earl J........St. James
Chambers, Winslow C........Blue Earth
Cooper, Maurice D........Winnebago
§ Coulter, Harold E........Madelia
Drexler, George W........Blue Earth
*†Farrish, R. C........Sherburn
Gardner, Victor H........Fairmont
§ Hanson, Louis........Frost
Heimark, Julius J........Fairmont
§ Hruza, William J........Madelia
Hunt, Roscoe C........Fairmont
*†Hunte, A. F........Alhambra, Calif.
Kraemer, George N........Fairmont
Krause, C. W........Fairmont
‡ Lester, Malcolm J., Jr........Truman
Lindahl, Merlyn J........Sherburn
McGroarty, John J........Easton
Mills, John L........Winnebago
§ Misbach, William D........Fairmont
§ Nickerson, John R........Fairmont
§ Nickerson, Neil D........Fairmont
§ Ourada, Anthony L........Ceylon
Parsons, Ralph L........Monterey
‡ Rollins, Troy G........Hoopa, Calif.
† Rowe, William H........Fairmont
§ Russ, Homer H........Blue Earth
§ Smith, Don V........Blue Earth
§ Snyder, Clifford D........Kiester
§ Thayer, Ellsworth A........Fairmont
Vaughan, Victor M........Truman
§ Virnig, Mark P........Wells
§ Virnig, Richard P........Wells
§ Wandke, Otto E........Fairmont
Watkins, John A........Wells
§ Williamson, Harold A........Fairmont
Wilson, Clyde E........Blue Earth
§ Zemke, Erhart E........Fairmont
Ziegler, Robert G........Welcome

BROWN COUNTY MEDICAL SOCIETY

Regular meetings, quarterly
Annual meeting, January
Number of Members—34

President
Fritsche, Theodore R........New Ulm

Secretary
Fritsche, Carl J........New Ulm

§ Black, William A........New Ulm
* Bregel, Fred L........St. James
§ Cairns, Robert J........Redwood Falls
Carthey, Frank J........New Ulm
§ Dubbe, Fredk. H........New Ulm
§ Dysterheft, Adolf F........Gaylord
§ Fesenmaier, Otto B........New Ulm
Flinn, James B........Redwood Falls
§ Fritsche, Albert........New Ulm
§ Fritsche, Carl J........New Ulm
§ Fritsche, Theodore R........New Ulm
† Glaeser, John H........Minneapolis
Goblirsch, Andrew P........Sleepy Eye
Hedlund, Charles J........St. James
Hovde, Rolf........Winthrop
Inglis, William........Redwood Falls
§ Kaiser, Milton L........New Ulm
§ Keithahn, Elmer E........Sleepy Eye
‡ Kitzberger, P. J........Bainbridge, Md.
§ Kruzick, S. J........Sleepy Eye
§ Kusske, A. L........New Ulm
† Kusske, B. W........St. Louis Park
Mattson, Albert D........St. James
Nuessle, Walter G........Springfield
† O'Leary, John B........Minneapolis
§ Penk, Engward L........Springfield
Peterson, Roy A........Vesta
Rayner, Ralph R........Gibbon
§†Reineke, George F........New Ulm
§ Saffert, Cornelius A........New Ulm
Schroeppel, John E........Winthrop
§ Seifert, Otto J........New Ulm
§ Vogel, Howard A. L........New Ulm
Wisness, Osmund A........Comfrey
§ Wohlrabe, Edwin J........Springfield

CAMP RELEASE DISTRICT MEDICAL SOCIETY

Chippewa, La Qui Parle and Yellow Medicine Counties
Regular meetings, 2nd and 4th weeks of April, May and September
Annual meeting third Thursday in October
Number of Members—32

President (Inactive)
Boody, George, Jr........Dawson
Acting President
Barr, Ronald W........Montevideo
Secretary
Lima, L. R., Jr........Montevideo

§ Allen, John H........Montevideo
Anderson, Chester A........Madison
§ Barr, Ronald W........Montevideo
§ Burns, Floyd M........Milan
§ Burns, M. Alpheus........Milan
Carroll, John J........Montevideo
‡ Flom, Robert S........Echo
Guilbert, G. D........Waukesha, Wis.
§ Hartfiel, Herbert........Montevideo
§ Hauge, Malvin I........Clarkfield
§ Hudec, Elwyn R........Echo
Hudspeth, William T........Madison

Hustad, Edward G..........Montevideo
‡ Johnson, Curtis M..........Dawson
§ Johnson, Vilhelm M..........Dawson
† Jordan, Kathleen B..........Granite Falls
Jordan, Lewis S..........Granite Falls
§ Kaufman, William C..........Appleton
§ Krystosek, Lee A..........Clara City
† Lee, Walter N..........Claremont, Calif.
§ Lima, Ludvig, Jr..........Montevideo
§ Lundell, Carl L..........Granite Falls
§ Odland, Olin M..........Granite Falls
§ Owens, William A..........Montevideo
§ Pertl, Albert L..........Canby
§ Plucker, Milton W..........Clarkfield
§ Roust, Henry A..........Montevideo
§ Schmidt, P. G., Jr..........Granite Falls
§ Smith, Leon G..........Montevideo
§ Westby, Magnus..........Madison
§ Westby, Norval M..........Madison
§ Wilcox, G. Charles..........Woodlake

CLAY-BECKER COUNTY MEDICAL SOCIETY

Regular meeting, none. Annual meeting, November 30
Number of Members—25

President
Johnson, Olga..........Moorhead

Secretary
Dodds, William C..........Detroit Lakes

*†Aborn, W. H..........Hawley
§ Bigler, Earl E..........Perham
§ Bigler, Ivan E..........Perham
Bottolfson, Bottolf T..........Moorhead
§ Carlson, Vernon J..........Moorhead
§ Dodds, William C..........Detroit Lakes
§ Duncan, James W..........Moorhead
Geib, Marvin J..........Moorhead
† Hagen, Olaf J..........Moorhead
§ Houglum, Arvid J..........Lake Park
§ Humphrey, Edward W..........Moorhead
§ Humphrey, Edward W., Jr...Moorhead
Johnson, Olga H..........Moorhead
§ Larson, Arnold..........Detroit Lakes
§ Lorentzen, Ernest..........Detroit Lake
§ Midthune, A. S..........Lake Park
§ Moberg, Clarence..........Detroit Lakes
§ Odland, Mark E..........Detroit Lakes
§ Oliver, James..........Moorhead
§ Otto, Henry C..........Frazee
§ Rice, Hagbart G..........Moorhead
§ Rutledge, Lloyd..........Detroit Lakes
Saxman, Gertrude Olsen..........Georgetown
* Seitz, S. B..........Barnesville
§ Simison, Carl..........Barnesville
† Thysell, Fred A..........Moorhead
§ Thysell, Vernon D..........Hawley

EAST CENTRAL MINNESOTA MEDICAL SOCIETY

Anoka, Chisago, Isanti, Kanabec, Mille Lacs, Pine and Sherburne Counties
Regular meetings, first Tuesday of February, April, June, August, October and December
Annual meeting, first Tuesday in December
Number of Members—39

President
Hubin, Edwin G..........Sandstone

Secretary
Tesch, Gordon H..........Elk River

Adkins, Galen H..........Anoka
§ Albrecht, H. H..........Chisago City
‡ Baars, C. W..........Camp Cook, Calif.
§ Berge, Harry L..........Mora
‡ Beyer, Eugene..........San Francisco, Calif.
§ Blomberg, Wm. R..........Princeton
† Bossert, Clarence S..........Mora
§ Bunker, B. W..........Anoka
§ Burseth, Edgar C..........Mora
† Dredge, Homer P..........Sandstone
§ Gully, Raymond J..........Cambridge
Halpin, Joseph E..........Rush City
‡ Henry, Harold W..........Benton, Ill.
Holmes, Alva E..........Rush City
§ Hubin, Edwin G..........Sandstone
Johnson, Aldridge F..........Isle
§ Kapsner, Alfred T..........Princeton
† Kelsey, Carleton G..........Saint Paul
Kragh, Lyle O..........Braham
Krieser, Albert E..........Anoka
§ Larson, Gerald E..........Cambridge
‡ Lorton, Wm. L..........Rochester
‡ Mach, Ralph F..........Minneapolis
‡ Magnuson, R. C..........Cambridge
§ March, Kenneth A..........Cambridge
§ McManus, William F..........Princeton
‡ Metcalf, Norman B..........Princeton
§ Nordman, Willard F..........Mora
Nygren, William T..........Braham
§ Roehlke, Arthur B..........Elk River
* Schlesselman, George H.....Minneapolis
§ Sherman, Hubert T..........Cambridge
Spurzem, R. J..........Anoka
‡ Stahn, Louis H..........El Paso, Texas
§ Stratte, Alf K..........Pine City
§ Swensen, R. G..........North Branch
§ Tesch, Gordon H..........Elk River
Vik, Melvin..........Anoka
† Waller, Jos. D..........Pine City
Woyda, William C..........Elk River

FREEBORN COUNTY MEDICAL SOCIETY

Regular meetings, every two months, third Thursday
Annual meeting, December
Number of Members—29

President
Hansen, Theodore M..........Albert Lea

Secretary
Steiner, Leon E..........Albert Lea

§ Barr, Lowell C..........Albert Lea
Burns, Catherine..........Albert Lea
§ Butturff, Carl R..........Freeborn
†§Calhoun, F. W..........Albert Lea
§ Demo, Robert A..........Albert Lea
§ Donovan, Daniel L..........Albert Lea
§ Egge, Sanford..........Albert Lea
§ Ellertson, Leonard..........Albert Lea
§ Erdal, Ove A..........Albert Lea
§ Folken, Frank G..........Albert Lea
† Freeman, John P..........Glenville
* Freligh, W. P..........Albert Lea
† Gullickson, Andrew......Longmont, Colo.
§ Hansen, Theodore M..........Albert Lea
Kaasa, Lawrence J..........St. Peter
† Kamp, Bryon A..........Albert Lea
§ Keil, Marcus A..........Albert Lea
† Leopard, Brand A.....Brownsville, Texas
Miller, Samuel..........Albert Lea
Neel, Harry B..........Albert Lea
§ Nelson, Clayton E..........Albert Lea
§ Nesheim, Martin O..........Emmons
Palmer, Clinton F..........Albert Lea
§ Person, John P..........Albert Lea
§ Prins, Leo R..........Albert Lea
§ Schmidt, Ruben F..........Alden
§†Schultz, J. Albert..........Albert Lea
§ Sherman, Alfred G..........Albert Lea
§ Steiner, Leon E..........Albert Lea
§ Whitson, Sidney A..........Albert Lea

GOODHUE COUNTY MEDICAL SOCIETY

Regular meeting, none
Annual meeting, December
Number of Members—28

President
Bridge, Ezra V..........Cannon Falls

Secretary
Hawley, George M. B..........Red Wing

Aanes, Almer M..........Red Wing
§ Akins, Willard M..........Red Wing
Allen, George S..........Cannon Falls
‡ Bagby, George W..........
Ft. Leonard Wood, Mo.
‡ Boswell, J. T..........Portsmouth, Va.
Bridge, Ezra V..........Cannon Falls
§ Brusegard, James F..........Red Wing
Claydon, Howard F..........Red Wing
§ DeGeest, James H..........Goodhue
‡ Dovenmuehle, R. H..........Durham, N. C.
Flom, Martin G..........Zumbrota
§ Graves, Richard B..........Red Wing
§ Hartnagel, G. F..........Red Wing
§ Hawley, G. M. B., II..........Red Wing
§ Hedin, Raymond F..........Red Wing
† Jones, Alvah W..........Red Wing
§ Juers, Edward H..........Red Wing
§ Kimmel, George C..........Red Wing
§ Larson, Oliver E. H..........Zumbrota
§ Liffrig, William W..........Red Wing
§ Miller, Winston R..........Red Wing
Molenaar, Robert E..........Cannon Falls
§ Sherman, Royal V..........Red Wing
† Smith, Myron W..........Red Wing
§ Steffens, Leon A..........Red Wing
§ Wasmund, Clarence W..........Red Wing
Weiss, Carl A..........Hastings
§ Williams, M. R..........Cannon Falls

HENNEPIN COUNTY MEDICAL SOCIETY

Regular meetings, first Monday of each month
Annual meeting, first Monday in October
Number of Members—923

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Holmberg, Conrad J............Minneapolis
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§ Agustsson, Hreidar............Minneapolis
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§ Anderson, Ernest............Minneapolis
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§ Arvidson, Carl G............Minneapolis
†§Aune, Martin............Minneapolis
† Aurand, Wm. H............Minneapolis
§ Baggenstoss, O. J............Minneapolis
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§ Baird, Joe W............Minneapolis
§ Baken, Melvin P............Minneapolis
Baker, A. B............Minneapolis
Baker, Alfred T............Minneapolis
† Baker, Looe............Minneapolis
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§ Balkin, Saml. G............Minneapolis
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‡§Barno, Alex............St. Louis Park
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†§Barron, Moses............Minneapolis
Barron, S. Steven............Minneapolis
†§Baxter, Stephen H............Minneapolis
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† Becker, Arnetta............Minneapolis
§ Bedford, Edgar W............Minneapolis
§ Beirstein, Saml............Minneapolis
§ Beiswanger, Richard H............Minneapolis
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§ Bellville, Titus P............Minneapolis
§ Belzer, Meyer S............Minneapolis
Benesh, Louis A............Minneapolis
*†Benjamin, A. E............Minneapolis
§ Benjamin, Edwin G............Minneapolis
§ Benjamin, Harold G............Minneapolis
Benson, Ellis S............Minneapolis
Berger, Alex G............Minneapolis
§ Bergh, George S............Minneapolis
Bergh, Solveig M............Minneapolis
Berglund, Eldon B............Minneapolis
§ Berkwitz, N. J............Minneapolis
Berman, Reuben............Minneapolis
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§ Blake, Allen J............Hopkins
§ Blake, James A............Hopkins
§ Blake, Paul S............Hopkins
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‡§Bohn, Donald G............Minneapolis
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Boreen, Clifton A............Minneapolis
†§Borgeson, Egbert J............Saint Paul
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§ Bowers, Gordon G............Minneapolis
Boynton, Ruth E............Minneapolis
§ Bratrud, Arthur F............Minneapolis
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§ Brekke, Harvey J............Minneapolis
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† Brown, Edgar D............Paynesville
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Brown, Wm. D............Minneapolis
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Bulkley, Kenneth............Minneapolis
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§ Bushard, Wilford J............Minneapolis
§ Buzzelle, Leonard............Minneapolis
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§ Cabot, Clyde M............Minneapolis
*§Cabot, V. S............Minneapolis
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Cameron, Isabell L............Minneapolis
Campbell, Lowell M............Minneapolis
§ Campbell, Orwood J............Minneapolis
§ Caplan, Leslie............Minneapolis
‡§Card, Wm. H............Minneapolis
§ Carey, James B............Minneapolis
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§ Carlson, Lawrence............Minneapolis
§ Carlson, Leonard T............Minneapolis
§ Caron, Robert P............Minneapolis
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†§Cavanor, Frank T............Minneapolis
§ Ceder, Elmer T............Minneapolis
Challman, Samuel A............Minneapolis
§ Chavez, Demetrio A............Minneapolis
Chesler, Merrill............Minneapolis
† Chesley, Albert J............Minneapolis
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§ Christensen, L. E............Minneapolis
* Christianson, H. W............Minneapolis
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§ Cochrane, Ray F............Minneapolis
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Cohen, Ephraim B............Minneapolis
Cohen, Maynard M............Saint Paul
§ Cohen, Sumner S............Oak Terrace
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§ Cooper, John P............Excelsior
§ Cooper, Robert R............Minneapolis
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§ Correa, Dale H............Minneapolis
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Cranston, Robt. W............Minneapolis
§ Creevy, Chas. D............Minneapolis
§ Creighton, Ralph H............Minneapolis
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§ Cundy, Donald T............Minneapolis
† Cutts, George............Minneapolis
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§ Daggett, Donald R............Minneapolis
§ Dahl, Elmer O............Minneapolis
‡ Dahl, James C............Minneapolis
§ Dahl, John A............Minneapolis
§ Daniel, Donald H............Minneapolis
Danyluk, Michael............Minneapolis
§ Dargay, Cyril P............Minneapolis
§ David, Reuben............Minneapolis
§ Davis, Jay C............Minneapolis
§ Davis, Wm. I............Mound
Dawson, Lorin D............Minneapolis
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§ Devereaux, Thos. J............Wayzata
Dewall, Richard............Minneapolis
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§ Diehl, Harold S............Minneapolis
Diessner, Henry D............Minneapolis
§ Donatelle, E. P............Minneapolis
§ Dorge, Richard I............Minneapolis
Dornbach, Robert A............Excelsior
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§ Dorsey, George C............Minneapolis
† Doxey, Gilbert L............Minneapolis
§ Doyle, Lawrence O............Minneapolis
†§Drake, Charles R............Minneapolis
§ Dredge, Thomas E............Minneapolis
§ Drill, Herman E............Hopkins
§ Duff, Edwin R............Minneapolis
Dummer, Donald J............Minneapolis
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§ Dupont, Joseph A............Excelsior
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† Dutton, C. E............Minneapolis
§ Dvorak, Benjamin A............Minneapolis
§ Dwan, Paul F............Minneapolis
§ Dworsky, Samuel D............Minneapolis
§ Ehrenberg, Claude J............Minneapolis
§ Ehrlich, Sol P............Minneapolis
§ Eich, Matthew............Minneapolis
§ Eichhorn, Edmund P............Minneapolis
Eisenstadt, David............Minneapolis
§ Eisenstadt, Wm. S............Minneapolis
§ Eitel, George D............Minneapolis
§ Ellison, David E............Minneapolis
§ Ellison, Ellis............Minneapolis
§ Emond, Albert J............Farmington
Emond, J. S............Farmington
Engel, Joseph P............Minneapolis
§ Engelhart, P. C............Minneapolis
Englund, Elvin F............Minneapolis
§ Engstrand, Oscar J............Minneapolis
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‡§Erickson, Laurence F............Minneapolis
§ Erickson, Myron E............Minneapolis
§ Erickson, Reuben F............Minneapolis
§ Ericson, Reinhold M............Wayzata
Esensten, Sidney............Minneapolis
§ Evans, Edward T............Minneapolis
Evans, Robt. D............Minneapolis
Fahr, George............Minneapolis
§ Fansler, Walter A............Minneapolis
§ Feeney, John M............Minneapolis
‡§Feigal, D. W.........Air Base, Wyoming
Feinberg, Philip............Minneapolis
§ Feinstein, Julius Y............Minneapolis
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Field, Charles W............Minneapolis
§ Fingerman, David L............Minneapolis
§ Fink, Leo Wm............Oak Terrace
§ Fink, Walter H............Oak Terrace
§ Fisher, Isadore I............Oak Terrace
† Fitzgerald, D. F............Wayzata
†§Fjeldstad, Christian A............Minneapolis
§ Fleeson, Wm. H............Minneapolis
† Fleming, A. S............Madison. N. J.
Fleming, Dean S............Hopkins
§ Fliehr, Richard R............Minneapolis
Flink, Edmund B............Minneapolis
§ Foker, Leslie W............Minneapolis
§ Folsom, Louis B............Minneapolis
§ Ford, Wm. H............Minneapolis
§ Foster, Orley W............Minneapolis
Fowler, Lucius H............Minneapolis
§ Fox, James R............Minneapolis
§ Frane, Donald B............Minneapolis
† Frear, Rosemary............Minneapolis
§ Fredericks, Geo. M............Minneapolis
Freeman, Craig W............Minneapolis
§ Freeman, Donald W............Minneapolis
§ French, Lyle A............Minneapolis
§ Frey, Richard J............Minneapolis
Friberg, Jos............Minneapolis
§ Fried, Louis A............Minneapolis
§ Friedell, Aaron............Minneapolis
§ Friedman, Harry S............Minneapolis
§ Friedman, Jack............Minneapolis
Friend, Charles A............Minneapolis
§ Frost, John B............Minneapolis
§ Frost, Russell H............Minneapolis
§ Frykman, Howard M............Minneapolis
Fuller, Alice H............Minneapolis
§ Funk, Victor K............Oak Terrace
Furman, Lucie C............Minneapolis
§ Gaard, Richard C............Minneapolis
§ Gallett, L. E............Minneapolis
† Galligan, Margaret............Minneapolis
§ Galloway, John B............Minneapolis

§ Gammell, John H........Minneapolis
§ Garske, George L........Minneapolis
Garten, Joseph L........Minneapolis
§ Gaviser, David........Minneapolis
§ Gibbs, Robert W........Minneapolis
§ Giebenhain, John N........Minneapolis
§ Giere, Jos. C........Minneapolis
§ Giere, Richard W........Minneapolis
Gilbert, Maurice G........Minneapolis
Gingold, Benj. A........Minneapolis
† Girvin, Richard B........Minneapolis
§ Goldberg, I. M........Minneapolis
§ Goldman, T. I........Minneapolis
§ Goldner, Meyer Z........Minneapolis
§ Goltz, Robert........Minneapolis
§ Good, Hoff D........Minneapolis
Gordon, Philip E........Minneapolis
Gordon, Sewell S........St. Louis Park
‡§ Grais, Melvin L........
Colorado Springs, Colo.
Grant, Suzanne........Minneapolis
§ Gratzek, F. R. E........Minneapolis
Gray, Royal C........Minneapolis
Green, Robert A........St. Louis Park
§ Greenberg, A. J........Minneapolis
§ Greenfield. Irving........Minneapolis
† Greishiemer, E. M........Philadelphia, Pa.
Grimes, Marian........Minneapolis
§ Gronvall, Paul R........Minneapolis
§ Grotting, J. K........Minneapolis
‡ Gullickson, Glen, Jr........Bethesda, Md.
§ Gunlaugson, E. G........Minneapolis
§ Gushurst, Edward G........Minneapolis
§ Gustason, Harold T........Minneapolis

Haberer, Helen R........Minneapolis
§ Hagen, Wayne S........Minneapolis
† Haggard, G. D........Minneapolis
§ Hall, Harry B........Minneapolis
§ Hall, Wendell H........Minneapolis
Hallberg, Chas. A........Minneapolis
§ Hannah, Hewitt B........Minneapolis
§ Hansen, Cyrus O........Minneapolis
§ Hansen, Erling W........Minneapolis
Hansen, Olga S........Minneapolis
‡§Hansen, R. M........Falls Church, Va.
§ Hanson, Harlow J........Minneapolis
Hanson, Henry V........Minneapolis
§ Hanson, Malcolm B........Minneapolis
§ Hanson, Mark C. L........Minneapolis
§ Hanson, Wm. A. L........Minneapolis
§ Happe, Laurence J........Minneapolis
Harris, L. D........Minneapolis
Hartman, Evelyn........Minneapolis
§ Hass, Fredk. M........Minneapolis
§ Hastings, Def. R........Minneapolis
§ Hastings, Donald W........Minneapolis
§ Hauge, Erling T........Minneapolis
‡§Haugen, George W........
San Francisco, Calif.
Haugen, John A........Minneapolis
Hauser, George W........Minneapolis
‡ Havel, Robert J........Minneapolis
§ Haven, Walter K........Minneapolis
§ Hawkinson, R. P........Minneapolis
Hayes, Jame M........Minneapolis
§ Hays, Albert T........Minneapolis
§ Head, Douglas P........Minneapolis
§ Hebbel, Robert........Minneapolis
‡§Heisler, John J........Minneapolis
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§ Henrikson, E. C........Minneapolis
† Henry, Clifford E........Kirksville, Mo.
* Henry, M. O........Minneapolis
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§ Hermann, Harold W........Minneapolis
†§Higgins, John H........Minneapolis
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§ Hill, Elmer M........Minneapolis
†§Hillis, S. J........East Bradenton, Fla.
Hinckley, Robert G........Minneapolis
§ Hirshfield, Frank R........Minneapolis
§ Hitchcock, Claude R........Minneapolis
Hoffbauer, F. W........Minneapolis
§ Hoffert, Henry E........Minneapolis
§ Hoffman, R. E........Minneapolis
§ Hoffman, Walter L........Minneapolis
§ Holmberg, Conrad J........Minneapolis
§ Holzapfel, Fred C........Minneapolis
‡ Horns, Howard L...Colo. Springs, Colo.
§ Horns, Richard C........Minneapolis
† Houkom, Bjarne........T. T. East Africa
‡ Houle, Rollin J........New Brighton
Hovland,. Melvin L........Minneapolis
Howard, Robert B........Minneapolis
Howard, S. E........Minneapolis
§ Howell, Carter W........Minneapolis
§ Huenekens, E. J........Minneapolis
*†§Hultkrans, J. C........Minneapolis
§ Hultkrans, R. E........Minneapolis

† Hurd, Annah........Minneapolis
‡ Hutchinson, C. J........Providence, R. I.
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† Hynes, John E........Minneapolis

Ide, Arthur W., Jr........Minneapolis
§ Idstrom, Linneus G........Minneapolis
† Irvine, Harry G........Minneapolis
§ Iverson, Rolf M........Minneapolis

Jacobson, Loren J........Minneapolis
§ Jacobson, W. E........St. Louis Park
§ Jay, Alan R........Minneapolis
‡ Jensen, Adrian R.....Long Island, N. Y.
§ Jensen, Harry C........Minneapolis
§†Jensen, M. J........Minneapolis
§ Jensen, N. K........Minneapolis
Jensen, Reynold A........Minneapolis
Jerome, Bourne........Minneapolis
Jerome, Elizabeth........Minneapolis
‡ Jeub, R. P........San Antonio, Texas
§ Johnson, Arthur B........Minneapolis
§ Johnson, August E........Minneapolis
§ Johnson, Emil W........Minneapolis
Johnson, Frank E........Minneapolis
§ Johnson, Harry A........Minneapolis
§ Johnson, James A........Minneapolis
§ Johnson, John W........Minneapolis
§ Johnson, Malcolm R........Minneapolis
Johnson, Norman P........Minneapolis
§ Johnson, Norton T........Minneapolis
§ Johnson, Reinald G........Minneapolis
§ Johnson, Reuben A........Minneapolis
§ Johnson, Robert E........Minneapolis
Johnson, Roger S........Wayzata
Johnson, Youbert T........Minneapolis
§ Jones, David G........Minneapolis
§†Jones, H. W., Jr........Minneapolis
§ Jones, Richard........Minneapolis
§ Jones, Wm. R........Minneapolis
Josewich, Alexander........Minneapolis
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§ Judd, W. H........Washington, D. C.
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§ Jurdy, Mitchell J........Minneapolis

Kadesky, Harold B........Minneapolis
§ Kalin, Oscar T........Minneapolis
§ Kallestad, Leonard L........Wayzata
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§ Karleen, Conrad I........Minneapolis
† Katz, Yale J........Minneapolis
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§ Kelly, John P........Minneapolis
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† Kennedy, Jane F........Minneapolis
§ Kerkhof, Arthur C........Minneapolis
Kiesler, Frank, Jr........Minneapolis
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King, Frances W........Oak Terrace
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† Knight, Ray R........Minneapolis
Knudsen, Helen L........Minneapolis
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Koller, Herman........Minneapolis
Koller, Louis R........Minneapolis
Koller, Robert........Minneapolis
Korchik, John P........Minneapolis
Kosiak, John........Minneapolis
Kottke, Frederic J........Minneapolis
§ Koucky, Rudolph W........Minneapolis
§ Kovack, Freeman D........Minneapolis
§ Kremen, Arnold J........Minneapolis
§ Kucera, Frank J........Hopkins
§ Kucera, Wm. J., Sr........Minneapolis
§ Kucera, W. J., Jr........Minneapolis
§ Kusz, Clarence V........Minneapolis

§ LaBree, John........St. Louis Park
Lagaard, Sheldon M........Minneapolis
Lajoie, John M........Minneapolis
§ Lamb, H. Douglas........Minneapolis
§ Lang, Leonard A........Minneapolis
Lange, Robert D........Minneapolis
§ Lapierre, Arthur P........Minneapolis
Lapierre, Jean T........Minneapolis
§ Larsen, Frank W........Minneapolis
§ Larson, Clarence M........Minneapolis
§ Larson, Donald M........Minneapolis
§ Larson, Lawrence M........Minneapolis
§ Larson, Leonard M........Oak Terrace
§ Larson, Loren........Minneapolis
Larson, Ralph H........Anoka
Larson, Roger C........Minneapolis
† LaVake, Rae T........Minneapolis
§ Laymon, Carl W........Minneapolis
† Lazar, Henry L........Excelsior
† Leavitt, H. H........Phoenix, Ariz.
Lebowske, Jos. A........Minneapolis
§ Leemhuis, Andrew J........Minneapolis
§ Leland, Harold R........Minneapolis
§ Lenz, Otto A........Minneapolis
§ Leonard, Lawrence J........Minneapolis
§ Leonard, Saml........Minneapolis
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Lillehei, Elmer J........Robbinsdale
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§ Lindberg, Vernon L........Minneapolis
Lindberg, Winston R........Minneapolis
Lindemann, Charles E........Minneapolis
Lindgren, Russell C........Minneapolis
Lindner, Janus C........Minneapolis
§ Lindquist, Richard H........Minneapolis
§ Linner, Gunnar........Minneapolis
§ Linner, Henry P........Minneapolis
§ Linner, John H........Minneapolis
§ Linner, Paul W........Minneapolis
Lippman, Emanuel S........Minneapolis
§ Lipschultz, Oscar........Minneapolis
§ Litchfield, John T........Minneapolis
Litman, Abraham B........Minneapolis
Lober, Paul H........Minneapolis
§ Lofsness, S. V........Oak Terrace
†§Logefeil, Rudolph C........Minneapolis
Loomis, Earl A........Minneapolis
§ Lott, F. H........Minneapolis
Lovett, Beatrice R........Oak Terrace
Lowry, Elizabeth........Minneapolis
§ Lowry, Paul........Minneapolis
Lowry, Thomas........Minneapolis
§ Lueck, W. W........Minneapolis
§ Lufkin, Nathaniel H........Minneapolis
Lund, George........Minneapolis
Lundberg, Ruth I........Minneapolis
Lundblad, Roy A........Minneapolis
Lundblad, S. W........Minneapolis
*§Lundgren, A. C........Minneapolis
§ Lundquist, V. J. P........Minneapolis
† Lynch, Matthew J........Minneapolis
Lysne, Henry........Minneapolis
§ Lysne, Myron........Minneapolis
Lysyj, Anatol........Minneapolis

§ MacDonald, D. A........Minneapolis
MacDonald, John W........Minneapolis
§ Mach, Frank B........Minneapolis
§ MacKinnon, Donald C........Minneapolis
*†Macnie, John S........Minneapolis
§ Maeder, Edward C........Minneapolis
§†Maland, Clarence O........Minneapolis
Maloney, Wm. F........Minneapolis
Mandel, Sheldon L........Minneapolis
§ Mankey, James C........Minneapolis
§ Marking, George H........Minneapolis
§ Martin, George R........Minneapolis
Martinson, Carl J........Wayzata
Martinson, Elmer J........Wayzata
†*Matchan, Glenn R........Saint Paul
§ Mattill, Peter M........Oak Terrace
§ Mattson, H. A. N........Minneapolis
§ Maxeiner, S. R........Minneapolis
§ McCaffrey, Fabein J........Minneapolis
§ McCann, Eugene J........Minneapolis
McCannel, M. A........Minneapolis
McCarthy, Donald........Saint Paul
McCartney, J. S........Minneapolis
§ McCormick, D. P........Minneapolis
† McDaniel, Orianna........Minneapolis
§ McFarland, A. H........Minneapolis
§ McGandy, Robt. F........Minneapolis
§ McGeary, Geo. E........Minneapolis
§ McInerny, M. W........Minneapolis
McKelvey, John L........Minneapolis
§ McKenzie, Chas. H........Minneapolis
§ McKinley, C. A........Minneapolis
§ McKinney, F. S........Minneapolis
§ McLaughlin, B. H........Minneapolis
§ McMurtrie, Wm. B........Minneapolis
McNeil, John J........Minneapolis
§ McPheeters, H. O........Minneapolis
† McQuarrie, Irvine........Minneapolis
§ Meller, Robert L........Minneapolis
§ Merkert, Chas. E........Minneapolis
§ Merkert, Geo. L........Minneapolis
† Merrick, Charlotte T........St. Paul
§ Meyer, Alvin J........Minneapolis
† Meyer, Ette L........Minneapolis
Meyer, Robert J........Minneapolis
Michael, Jos. C........Minneapolis
§ Michel, Henry H........Minneapolis
† Michelsen, Emma F........Minneapolis

§ Michelson, Henry E.	Minneapolis
§ Miller, Arden L.	Minneapolis
§ Miller, Harold E.	Minneapolis
§ Miller, Hugo E.	Minneapolis
§ Miller, John C.	Minneapolis
§ Millett, D. Keith	Minneapolis
§ Milton, John S.	Minneapolis
Minsky, Armen A.	Minneapolis
Mintz, Charles M.	Minneapolis
§ Mitby, Irving	Minneapolis
§ Mitchell, Berton D.	Minneapolis
Mitchell, E. C.	Minneapolis
§ Mitchell, M. T.	Minneapolis
‡§Mixer, Harry W.	Fort Hood, Texas
§ Moe, John H.	Minneapolis
§ Moehn, John T.	Minneapolis
§ Moen, J. K., Jr.	Minneapolis
† Monahan, Elizabeth S.	Minneapolis
§ Monson, Einer M.	Minneapolis
§ Moore, Irvin H.	Minneapolis
Moorhead, Marie	Minneapolis
§ Moos, Daniel J.	Minneapolis
*†Moren, Edward	Minneapolis
§ Mork, Arthur H.	Anoka
§ Mork, Frank E.	Anoka
Morrison, Charlotte	Minneapolis
§ Mosser, Donn G.	Minneapolis
§ Moyer, Leonard B.	Minneapolis
§ Mulholland, Wm. M.	Minneapolis
Murphy, E. P.	Minneapolis
† Murray, Elizabeth M.	Minneapolis
Muske, Marvin M.	Minneapolis
§ Musty, Nicholas J.	Minneapolis
§ Myers, Jay A.	Minneapolis
§ Myhre, James A.	Minneapolis
§ Naslund, Ames W.	Minneapolis
Nauth, Bernard S.	Minneapolis
§ Neal, Joe M.	St. Paul
§ Neary, Richard P.	Minneapolis
Nelson, Bernette G.	Minneapolis
Nelson, Bernice A.	Minneapolis
Nelson, C. Barton	Minneapolis
§ Nelson, Edward N.	Minneapolis
§ Nelson, Harvey	Minneapolis
§ Nelson, L. S.	Minneapolis
Nelson, Maxine O.	Minneapolis
§ Nelson, Maynard C.	Minneapolis
§ Nelson, O. L. N.	Minneapolis
§ Nelson, Wallace I.	Minneapolis
§ Nerenberg, Samuel T.	Minneapolis
§ Nesbitt, Saml.	Minneapolis
§ Nesset, Lauren B.	Minneapolis
§ Nesset, Wm. D.	Minneapolis
§ Neumeister, Charles	Minneapolis
§ Nice, Charles M.	Minneapolis
Noonan, Wm. J.	Minneapolis
Noran, Axel S. N.	Minneapolis
§ Noran, Harold H.	Minneapolis
§ Nord, Robert E.	Minneapolis
§ Nordin, Gustaf T.	Minneapolis
§ Nordland, Martin, Sr.	Minneapolis
§ Nordland, Martin, Jr.	Minneapolis
§ Norum, Henry A.	Minneapolis
Norval, Mildred	Minneapolis
† Noth, Henry W.	Minneapolis
§ Nydahl, Malvin J.	Minneapolis
Nylander, E. G.	Minneapolis
O'Donnell, James E.	Minneapolis
§ O'Hanlon, John A.	Minneapolis
§ O'Phelan, E. Harvey	Minneapolis
§†Oberg, Carl M.	Minneapolis
Olavs, Olga	Minneapolis
§ Olsen, Edw. G.	Minneapolis
§ Olson, Alton C.	Minneapolis
§ Olson, Carl J.	Minneapolis
Olson, C. Kent	Minneapolis
Olson, Detlof M.	Minneapolis
† Olson, Olof A.	Minneapolis
§ Oppen, E. Gerhard	Minneapolis
§ Oppen, Melvin G.	Wayzata
§ Opstad, Earl T.	Oak Terrace
§ Palen, Benjamin J.	Minneapolis
Papermaster, T. C.	Minneapolis
Park, Wilford E.	Minneapolis
Peltier, Leonard F.	Minneapolis
‡§Peluso, Charles R.	Phoenix, Ariz.
§ Peppard, Thos. A.	Minneapolis
§ Perlman, Herschel L.	Minneapolis
§ Perlman, Everett C.	Minneapolis
§ Peteler, Jennings C. L.	Minneapolis
§ Petersen, Glenn L.	Minneapolis
*§†Petersen, John R.	Minneapolis
Petersen, Peter C.	Minneapolis
§ Petersen, William E.	Minneapolis
Peterson, Henry	Minneapolis
§ Peterson, Herbert W.	Minneapolis
§ Peterson, Nordahl P.	Minneapolis
Peterson, O. H., Sr.	Minneapolis
†§Peterson, O. H., Jr.	Minneapolis
§ Peterson, Palmer A.	Minneapolis
§ Peterson, Peter E.	Minneapolis
§ Peterson, Willard C.	Minneapolis
§ Petit, Julien V.	Minneapolis
§ Petit, Leon J.	Minneapolis
§ Pewters, John T.	Minneapolis
§ Peyton, Wm. T.	Minneapolis
§ Phelps, Kenneth A.	Minneapolis
Plasha, Matthew K.	Anoka
§ Plass, H. F. R.	Minneapolis
§†Platou, Erling S.	Minneapolis
§ Plimpton, N. C., Jr.	Minneapolis
§ Pohl, John F. M.	Minneapolis
†§Pollock, David K.	Minneapolis
§ Polzak, Jacob A.	Minneapolis
† Poppe, F. H.	Minneapolis
Potek, David	Minneapolis
§ Potter, R. B.	Minneapolis
† Pratt, F. J., Sr.	Minneapolis
‡§Pratt, F. J., Jr.	N. Y., N. Y.
Preine, Irving A.	Osseo
§ Preston, Paul J.	Minneapolis
§ Priest, Robt. E.	Minneapolis
§†Prim, Jos. A.	Minneapolis
§ Proffit, Wm. E.	Minneapolis
§ Proshek, Chas. E.	Minneapolis
§ Quello, Robert O.	Minneapolis
§†Quist, Henry W., Sr.	Minneapolis
§ Quist, H. W., Jr.	Minneapolis
§ Ransom, H. R.	Osseo
§ Reader, Donald R.	Minneapolis
§ Regnier, Edward A.	Minneapolis
Reid, Lewis M.	Minneapolis
§ Reif, Harold A.	Minneapolis
§ Reiley, Richard E.	Minneapolis
Reitmann, John H.	Anoka
Remole, William D.	Minneapolis
§ Resch, Joseph A.	Minneapolis
§ Rice, Carl O.	Minneapolis
Rice, Frank B.	Minneapolis
§ Rice, Fred A.	Minneapolis
§ Richdorf, Lawrence F.	Minneapolis
§ Rieke, W. W.	Wayzata
Rigler, Leo G.	Minneapolis
Riordan, Elsie M.	Minneapolis
§ Risch, Ronald E.	Minneapolis
§ Rizer, Dean K.	Minneapolis
Rizer, Robert I.	Minneapolis
Robb, Edwin F.	Minneapolis
§ Robbins, Owen F.	Minneapolis
Roberts, Byron H.	Minneapolis
§ Roberts, Lewis J.	Minneapolis
§ Roberts, Stanley W.	Minneapolis
†§Roberts, Wm. B.	Minneapolis
Robinson, C. O.	Minneapolis
§ Rocknem, Robert E.	Minneapolis
§ Rockwell, C. V.	Minneapolis
† Rodda, Fredk. C.	Minneapolis
§ Rodgers, Richard S.	Minneapolis
‡ Romness, Kenneth	Mound
† Rosander, Phyllis	Minneapolis
§ Rosenbaum, D. L.	Minneapolis
§ Rosendahl, F. G.	Minneapolis
§ Rosenfield, A. B.	Minneapolis
§ Rosenow, John H.	Minneapolis
† Rosenwald, R. M.	Anoka
§ Ross, A. J.	Minneapolis
Rossen, Ralph	Hastings
§ Rothnem, Morris	Minneapolis
§ Rucker, Wm. H.	Minneapolis
Rud, Norman E.	Minneapolis
Rudell, Gustaf	Minneapolis
§ Russeth, Arthur N.	Minneapolis
§ Rusten, Elmer M.	Minneapolis
Rydburg, Wayne C.	Minneapolis
§ St. Cyr, Harry M., Jr.	Robbinsdale
§ St. Cyr, Kenneth J.	Robbinsdale
§ Sadler, Wm. P., Jr.	Minneapolis
§ Saliterman, B. I.	Minneapolis
§ Samuelson, Sml.	Minneapolis
§ Sandt, Karl E.	Minneapolis
§ Sawatzky, W. A.	Minneapolis
§ Sborov, A. M.	Minneapolis
§ Schaaf, F. H. K.	Minneapolis
Schaar, Frances E.	Minneapolis
Schaefer, W. G.	Minneapolis
† Scheldrup, N. H.	Minneapolis
§ Scherer, Leslie R.	Minneapolis
Scherling, Sidney S.	Minneapolis
§ Schiele, Burtrum C.	Minneapolis
Schissel, Gregory A.	Minneapolis
§ Schmidt, W. R.	Minneapolis
† Schmitt, S. C.	El Cajon, Calif.
Schneck, Jack I.	Waukesha, Wisc.
§ Schottler, Max E.	Minneapolis
§ Schroeder, A. J.	Minneapolis
Schultz, Donald O.	Minneapolis
§ Schultz, J. H.	Minneapolis
§ Schultz, P. J.	Minneapolis
§ Schulze, Wm. M.	Minneapolis
Schumacher, John	Minneapolis
§ Schwartz, E. R.	Minneapolis
§ Schwartz, Virgil J.	Minneapolis
§ Scott, Horace G.	Minneapolis
§ Seaberg, John A.	Minneapolis
Seham, Max	Minneapolis
Seifert, Milton H.	Minneapolis
§ Seljeskog, S. R.	Minneapolis
§ Semsch, Robert D.	Minneapolis
§ Shandorf, James F.	Minneapolis
Shaperman, Eva P.	Minneapolis
§ Shapiro, Sidney K.	Minneapolis
§ Sharp, David	Minneapolis
§ Shaw, Howard A.	Minneapolis
§ Shea, Andrew W.	Minneapolis
§ Sher, Lewis	Minneapolis
§ Sherman, Lloyd F.	Minneapolis
Shillington, M. A.	Minneapolis
§ Shronts, John F.	Minneapolis
§ Siegmann, Wm. C.	Minneapolis
Silas, Ralph M.	Minneapolis
Silver, John D.	Minneapolis
§†Simons, Jalmar	Minneapolis
§ Simmonson, Donald B.	Minneapolis
† Simpson, E. De	Phoenix, Ariz.
§ Sinykin, M. B.	Minneapolis
Siperstein, D. M.	Minneapolis
‡§Sisterman, Thomas J.	Minneapolis
† Sivertsen, Andrew	Minneapolis
†§Sivertsen, Ivar	Minneapolis
§ Skjold, Arthur C.	Minneapolis
† Smiley, John T.	Minneapolis
§ Smisek, F. M. E.	Minneapolis
Smith, Adam M.	Minneapolis
§ Smith, Archie M.	Minneapolis
§ Smith, Baxter A., Jr.	Minneapolis
§ Smith, Graham G.	Minneapolis
† Smith, Homer R.	Minneapolis
† Smith, Margaret I.	Robbinsdale
§ Smith, Norvin R.	Willmar
§ Smith, Theo. S.	Minneapolis
§ Smith, Wm. T.	Minneapolis
§ Soderlind, Ragnar	Minneapolis
§ Solhaug, Saml. B.	Minneapolis
§ Solvason, Harold M.	Minneapolis
§ Spano, Jos. P.	Minneapolis
Spencer, Bernard J.	Minneapolis
§ Spink, Wesley W.	Minneapolis
§ Sponsel, Kenath	Minneapolis
† Spratt, Chas. N.	Minneapolis
§ Stahr, Aubrey C.	Hopkins
§ Stanford, Charles E.	Minneapolis
‡ Staub, Henry P.	Minneapolis
† Steigler, Farrell S.	St. Paul
§ Stelter, Lloyd A.	Minneapolis
§ Stennes, John L.	Minneapolis
Stenstrom, Annette E. T.	Minneapolis
Sterrie, Norman A.	Minneapolis
Stewart, Marvin J.	Minneapolis
§ Stewart, Rolla I.	Minneapolis
§ Stillwell, George K.	Minneapolis
§ Stoesser, Albert V.	Minneapolis
Stoltz, Robert C.	Minneapolis
† Stomel, Joseph	Mayview, Pa.
§ Stone, Stanley P.	Minneapolis
† Strachauer, A. C.	Minneapolis
§ Strickler, Jacob H.	Minneapolis
§ Strom, Gordon W.	Minneapolis
§ Stromme, William B.	Minneapolis
Stromgren, Delph T.	Minneapolis
Stroomgren, Delph T.	Minneapolis
Strunk, Clarence A.	Minneapolis
§ Sturges, Robert L.	Minneapolis
§†Subby, Walter	Minneapolis
Sukov, Marvin	Minneapolis
§ Swain, Francis M.	Minneapolis
§ Swanson, Roy E.	Minneapolis
§ Sweetser, Horatio B.	Minneapolis
§ Sweetser, Theo. H.	Minneapolis
Sweetser, Theo, H., Jr.	Minneapolis
† Sweitzer, Saml. E.	Minneapolis
†§Swendseen, Carl G.	Minneapolis
Syverton, Jerome T.	Minneapolis
Tam, Ernest C.	Minneapolis
§ Tangen, George M.	Minneapolis
Taylor, Jos. H.	Minneapolis
§ Tenner, Robert J.	Minneapolis
† Thomas, George E.	Minneapolis
Thomes, A. Boyd	Minneapolis
§ Thompson, Willis H.	Minneapolis
§ Thorsen, David S.	Minneapolis
§ Thorson, Stuart V.	Minneapolis
§ Thysell, D. M.	Minneapolis
Tichy, Fae Y.	Minneapolis
† Tingdale, A. C.	Minneapolis
§ Tinkham, Robert G.	Minneapolis
§ Titrud, Leonard	Minneapolis
§ Tobin, John D.	Minneapolis
§ Trach, Benedict B.	Minneapolis
§ Trow, James E.	Minneapolis
§ Trow, Wm. H.	Minneapolis
Trueman, Harold S.	Minneapolis

Tsai, Shih Hao..........Oak Terrace
§ Tudor, Richard B..........Minneapolis
*†Tundstead, H. J..........Minneapolis
†§Turnacliff, D. D..........St. Paul
Twomey, John E..........Minneapolis
§ Ude, Walter H..........Minneapolis
†§Ulrich, Henry L..........Minneapolis
§ Ulvestad, Harold S..........Minneapolis
§ Undine, Clyde A..........Minneapolis
Van Buskirk, Chas..........Minneapolis
Vermund, Halvor..........Minneapolis
Vik, A. Elliott..........Minneapolis
Vitols, T. M..........Minneapolis
§ Wahlquist, H. F..........Minneapolis
Waldron, C. W..........Minneapolis
§ Wall, Carl R..........Minneapolis
§ Walonick, Albert L..........St. Louis Park
§ Walsh, Francis M..........Minneapolis
§ Walsh, Wm. T..........Minneapolis
Wangensteen, O. H..........Minneapolis
Ward, Percy A..........Minneapolis
§ Watson, C. Gordon..........Minneapolis
Watson, Cecil J..........Minneapolis

† Weaver, M. Mc..........Vancouver, B. C., Canada
§ Webb, Edgar A..........Minneapolis
Webb, Roscoe C..........Minneapolis
§ Webber, Richard J..........St. Louis Park
§ Weber, Lowell..........Minneapolis
§ Weisberg, R. J..........Minneapolis
§ Wendland, John P..........Minneapolis
† Werner, George..........Minneapolis
† West, Catherine C..........Minneapolis
*§†Wethall, Anton G..........Minneapolis
Wetherby, Macnider..........Minneapolis
Wheeler, Robert W..........Minneapolis
§ White, Asher A..........Minneapolis
§ White, Willard D..........Minneapolis
§ Whitesell, L. A..........Minneapolis
§ Widen, Wilford F..........Minneapolis
*†Wilcox, Archa E..........Minneapolis
† Wilder, Kenneth W..........Minneapolis
§ Wilder, Robert L..........Minneapolis
§ Wilder, R. M., Jr..........Minneapolis
§ Wilken, Paul A..........Minneapolis
† Willcutt, C. E..........Phoenix, Ariz.

† Williams, Robert..........New York, N. Y.
Winchell, Paul..........Minneapolis
Winther, Nora M. C..........Minneapolis
§ Wippermann, Frederic F..........Minneapolis
§ Witham, Carl A..........Minneapolis
Wittich, Fredk. W..........Minneapolis
§ Wohlrabe, Arthur A..........Minneapolis
§ Wohlrabe, A. Cabot..........Minneapolis
§ Wolf, Alfred H..........Minneapolis
Wolter, Frederick H..........Minneapolis
Wright, Thomas D..........Minneapolis
§ Wright, Wale S..........Minneapolis
§ Wright, Wm. S..........Minneapolis
Wyatt, Oswald S..........Minneapolis
Wynne, H. M. N..........Minneapolis
§ Ylvisaker, R. S..........Minneapolis
§ Yoerg, Otto W..........Minneapolis
Zahrendt, O. Lewis..........Minneapolis
§ Zarling, V. Richard..........Minneapolis
§ Zaworski, Leo A..........Minneapolis
§ Zierold, Arthur A..........Minneapolis
§ Zinter, F. A..........Minneapolis
§ Ziskin, Thos..........Minneapolis

KANDIYOHI-SWIFT-MEEKER COUNTY MEDICAL SOCIETY

Regular meetings, third Thursday of each month
Annual meeting, November
Number of Members—49

President
Griffin, R. P..........Benson

Secretary
Opsahl, Lawrence J..........Willmar

Allison, David D..........Litchfield
Anderson, Richard E..........Willmar
Austrian, Sol..........Cosmos
Bosland, Howard G..........Willmar
Bradley, N. J..........Willmar
† Chadbourn, Wayne A..........Minneapolis
Chunn, Stanley S..........Willmar
† Daignault, Oscar..........Benson
† Danielson, Karl A..........Litchfield
Dille, Donald E..........Litchfield
Douglas, Kenneth W..........Willmar
Eberley, Tobe S..........Benson
Ellinger, Albert J..........Willmar

Frederickson, Alice C..........Willmar
† Frost, Edward H..........Willmar
Giere, Silas W..........Benson
Gilman, Lloyd C..........Willmar
Griffin, R. P..........Benson
‡ Guy, Jack A..........New London
Herbst, Richard F..........Willmar
Hinz, Walter E..........Willmar
Hodapp, Robert V..........Willmar
Holm, Donald..........Benson
Houts, Joseph C..........Dassel
Jacobs, Douglas L..........Willmar
†§Jacobs, Johannes C..........Willmar
* Katzberg, Lewis..........Willmar
Kaufman, Edward J..........Appleton
Lundblad, Robert M..........Clara City
Macklin, William, Jr..........Willmar
McCarthy, Austin M..........Willmar
Michels, Roger P..........Willmar

† O'Connor, Daniel C..........Eden Valley
Opsahl, Lawrence J..........Willmar
Ostling, Burton C..........Kerkhoven
Pedersen, Robert L..........Brainerd
† Penhall, Fletcher W..........Willmar
Peterson, Willard E..........Willmar
Proeschel, Ray K..........Willmar
Rorem, Joseph A..........Appleton
Rygh, Harold N..........Atwater
Schnell, Frederick S..........Litchfield
Schutz, John C..........Eden Valley
* Sellers, G. K..........Dassel
Solsem, F. N. S..........Spicer
Sommerness, M. Duane..........Willmar
Sorum, F. T..........Willmar
Sutherland, W. H..........New London
Verby, John E., Jr..........Litchfield
Wilmot, Cecil A..........Litchfield
Wilmot, Harold E..........Litchfield

LYON-LINCOLN COUNTY MEDICAL SOCIETY

Regular meetings, last Tuesday of September, October, April and May
Annual meeting, last Tuesday of October
Number of Members—27

President
Hedenstrom, Philip C..........Marshall

Secretary
Purves, George H..........Hendricks

Bodaski, Albert A..........Tyler
Eckdale, John E..........Marshall
Ferguson, William C..........Walnut Grove
Ford, Burton C..........Marshall
Friedell, George..........Ivanhoe
† Gray, Frank D..........Marshall

Hedenstrom, Philip C..........Marshall
Helferty, J. K..........Boise, Idaho
Hermanson, Peter E..........Hendricks
† Hoidale, Andrew D..........Tracy
Johnson, C. Percy..........Tyler
Kreuzer, Titus..........Marshall
Lee, Norman J..........Tracy
Monson, Leonard J..........Canby
Murphy, Joseph..........Marshall
Peterson, Kenneth A..........Marshall
Purves, George H..........Hendricks

Remsberg, R. R..........Tracy
*†Robertson, J. B..........Minneapolis
Sether, Arthur F..........Ruthton
Smith, Lloyd A..........Willmar
Thompson, Carl O..........Hendricks
Turbak, Charles E..........Canby
§ Vadheim, Alfred L..........Tyler
†§Valentine, Walter H..........Tracy
Wolstan, Simon D..........Minneota
Workman, Warner G..........Tracy
Yaeger, Wilbert W..........Marshall

McLEOD COUNTY MEDICAL SOCIETY

Regular meeting, third Thursday of each month
Annual meeting, third Thursday in December
Number of Members—18

President
Griebie, Grant L..........Brownton

Secretary
Smyth, John J..........Lester Prairie

§ Brink, Donald M..........Hutchinson
† Clement, John B..........Lester Prairie
§ Griebie, Grant L..........Brownton
*§Holm, Hilliard H..........Glencoe

Howell, Milton..........Glencoe
§ Huebert, Dan W..........Hutchinson
§ Jensen, A. M..........Brownton
§ Klima, William W..........Stewart
*†§Leitschuh, Linus F..........Winsted
§ Lippmann, Elmer W..........Hutchinson
§ Neumeier, Arthur..........Glencoe
§ Peterson, Kenneth H..........Hutchinson

Pone, John..........Silver Lake
Sahr, Walter G..........Hutchinson
Scholpp, Otto W..........Hutchinson
§ Selmo, Joseph D..........Norwood
§ Sheppard, C. G..........Hutchinson
‡§Smith, George R..........Hutchinson
§ Smyth, John J..........Lester Prairie
§ Truesdale, Clark W..........Glencoe

MOWER COUNTY MEDICAL SOCIETY

Regular meeting, last Thursday of each month
Annual meeting, December
Number of Members—36

President	
Twiggs, Leo F.	Austin
Secretary	
Rosenthal, F. H.	Austin
§ Anderson, David P.	Austin
§ Anderson, Harold J.	Austin
§ Anderson, Wallace R.	Austin
§ Barber, Tracy	Austin
§ Cronwell, Bernhard J.	Austin
Elliot, Harold J.	Hayfield
§ Fisch, Herbert M.	Austin
§ Flanagan, Leonard G.	Austin
§ Grise, William B.	Austin
† Havens, John G. W.	Austin
† Hegge, Olav H.	Austin
§ Hegge, R. S.	Austin
† Hertel, Garfield E.	Austin
§ Hesla, Inman A.	Austin
§ Leck, Paul C.	Austin
‡ Lewis, Joyce, Jr.	Minneapolis
§ Lommen, Peter A.	Austin
McKenna, Elizabeth M.	Austin
§ McKenna, Jay K.	Austin
† Melzer, George R.	Lyle
§ Miller, Herman	Grand Meadow
Morse, Morton P.	LeRoy
§ Nesse, J. A.	Austin
§ Osborn, Donald D.	Austin
§ Peterson, Stanley C.	Austin
§ Robertson, Paul A.	Austin
§ Rosenthal, F. H.	Austin
§ Sargent, E. C., Jr.	Austin
Schneider, Paul J.	Adams
§ Seery, Thomas M.	Austin
§ Sheedy, Chester L.	Austin
§ Thomson, James M.	Austin
§ Twiggs, Leo F.	Austin
§ Van Cleve, Horatio	Austin
‡ Wilson, F. C.	Fort Belvoir, Va.
§ Wright, Robert R.	Austin

NICOLLET-LE SUEUR COUNTY MEDICAL SOCIETY

Regular meeting, not scheduled
Annual meeting, December
Number of Members—24

President	
Olson, Duane O.	Gaylord
Secretary	
Gridley, John W.	Arlington
†§Aikens, Herbert B.	LeCenter
§ Branham, Donald S.	St. Peter
§ Canfield, Wayne W.	St. Peter
§ Curtis, Reuben A.	LeCenter
† Erickson, Swan	Le Sueur
*†§Giroux, Alcibiades	Mankato
Gridley, John W.	Arlington
§ Grimes, Burton P.	St. Peter
Henry, M. R.	St. Peter
§ Hiniker, Peter J.	Le Sueur
Kabrick, Ola A.	St. Peter
Kath, Reinhard H.	Arlington
Larson, Milo H.	Lake Benton
§ Lenander, Mellvin E.	St. Peter
§ Nilson, Helmer J.	No. Mankato
§ Olmanson, Edmund G.	St. Peter
§ Olson, Duane O.	Gaylord
‡ Orwoll, Harold S.	Japan
§ Rudie, Clifford N.	St. Peter
§ Sjostrom, Lawrence	St. Peter
§ Sonnesyn, Nels N.	Le Sueur
§ Strathern, Carleton	St. Peter
†§Strathern, Fred P.	St. Peter
Traxler, J. Felix	Henderson
§ Wohlrabe, Clarence	No. Mankato

OLMSTED-HOUSTON-FILLMORE-DODGE COUNTY MEDICAL SOCIETY

Regular meetings, first Wednesday of every other month
Annual meeting, first Monday in November
Number of Members—537

President	
Carryer, Haddon M.	Rochester
Secretary	
Wellman, William	Rochester
‡ Abbott, Albert R.	Rochester
§ Achor, Richard W. P.	Rochester
§ Adams, Richard C.	Rochester
§ Affeldt, Danl. E.	Kasson
† Aga, John H.	Rochester
§ Ahlfs, Jacob J.	Caledonia
Akey, Robert M.	Rochester
§ Allen, E. V. N.	Rochester
Allen, Raymond A.	Rochester
† Amberg, Samuel	Rochester
§ Andersen, Howard A.	Rochester
§ Anderson, Mark J.	Rochester
‡ Anderson, Milton	Rochester
Antel, John J.	Rochester
† Ardan, Nicholas, Jr.	Rochester
§ Atmore, William G.	Rochester
§ Baggenstoss, Archie H.	Rochester
‡ Bain, Robert C.	Oakland, Calif.
Bair, Hugo L.	Rochester
§ Baker, George S.	Rochester
†§Baker, Harry R.	Hayfield
‡ Baker, Hillier L.	West Point, N. Y.
Baker, R. L.	Madison, Wisc.
†§Balfour, Donald C.	Rochester
§ Balfour, William M.	Rochester
§ Banner, Edward A.	Rochester
§ Bargen, J. Arnold	Rochester
§ Barker, Nelson W.	Rochester
Barnes, Arlie R.	Rochester
§ Bastron, James A.	Rochester
‡§Bayard, E. D.	Rochester
§ Beahrs, Oliver H.	Rochester
† Beddow, Ralph M.	Rochester
‡§Bender, L. F.	San Diego, Calif.
§ Bennett, Warren A.	Rochester
Benoit, Clair F.	Rochester
† Berge, Henry B.	Rochester
† Berge, Kenneth G.	Rochester
† Berkman, David	Oronoco
§ Berkman, John M.	Rochester
‡§Bernatz, Philip E.	San Francisco, Calif.
§ Bickel, William H.	Rochester
† Bigelow, Chas. E.	Dodge Center
§ Black, B. M.	Rochester
Blackburn, Chas. M.	Rochester
Block, Melvin A.	Detroit, Mich.
Bonnet, John D.	Rochester
*†Boothby, W. M.	Alberquerque, N. M.
Botham, Richard J.	Rochester
† Bowes, Donald E.	Rochester
§ Boyd, D. A., Jr.	Rochester
† Boyd, George K.	Rochester
§ Braasch, John W.	Rochester
†§Braasch, Wm. F.	Rochester
† Brackett, Ralph D.	Rochester
Brady, Joan	Rochester
Brandenburg, John	Rochester
§ Brandenburg, Robert O.	Rochester
Braun, Robert A.	Rochester
§ Brickley, Paul M.	Rochester
Brindley, Clyde O.	Rochester
Broadbent, James C.	Rochester
‡ Broders, C. W.	Rochester
§ Brown, Alexander	Rochester
§ Brown, Henry A.	Rochester
§ Brown, Joe R.	Rochester
Brown, Philip W.	Rochester
Brown, Roland G.	Rochester
§ Brunsting, Louis A.	Rochester
§ Butt, Hugh R.	Rochester
§ Buie, Louis A.	Rochester
§ Burchell, Howard B.	Rochester
§ Burke, Edmund C.	Rochester
§ Butt, Hugh R.	Rochester
§ Cain, James C.	Rochester
Campagna, Mario J.	Rochester
Campbell, Donald C.	Rochester
§ Carey, John M.	Rochester
§ Carr, David T.	Rochester
§ Carryer, Haddon M.	Rochester
Caruolo, Joseph E.	Rochester
Chance, Donald P.	Rochester
§ Childs, D. S., Jr.	Rochester
† Chiles, N. H.	Rochester
§ Christensen, N. A.	Rochester
§ Clagett, Oscar T.	Rochester
Clark, Doyce M.	Rochester
§ Clark, Leslie W.	Spring Valley
† Clayburgh, Bennie J.	Rochester
Colby, M. Y., Jr.	Rochester
† Collins, Robt. A., Jr.	Rochester
§ Comfort, Mandred W.	Rochester
Connelly, Michael E.	Rochester
Cook, Edward N.	Rochester
§ Cooley, Jack C.	Rochester
Cooney, James F.	Rochester
Corbin, Kendall B.	Rochester
† Costello, Addis C.	Rochester
Counseller, Virgil	Rochester
§ Coventry, Markham B.	Rochester
Craig, Joe D.	Rochester
§ Craig, W. M.	Rochester
*†§Crenshaw, J. L.	Sanford, Fla.
† Crumbley, James J.	Rochester
Culp, Ormond S.	Rochester
§ Dahlin, David C.	Rochester
‡ Daly, David	Rochester
§ Daugherty, Guy W.	Rochester
§ Davis, Austin C.	Rochester
† Davis, Donald R.	Rochester
§ Davis, George D.	Rochester
† Davis, Harry L.	Rochester
Davis, R. E.	Rochester
Dearing, William H.	Rochester
Decker, David G.	Rochester
§ Devine, Kenneth D.	Rochester
§ DeWeerd, James H.	Rochester
§ Diessner, Grant R.	Rochester
‡§Dillard, P. G., Jr.	Camp Carson, Colo.
Dixon, Claude F.	Rochester
Doane, Joseph C.	Rochester
Dobson, Ross A.	Rochester
§ Dockerty, Malcolm B.	Rochester
Dodge, Henry W., Jr.	Rochester
Donin, J. F.	Winnepeg, Mani., Can.
§ Donoghue, Francis E.	Rochester
Dorsey, Cletis S.	Rochester
§ Douglass, Bruce E.	Rochester
Doyle, James R.	Rochester
† Drips, Della G.	Rochester
§ Dry, Thos. J.	Rochester
Dunn, Jack, Jr.	Rochester
DuShane, James Wm.	Rochester
‡ Dykstra, Peter C.	West Point, N. Y.
§ Eaton, Lealdes M.	Rochester
Edwards, Jesse E.	Rochester
§ Elkins, Earl C.	Rochester
§ Ellis, F. Henry	Rochester
† Elstner, Howard L.	Rochester
Emmett, John L.	Rochester
§ Erich, John B.	Rochester

§ Erickson, D. J.	Rochester
Erickson, John G.	Rochester
† Esser, Robert A.	St. Charles, Ill.
‡§Estes, J. E.	San Francisco, Calif.
†§Eusterman, George B.	Rochester
† Evans, E. G., Jr.	Rochester
† Evarts, Arrah B.	Rochester
§ Faber, John E.	Rochester
Faulconer, Albert, Jr.	Rochester
Fergeson, James	Rochester
§ Ferris, Deward O.	Rochester
† Fieldman, E. Jay	Rochester
Figi, Fredk. A.	Rochester
† Fly, Orceneth A., Jr.	Rochester
Foss, Edward L.	Rochester
§ Freedman, Marshall A.	Rochester
Frerichs, Cletus T.	Rochester
†Freymann, John G.	Boston, Mass.
§ Fricke, Robt. E.	Rochester
†§Gambill, Carl M.	Rochesster
Gambill, Earl E.	Rochester
† Garrett, Chas. M., Jr.	Rochester
‡§Gastineau, Chas. F.	Rochester
‡ Gatchell, Frank G.	Savannah, Ga.
‡ Gaunt, William D.	Rochester
Geraci, Jos. E.	Rochester
§ Ghormley, Ralph K.	Rochester
§ Gibb, Robert P.	Rochester
§ Giberson, Raymond G.	Rochester
†§Giffin, Herbert Z.	Rochester
Giffin, Mary E.	Rochester
§ Gifford, R. W., Jr.	Rochester
† Ginsberg, James P.	Rochester
§ Ginsberg, Robert L.	Rochester
‡§Goldstein, N. P.	Bethesda, Maryland
§ Good, C. Allen	Rochester
† Gordon, Donald L.	Rochester
Gossel, John D.	Rochester
‡§Graham, George G.	Millington, Tenn.
Grattan, Robert T.	Rochester
§ Gray, Howard K.	Rochester
Green, Paul A.	Rochester
† Green, Robt. C., Jr.	Rochester
Greene, Laurence F.	Rochester
§ Griffin, G. D. J., Jr.	Rochester
§ Grindlay, John H.	Rochester
‡§Gross, John B.	N. Y., N. Y.
Gulley, John L.	Rochester
Gwinn, John L.	Rochester
§ Habein, Harold C., Sr.	Rochester
Habein, Harold C., Jr.	Rochester
‡§Hagedorn, Albert B.	N. Y., N. Y.
† Haigler, F. H., Jr.	Rochester
§ Haines, Saml. F.	Rochester
§ Hallberg, Olav E.	Rochester
†§Hallenbeck, D. F.	LaJolla, Calif.
§ Hallenbeck, Geo. A.	Rochester
Hanlon, David G.	Rochester
§ Hanson, Norbert O.	Rochester
§ Harrington, Stuart W.	Rochester
§ Harris, Lloyd E.	Rochester
Hartman, Howard R.	Rochester
§ Havens, Fred Z.	Rochester
Hayes, Alvin B.	Rochester
Healy, John R.	Rochester
Heck, Frank J.	Rochester
†§Heilman, D. M. H.	San Diego, Calif.
§ Heilman, F. R.	Rochester
† Heimark, John J.	Rochester
Helden, Rudolph A.	Rochester
† Helland, Gustav M.	Spring Grove
† Helland, John W.	Spring Grove
†§Helmholz, Henry F.	Rochester
†§Hempstead, Bert E.	Rochester
Hench, Philip S.	Rochester
§ Henderson, Edward D.	Rochester
§ Henderson, John W.	Rochester
Henderson, Lowell	Rochester
†§Henderson, Melvin S.	Rochester
† Hendricks, Everett D.	Rochester
Henn, Mary J.	Rochester
Hepper, Norman G.	Rochester
† Hewitt, Edith	Rochester
†§Hewitt, Richard M.	Rochester
†§Heyerdale, O. C.	Rochester
§ Hill, John R.	Rochester
Hines, Edgar A., Jr.	Rochester
Hodgson, Corrin H.	Rochester
§ Hodgson, John R.	Rochester
Hollenhorst, Robert	Rochester
§ Holman, Colin B.	Rochester
§ Holt, Allen H.	Rochester
‡§Hood, Robt. T.	San Francisco, Calif.
Horton, Bayard T.	Rochester
§ Howell, Llewelyn P.	Rochester
§ Huizenga, K. A.	Rochester
§ Hunt, Arthur B.	Rochester
§ Hunt, William	Rochester
§ Hunter, J. S.	Rochester
§ Ivins, John C.	Rochester
§ Jackman, Raymond J.	Rochester
‡ Jackson, Richard L.	Rochester
‡§Jamplis, R. W.	San Francisco, Calif.
§ Janes, Joseph M.	Rochester
Johnson, Adelaide McF.	Rochester
§ Johnson, Carl E.	Rochester
Johnson, Einer W.	Rochester
Johnson, Ralph B.	Lanesboro
Johnston, Edward V.	Rochester
Jones, Richard F.	Rochester
Jones, Rabecliff V.	Rochester
† Jordan, Stanley	Rochester
§ Judd, Edward S., Jr.	Rochester
† Juergens, John L.	Rochester
Kearns, Thomas P.	Rochester
§ Keating, F. R., Jr.	Rochester
§ Keith, Haddow M.	Rochester
†§Keith, Norman M.	Rochester
§ Kennedy, R. L. J.	Rochester
‡ Kent, George B.	Ft. McPherson, Ga.
§ Kernohan, James W.	Rochester
‡§Kiely, James	San Francisco, Calif.
‡ Kiely, Joseph	Joliet, Ill.
§ Kierland, Robt. R.	Rochester
‡ Kilby, Ralph A.	Columbia, So. Car.
Kirby, Thos. J., Jr.	Rochester
§ Kirklin, Byrl R.	Rochester
§ Kirklin, John W.	Rochester
Klakeg, Clayton H.	Rochester
† Klass, Donald W.	Rochester
Knutson, Lewis A.	Spring Grove
§ Koelsche, Giles A.	Rochester
Kolb, Lawrence	Rochester
§ Kroboth, F. J., Jr.	Rochester
‡ Kroll, Harry G.	Rochester
‡§Krout, R. M.	New Cumberland, Pa.
§ Krusen, Frank H.	Rochester
§ Kunkel, Wm. M., Jr.	Rochester
§ Kvale, Walter F.	Rochester
† Lacy, Paul E.	Rochester
§ Lake, Clifford F.	Rochester
† Law, William M.	Rochester
§ Lazarte, Jorge A.	Rochester
§ Leddy, Eugene T.	Rochester
† Lee, Philip R.	Rochester
†§Lemon, Willis S.	Clifton Falls, Va.
Lescoe, Richard J.	Rochester
Lev, Ralph	Rochester
Lewis, Barton L.	Rochester
‡§Lick, Louis C.	San Francisco, Calif.
§ Lillie, Harold I.	Rochester
Lillie, John C.	Rochester
§ Lipscomb, Paul R.	Rochester
§ Litin, E. M.	Rochester
Lofgren, Karl A.	Rochester
†§Logan, Archibald H.	Rochester
§ Logan, George B.	Rochester
§ Love, J. G.	Rochester
§ Lundy, John S.	Rochester
† Lynn, Thomas E.	Rochester
§ MacCarty, Collin S.	Rochester
†§MacCarty, W. C.	Rochester
§ Madison, Mitchell S.	Rochester
§ Magath, Thos. B.	Rochester
Manger, Wm. M.	Rochester
Mankin, Harold T.	Rochester
Mann, Frank D.	Rochester
Mann, Joseph D.	Rochester
‡ Manning, P. R.	Rantoul, Ill.
‡§Markle, G. B., IV	Ft. Monroe, Va.
Marr, Norval M.	Rochester
§ Marten, Theo. G.	Rochester
§ Martin, George M.	Rochester
§ Martin, William J.	Rochester
Masson, Duncan M.	Rochester
†§Masson, James C.	Rochester
§ Masson, James K.	Rochester
Mathieson, Don R.	Rochester
‡§Maxeiner, S. R., Jr.	Ft. Monroe, Va.
† Maxfield, Robert G.	Rochester
§ Mayo, Chas. W.	Rochester
McBean, J. B.	Rochester
McCarran, Samuel P.	Rochester
‡ McClure, R. W., Jr.	Fairmont, W. Va.
McConahey, W. M., Jr.	Rochester
McDonald, John R.	Rochester
McFarlane, John A.	Rochester
McKaig, Carle B.	Pine Island
‡ McKee, Robert E.	Homestead, Pa.
McQuay, N. W.	Rochester
§ McWhorter, Henry E.	Rochester
Mears, Thomas W.	Rochester
Medoff, Alvin S.	Rochester
Menges, Charles G. H.	Rochester
§ Merritt, Wallace A.	Rochester
† Meyerding, Henry W.	Rochester
‡ Miller, J. M.	Detroit, Mich.
§ Miller, R. D.	Rochester
Miller, Ross H.	Rochester
Miller, Wm. J.	Rochester
Millikan, Clark H.	Rochester
§ Mills, Stephen D.	Rochester
§ Moersch, Herman J.	Rochester
§ Montgomery, Hamilton	Rochester
† Moores, Ken D.	Rochester
§ Morlock, Carl G.	Rochester
Morris, Kenneth L.	Rochester
Mortensen, J. D.	Rochester
‡ Mulder, Donald W.	Rochester
Mussey, Mary E.	Rochester
Mussey, Robert D.	Rochester
Myers, C. III	Rochester
† Myers, Richard L.	Rochester
Myers, Thomas T.	Rochester
Myre, Theo. T.	Rochester
† Neary, Angus J.	Rochester
Nehring, Jesse P.	Preston
‡ Nelson, Wm. C.	Rochester
Neuman, H. W.	Rochester
Nichols, Donald R.	Rochester
Norris, Neil T.	Caledonia
‡ O'Keefe, Matthew	Rochester
O'Leary, Paul A.	Rochester
O'Neil, Richard L.	Rochester
‡§O'Shaugnessy, E. J.	Rochester
§ Odel, Howard M.	Rochester
Ogburn, Paul L.	Rochester
‡§Olive, J. T., Jr.	Ft. Riley, Kansas
§ Olsen, Arthur M.	Rochester
Olson, Ernest A.	Pine Island
Olson, Grant E.	West Concord
Onsgard, L. K.	Houston
‡§Openshaw, C. R.	Salt Lake City, Utah
§ Osborn, John E.	Rochester
Owen, Charles A. J.	Rochester
Parker, Harry L.	Rochester
§ Parker, Robt. L.	Rochester
Parkhill, Edith M.	Rochester
‡§Parsons, W. B., Jr.	San Francisco, Calif.
Patrick, Robt. T.	Worrington, Fla.
§ Paulson, John A.	Rochester
§ Paynter, Camen R.	Chicago, Ill.
‡ Pemberton, A. H.	San Francisco, Calif.
† Pemberton, John des	Rochester
Pender, John Wm.	Rochester
§ Perry, Harold	Rochester
Person, Waldemar W.	Rochester
§ Peters, Gustavus A.	Rochester
† Peters, Myra A.	Rochester
Petersen, Arthur	Rochester
§ Petersen, M. C.	Rochester
Peterson, W. H.	Spring Valley
Pettersen, George R.	Mabel
Pettet, John R.	Rochester
Phillips, John W.	Rochester
Piper, Monte C.	LaCanada, Calif.
† Place, Virgil A.	Rochester
Polley, Howard F.	Rochester
§ Pool, Thomas L.	Rochester
Powelson, Myron H.	Rochester
* Powers, F. H.	Rochester
*§Pragen, A. D.	Rochester
‡ Pratt, G. F.	San Francisco, Calif.
§ Pratt, J. H., Jr.	Rochester
§ Prickman, Louis E.	Rochester
§ Priestley, James T.	Rochester
§ Pruitt, Raymond D.	Rochester
§ Pugh, David G.	Rochester
§ Quer, Erich A.	Kansas City, Mo.
Rabens, Robert A.	Rochester
§ Ralston, Donald E.	Rochester
§ Randall, Lawrence M.	Rochester
Randall, Raymond V.	Rochester
§ Rasmussen, W. C.	Rochester
Reitemeier, Richard J.	Rochester
§ ReMine, W. H., Jr.	Rochester
Rice, Roberta G.	Grand Is., Neb.
§ Ridley, Roger W.	Rochester
Rigler, Robert G.	Rochester
‡ Ripepi, James D.	N. Y., N. Y.
§ Risser, Alden F.	Stewartville
Rogers, Arnold G.	Rochester
Rogne, Wm. Gustav	Spring Grove
§ Rome, Howard P.	Rochester
§ Romness, J. O.	Schenectady, N. Y.
§ Rook, E. D.	Rochester
Rothwell, Walter S.	Rochester
Rovelstad, R. A.	Rochester
§ Rucker, Chas. W.	Rochester
Rushton, J. G.	Rochester
§ Rynearson, E. H.	Rochester
§ Salassa, Robert M.	Rochester
†§Sanford, Arthur H.	Rochester
Sauer, Wm. G.	Rochester
Sayre, George P.	Rochester
§ Scanlon, Paul W.	Rochester
§ Scheidel, Alois M.	Rochester
§ Scheifley, C. H.	Rochester
§ Schmidt, Herbert W.	Rochester
† Schwartz, Carl A.	Rochester

Schwarz, Bert E.......Rochester
§ Scudamore, Harold H.......Rochester
§ Seldon, Thomas H.......Rochester
Seybold, Herbert M.......Rochester
Shick, Richard M.......Rochester
Shields, Jack W.......Rochester
Shocket, Everett.......Rochester
§ Sholl, Philip R.......Rochester
Siekert, R. G.......Rochester
Silver, Arthur W.......Rochester
† Simon, Howard B.......Rochester
§ Simonton, Kinsey M.......Rochester
Simpson, H. M., Jr.......Rochester
Skrdla, Willard B.......Rochester
§ Slocumb, Charles H.......Rochester
Smith, Ballard F.......Rochester
†§Smith, Fredk. L.......Rochester
§ Smith, Harry L.......Rochester
Smith, Lowell R.......Rochester
§ Smith, Lucian A.......Rochester
‡ Smith, M. P.......Mare Island, Calif.
Smith, Newton D.......Rochester
Smith, Roger C.......Rochester
† Smith, Ross.......Rochester
§ Soule, Edward H.......Rochester
Spittel, John A.......Rochester
Sprague, Randall G.......Rochester
Starr, Grier F.......Rochester
Steinhilber, R. M.......Rochester
‡ Stevens, Grant M.......San Diego, Calif.
Stewart, Allan H.......Rochester
§ Stickney, J. M.......Rochester
§ Stilwell, George G.......Rochester
§ Stroebel, C. F., Jr.......Rochester
Struthers, Alfred M.......Rochester
‡ Stuhler, Louis G.......San Francisco, Calif.
Svien, Hendrik J.......Rochester
Symmonds, Richard E.......Rochester
Tarantino, Joseph C.......Rochester
Tarun, Donald W.......Rochester
† Taub, Robert G.......Rochester
Thompson, Gershom J.......Rochester
‡ Tihen, Edward N.......Wichita, Kansas
§ Tillisch, Jan H.......Rochester
‡ Todd, John W.......San Francisco, Calif.
‡ Tuerk, Milton.......Allentown, Pa.
Ubel, Frank S.......Rochester
§ Uihlein, Alfred.......Rochester
Ulery, Richard M.......Rochester
Underdahl, Laurentius.......Rochester
† Utne, John R.......Rochester
‡§Utz, David C.......San Francisco, Calif.
§ Van Herik, Martin.......Rochester
§ Vaughn, Louis D.......Rochester
Virnig, Hildegard J.......Rochester
§ Wagener, Henry P.......Rochester
Wagoner, James M.......Harmony
Wakefield, Elmer G.......Rochester
†§Wakim, Khalil G.......Rochester
§ Walters, Waltman.......Rochester
‡ Wang, Jun-Chuan.......
Ft. Sam Houston, Texas
‡ Ward, Bert B.......Rochester
§ Ward, Louis E.......Rochester
§ Watkins, Chas. H.......Rochester
Watson, Eleanor.......Rochester
Waud, R. E.......Rochester
§ Waugh, John M.......Rochester
Weaver, Rex W.......Rochester
§ Weber, Harry.......Rochester
§ Weed, Lyle A.......Rochester
Wehr, M. B.......Rochester
Weidman, William.......Rochester
§ Weir, James F.......Rochester
Welch, John S.......Rochester
§ Wellman, Wm. E.......Rochester
Wellner, Theo. O.......Rochester
§ Wente, Harold A.......Rochester
§ Westrup, John E.......Lanesboro
† White, Roy A.......New Orleans, La.
Wierman, Wm. H.......Rochester
† Wilder, Russell M.......Bethesda, Md.
‡ Wilkinson, G. R., Jr.......Rochester
Williams, George E.......Rochester
Williams, Henry L., Jr.......Rochester
† Williams, Rasmus V.......Rushford
Wilson, Leo H., Jr.......Rochester
§ Wilson, Robert B.......Rochester
‡ Wilson, Theo. W.......Great Lakes, Ill.
§ Wilson, V. O.......Rochester
‡ Winkelmann, Richard Birmingham, Ala.
Winterringer, James R.......Rochester
† Wise, Fred E., Jr.......Rochester
Wittchow, Allen W.......Rochester
Wollaeger, Eric.......Rochester
§ Woltman, Henry W. F.......Rochester
†§Wood, Harry G.......Rochester
§ Woolner, Lewis B.......Rochester
‡ Wuest, J. H.......Los Angeles, Calif.
§ Young, Henry H.......Rochester
‡ Zeller, Wm. W.......Philadelphia, Pa.
Zimmer, J. F.......Rochester

PARK REGION DISTRICT AND COUNTY MEDICAL SOCIETY

Douglas, Grant, Otter Tail and Wilkin Counties
Regular meeting, last Wednesday even numbered months
Annual meeting, December
Number of Members—64

President
Wasson, Loren F.......Alexandria

Secretary
Shaver, Ward.......Fergus Falls

§ Arndt, Harry W.......Detroit Lakes
Baker, Jeannette L.......Fergus Falls
§ Baker, Norman H.......Fergus Falls
Berquist, Karl E.......Battle Lake
† Blakey, Adam R.......Osakis
§ Boline, Clifford A.......Battle Lake
*§Boyd, L. M.......Alexandria
† Boysen, Peter.......Bemidji
§ Cain, James H.......Hoffman
§ Carlson, Carl E.......Alexandria
§ Clifford, G. W.......Alexandria
§ Combacker, Leon C.......Fergus Falls
§ Daehlin, Rolf.......Fergus Falls
§ DeKruif, Hendrik.......Fergus Falls
§ Doms, Vernon A.......Elbow Lake
*§Estrem, C. O.......Fergus Falls
§ Estrem, Robert D.......Fergus Falls
§ Estrem, Ralph L.......Fergus Falls
Freeman, John G.......Jamestown, N.D.
§ Geiser, Peter M.......Alexandria
George, Robert P.......Fergus Falls
Hanson, Everett C.......New York Mills
§ Hanson, LeRoy W.......Pelican Rapids
† Harris, Evelyn S.......Seattle, Wash.
§ Heiberg, Emmett A.......Fergus Falls
§ Helseth, Hovald K.......Pelican Rapids
Hom, Leong Y. W.......Battle Lake
§ Jacobson, Clifford W.......Breckenridge
§ Kevern, Jay L.......Henning
§ Korda, Henry A.......Pelican Rapids
Larson, Donald M.......Fergus Falls
Leibold, Herbert H.......Parkers Prairie
§ Lewis, Arthur J.......Henning
Love, Fredk. A.......Carlos
§ Lund, Carl J. T.......Fergus Falls
§ Mahowald, Aloys.......Fergus Falls
Mortensen, Nels G.......Minneapolis
§ Mouritsen, Glenn J.......Fergus Falls
†§Naegeli, Frank.......Fergus Falls
Nelson, Roy A.......Fergus Falls
§ Nelson, Wilburn O. B.......Fergus Falls
§ O'Brien, Louis T.......Breckenridge
Ostergaard, Erling.......Evansville
§ Palmerton, Ernest S.......Fergus Falls
Parson, Lillian B.......Elbow Lake
Parson, Lester R.......Elbow Lake
† Patterson, Wm. L.......Fergus Falls
† Paulson, Theo. S.......Fergus Falls
Perkins, Douglass E.......Alexandria
* Randall, A. M.......Ashby
Rockwood, Philo H.......Fergus Falls
§ Sanderson, David J.......Fergus Falls
† Satersmoen, Theo.......Pelican Rapids
§ Sather, Edgar R.......Alexandria
§ Schamber, Walter F.......Parkers Prairie
Schoeneberger, P. B.......Perham
§ Shaver, Ward.......Fergus Falls
§ Stremsrud, Harold L.......Alexandria
§ Struxness, David F.......Alexandria
Sutton, Harris R.......Hoffman
§ Tanquist, Edwin J.......Alexandria
Thompson, Harlow B.......St. Cloud
Warner, James J.......Perham
§ Wasson, Loren F.......Alexandria
‡§Weyhrauch, Robert A.......Alexandria
Williams, Richard A.......Rothsay
† Wray, William E.......Campbell

RAMSEY COUNTY MEDICAL SOCIETY

Regular meetings, last Monday in every month excepting June, July, August
Annual meeting, January
Election in November
Number of Members—461

President
Ritchie, Wallace P.......St. Paul

Secretary
Hilger, Lawrence D.......St. Paul

§ Adair, Albert F., Jr.......St. Paul
*†§Ahrens, A. H.......Sandstone
†§Ahrens, Albert E.......St. Paul
Ahrens, Robert M.......St. Paul
‡§Alden, J. F., Jr.......Ft. Smith, Ark.
§ Arnquist, Andrew S.......St. Paul
§ Arny, Fredk. P.......St. Paul
§ Arzt, Philip K.......St. Paul
§ Aurelius, J. Richards.......St. Paul
§ Ausman, Duane R.......St. Paul
§ Babb, Frank S.......St. Paul
§ Bacon, Donald K.......St. Paul
§ Baer, Walter.......St. Paul
§ Balcome, M. M.......St. Paul
Barnett, Joseph M.......St. Paul
Baronofsky, Ivan D.......Minneapolis
*§Barry, L. W.......St. Paul
† Barsness, Nellie O. N.......St. Paul
Bauer, Eugene L.......St. Paul
†§Beals, Hugh.......St. Paul
§ Beech, Raymond H.......St. Paul
§ Beek, Harvey O.......St. Paul
§ Beer, John J.......St. Paul
§ Bell, Chas. C.......St. Paul
Bellomo, James.......St. Paul
‡ Bellomo, John.......St. Paul
§ Benepe, James L.......St. Paul
† Bennion, P. H.......Isway, Montana
§ Bentley, Norman P.......St. Paul
Bernier, M. J.......No. St. Paul
§ Bernstein, Wm. C.......St. Paul
§ Bicek, Jos. F.......St. Paul
† Binger, Henry E.......Phoenix, Ariz.
Black, Earl J.......St. Paul
†§Bock, Rolland A.......St. Paul
† Boeckmann, Egil.......St. Paul
§ Bolender, Harold L.......St. Paul
§ Bonello, Frank J.......St. Paul
§ Borg, Jos. F.......St. Paul
† Bouma, Lewis R.......St. Paul
§ Brand, George D.......St. Paul
† Bray, Elwyn R.......St. Paul
§ Briggs, John F.......St. Paul
§ Broadie, Thos. E.......St. Paul
§ Brodie, Walter D.......St. Paul
† Brown, J. C.......Los Gatos, Calif.
§ Bulinski, Theodore J.......St. Paul
§ Burch, E. P., II.......St. Paul
† Burch, Frank E.......St. Paul

§ Burkland, E. D..........St. Paul
§ Burlingame, D. A..........St. Paul
§ Burmeister, Richard..........St. Paul
†§Burns, Robert M..........St. Paul
Burton, Carl G..........St. Paul
§ Bush, Robert P..........St. Paul
§ Busher, Herbert H..........St. Paul

§ Cain, C. L..........St. Paul
Callahan, F. F..........St. Paul
§ Carley, Walter A..........St. Paul
§ Carroll, Wm. C..........St. Paul
Cedarleaf, Cherry B..........St. Paul
§ Chadbourn, Chas. R..........St. Paul
§ Chatterton, Carl C..........St. Paul
§ Christiansen, Andrew..........St. Paul
§ Clark, Henry B., Jr..........Minneapolis
Cochrane, Bryon B..........St. Paul
Coddon, Walter D..........St. Paul
Cohen, Ellis N..........St. Paul
§ Colby, W. L..........St. Paul
§ Cole, Wallace H..........St. Paul
Coleman, John B..........St. Paul
† Collie, H. G.........St. Petersburg, Fla.
‡§Connolly, C. J..........Fort Belvoir, Va.
Connor, Chas. E..........St. Paul
§ Cook, Carroll K..........St. Paul
§ Cooper, Charles C..........St. Paul
§ Countryman, Roger S..........St. Paul
§ Craig, David M..........St. Paul
§ Critchfield, Lyman R..........St. Paul
Crowley, James H..........St. Paul
§ Crudo, Vincent D..........St. Paul
§ Crump, James W..........St. Paul
§ Culligan, John M..........St. Paul
§ Culver, L. G..........St. Paul

§ Davis, Edward..........St. Paul
Dawson, James R..........St. Paul
Decker, Charles H..........St. Paul
§ Derauf, Benj. I..........St. Paul
§ Deters, Donald C..........St. Paul
† Dickson, Thos. H..........St. Paul
* Donohue, P. F..........St. Paul
§ Drake, Carl B..........St. Paul
§ Dunn, James N..........St. Paul

§ Earl, George A..........St. Paul
§ Earl, John R..........St. Paul
§ Edlund, Gustaf..........St. Paul
Edwards, Jos. W..........St. Paul
Edwards, L. G..........St. Paul
§ Edwards, Thomas J..........St. Paul
§ Eginton, Charles T..........St. Paul
Ely, Orriman S..........So. St. Paul
Emerson, Edward C..........St. Paul
§ Emmons, R. W..........St. Paul
§ Endress, Edward K..........St. Paul
§ Enroth, Oscar E..........St. Paul
† Ernest, G. C. H.....St. Petersburg, Fla.
§ Ersfeld, Murray P..........St. Paul
† Eshelby, E. C..........St. Paul
‡ Evert, John A., Jr..........St. Paul
Farkas, John V..........St. Paul
Fee, John G..........St. Paul
Felder, Davitt A..........St. Paul
§ Felion, Arthur J..........St. Paul
†§Ferguson, James C..........St. Paul
§ Fesler, Harold H..........St. Paul
Field, Anthony H..........Farmington
Fink, Daniel L..........St. Paul
§ Fisher, Dan W..........St. Paul
§ Flanagan, Harold F..........St. Paul
§ Flannery, H. F..........St. Paul
Flom, R. P..........St. Paul
§ Flynn, L. L., Jr..........St. Paul
† Fogarty, Charles W., Sr..........St. Paul
§ Fogarty, Charles W., Jr..........St. Paul
§ Fogelberg, Emil J..........St. Paul
§ Foley, F. E. B..........St. Paul
Forsythe, James R..........St. Paul
§ Freeman, C. D., Jr..........St. Paul
‡ Freeman, Gerald I..........St. Paul
§ Freidman, Louis L..........St. Paul
§ Fritz, W. L..........St. Paul
§ Froats, Chas. W..........St. Paul
§ Fuller, Benjamin F..........St. Paul
Furnell, D. Q..........St. Paul
Galligan, John J..........St. Paul
§ Garbrecht, Arthur W..........St. Paul
§ Gardner, Walter P..........St. Paul
Garrow, Douglas M..........St. Paul
§ Gehlen, Jos. N..........St. Paul
§ Ghent, Chas. H..........St. Paul
Gibbs, Edward C..........St. Paul
* Gilkey, S. E..........St. Paul
§ Gillespie, Delmar R..........St. Paul
§ Gleason, W. A..........St. Paul
Goldsmith, Joseph W..........St. Paul
†§Goltz, Edward V..........St. Paul
‡ Goltz, Neill F..........St. Paul
§ Grant, Hendrie W..........St. Paul
§ Gratzek, Thos..........St. Paul

§ Grau, R. K..........St. Paul
† Gruenhagen, Arnold P..........St. Paul

§ Hall, Barnard..........St. Paul
§ Hammes, Ernest M., Sr..........St. Paul
§ Hammes, Ernest M., Jr..........St. Paul
† Hammond, James F..........St. Paul
§ Hanson, Harold B..........St. Paul
Harbaugh, John..........St. Paul
§ Harmon, G. E..........St. Paul
§ Hartfiel, Wm. F..........St. Paul
Hartig, Marjorie..........St. Paul
§ Hartley, Everett C..........St. Paul
§ Hauser, Victor P..........St. Paul
§ Hayes, Albert F..........St. Paul
§ Heck, Wm. W..........St. Paul
Hedenstrom, Frank G..........St. Paul
Heilig, William R..........St. Paul
Henderson, A. J. G..........No. St. Paul
§ Hengstler, Wm. H..........St. Paul
† Hensel, Charles N..........St. Paul
Herman, Samuel M..........St. Paul
§ Heron, Roy C..........St. Paul
§ Herrmann, Edgar T..........St. Paul
Hertz, Myron J..........St. Paul
†§Hilger, Andrew W..........St. Paul
§ Hilger, Jerome A..........St. Paul
§ Hilger, Lawrence D..........St. Paul
Hilker, Marcus D..........St. Paul
Hiniker, Louis P..........St. Paul
§ Hochfilzer, John J..........St. Paul
Hodgson, Jane E..........St. Paul
† Holcomb, O. W..........St. Paul
§ Hollinshead, W. H..........St. Paul
§ Holmen, Robert W..........St. Paul
§ Holt, John E..........St. Paul
Hopkins, George W..........St. Paul
Howard, Merrill A..........St. Paul
Howard, Willard S..........St. Paul
§ Howe, Newell W..........St. Paul
§ Hullsiek, Harold E..........St. Paul
§ Hullsiek, Richard..........St. Paul
§ Hunter, Murray H..........Farmington
§ Hurwitz, Milton M..........St. Paul

†§Ide, Arthur W..........St. Paul
Ikeda, Kano..........St. Paul
Ingerson, Carl A..........St. Paul
§ James, Ellery..........St. Paul
James, John W..........White Bear Lake
§ Janssen, Martin..........St. Paul
Jarvis, Marilyn A..........St. Paul
Jesion, Jos. W..........St. Paul
§ Johanson, W. G..........St. Paul
§ Johnson, Carl E..........St. Paul
§ Johnson, Herbert..........St. Paul
† Johnson, Morris B..........St. Paul
Johnson, R. J..........St. Paul
† Jones, E. Mendelssohn..........St. Paul
§ Kamman, G. R..........St. Paul
Karon, Irving M..........St. Paul
§ Kasper, Eugene M..........St. Paul
Katz, L. J..........Long Beach, Calif.
Keefe, Rolland E..........St. Paul
†§Kelly, John V..........St. Paul
§ Kelsey, Chauncey M..........St. Paul
§ Kenefick, Emmett V..........St. Paul
§ Kenyon, Thomas J..........St. Paul
§ Kesting, Herman..........St. Paul
† King, G. L..........Hudson, Wisc.
§ Klein, Henry N..........St. Paul
§ Knutson, Gerald E..........St. Paul
Knutson, Robert..........St. Paul
Koza, Donald N..........St. Paul
Krezowski, Thomas..........St. Paul
§ Kugler, Alex A..........St. Paul
§ Kuske, Albert W..........St. Paul
Kusske, Douglas R..........St. Paul
Kvitrud, Gilbert..........St. Paul
§ Lannin, Bernard G..........St. Paul
§ Lannin, Donald R..........St. Paul
Larrabee, Walter..........St. Paul
Larson, Eva-Jane..........St. Paul
Larson, James T..........So. St. Paul
Larson, Kenneth R..........St. Paul
Larson, Martin L..........St. Paul
Lax, Morris H..........St. Paul
†§Leahy, Bartholomew..........St. Paul
§ Leavenworth, R. O., Sr..........St. Paul
† Leavenworth, R. O., Jr..........St. Paul
§ Leick, Richard M..........St. Paul
Leitch, Archibald..........St. Paul
§ Lepak, John A..........St. Paul
† Lerche, Wm..........Cable, Wisc.
§ Leven, Nathaniel L..........St. Paul
§ Leverenz, Carlton W..........St. Paul
Levin, Bert G..........St. Paul
Levitt, George X..........St. Paul
§ Lick, Chas. L..........St. Paul
Lick, Wm. J., Jr..........St. Paul
§ Lien, Richard J..........St. Paul
Lilleberg, Norbert J..........St. Paul

§ Lindell, Robert..........St. Paul
Lippman, Hyman S..........St. Paul
§ Loken, Selmur M..........St. Paul
Lowe, Earl R..........So. St. Paul
Lowe, Thos. A..........So. St. Paul
Lightbourn, E. L..........St. Paul
§ Lundholm, Arthur M..........St. Paul
§ Lynch, Francis W..........St. Paul

‡ Mackoff, Sam M..........Scottfield, Ill.
§ Madden, John F..........St. Paul
Madland, Robert S..........St. Paul
Maertz, William F..........St. Paul
§ Malerich, J. A..........St. Paul
Marks, Roger W..........St. Paul
§ Martin, Dwight L..........St. Paul
§ Martineau, Jos. L..........St. Paul
Matthews, James H..........Minneapolis
§ McCabe, James S..........St. Paul
§ McCain, Donovan L..........St. Paul
McCarthy, Jos. J..........St. Paul
* McCarthy, Wm. R..........St. Paul
† McClanahan, J. H..........St. Paul
McClanahan, T. S.....White Bear Lake
§ McCloud, Charles N..........St. Paul
§ McEwan, Alexander..........St. Paul
§ McGroarty, Brian J..........St. Paul
McKenzie, Eva E..........St. Paul
§ McNeill, J. A..........St. Paul
§ Meade, John R..........St. Paul
§ Mears, Burtis J..........St. Paul
§ Medelman, John P..........St. Paul
Melancon, Joseph F..........St. Paul
§ Menold, Wm. F..........St. Paul
Merner, T. B..........Faribault
§ Merrick, Robert L..........St. Paul
†§Meyerding, Edward A..........St. Paul
Michienzi, Leonard J..........St. Paul
Midboe, Gilbert..........St. Paul
‡ Miller, William T..........St. Paul
Miller, Z. R..........St. Paul
§ Milnar, Frank J..........St. Paul
§ Mishek, Charles J..........St. Paul
§ Moga, John A..........St. Paul
Molander, Herbert A..........St. Paul
§ Monahan, Robert H..........St. Paul
§ Mooney, Robert D..........St. Paul
† Moquin, Marie A..........St. Paul
† Moren, J. Adelaide..........St. Paul
Moriarty, Berenice..........St. Paul
Moriarty, Cecile R..........St. Paul
§ Muller, A. E..........No. St. Paul
Mundahl, Harold R..........St. Paul
§ Murphy, Jack T..........St. Paul

§ Nash, L. A..........St. Paul
Neibergs, Lidija..........St. Paul
Neibergs, Pauls..........St. Paul
† Nelson, Louis A..........St. Paul
Nimlos, Kenneth O..........St. Paul
† Nimlos, Lenore O..........St. Paul
§ Noble, John F..........St. Paul
§ Noble, John L..........St. Paul
Nye, Katherine A..........St. Paul
Nye, Lillian L..........St. Paul

O'Brien, J. C..........St. Paul
O'Connor, Loren J..........St. Paul
O'Kane, Thomas W..........St. Paul
‡ O'Malley, Valentine..........St. Paul
§ O'Reilley, Bernard E..........St. Paul
§ Ockuly, Orville..........St. Paul
§ Ogden, Warner..........St. Paul
§ Ohage, Justus..........St. Paul
§ Olsen, Ralph L..........St. Paul
Olson, Charles A..........St. Paul
§ Ostergren, Edw. W..........St. Paul
§ Ouellette, Alfred J..........St. Paul
§ Owens, F. M., Jr..........St. Paul
Paulson, Wallace..........St. Paul
Pearson, Fritz R..........St. Paul
§ Pearson, Malcolm M..........St. Paul
Pedersen, Arthur H..........St. Paul
Peterson, David B..........St. Paul
§ Peterson, Donald H..........St. Paul
§ Peterson, Harold O..........St. Paul
Peterson, J. L. E..........St. Paul
Peterson, Roy L..........White Bear Lake
†§Plondke, Fred J..........St. Paul
§ Plotke, Harry L..........St. Paul
Polski, Paul G..........So. St. Paul
Quattlebaum, Frank..........St. Paul
§ Ralph, James R..........St. Paul
† Ramsey, Walter R..........St. Paul
Rasmussen, Ramby C..........St. Paul
§ Ravits, Harold G..........St. Paul
§ Rea, Charles E..........St. Paul
Reid, James W..........St. Paul
§ Richards, E. T. F..........St. Paul
Richardson, E. J., Jr..........St. Paul
§ Richardson, Robert J..........St. Paul
Rick, Paul F. W..........St. Paul
Rinkey, Eugene..........St. Paul

§ Ritchie, Wallace P..........St. Paul
§ Ritt, Albert E..........St. Paul
§ Rogers, Sydney F..........St. Paul
† Rogin, Norton..........St. Louis Park
Rolig, David H..........St. Paul
§ Rollie, O. O..........St. Paul
§ Rosenthal, Robert..........St. Paul
§ Roth, George C..........St. Paul
†§Rothschild, Harold J..........St. Paul
§ Roy, Phil. C..........St. Paul
† Ruhberg, Geo. N..........Tarzona, Calif.
§ Rusterholz, Alan P..........St. Paul
§ Ryan, John J..........St. Paul
§ Ryan, Jos. M..........St. Paul
§ Sarnecki, M. M..........St. Paul
† Satterlund, Victor L..........St. Paul
†§Savage, Francis J..........St. Paul
§ Schmidtke, R. L..........St. Paul
† Schoch, Robert B. J..........St. Paul
§ Schons, Edward..........St. Paul
§ Schroeckenstein, H. F..........St. Paul
†§Schuldt, Fredk. C..........St. Paul
†§Schulze, Albert G..........St. Paul
Schumacher, John W..........Robbinsdale
§ Schwyzer, Hanns C..........St. Paul
Schwyzer, Marguerite..........St. Paul
§ Scott, Eugene E..........St. Paul
Sekhon, Mohan S..........St. Paul
Sells, Richard J..........No. St. Paul
† Senkler, George E..........St. Paul
§ Setzer, Hobert J..........St. Paul
Shannon, Wm. R..........St. Paul
† Shellman, J. L. Pacific Palisado, Calif.
Short, Jacob..........St. Paul
Siegel, Clarence..........St. Paul
‡ Siegel, S. C..........St. Paul
§ Simons, Leander T..........St. Paul
Singer, Benj. J..........St. Paul
§ Skinner, Abbott..........St. Paul
† Skinner, Harvey O..........St. Paul
§ Smisek, Elmer A..........St. Paul
Smith, Clyde..........Bismarck, N. D.
§ Smith, Vernon D. E..........St. Paul
§ Snyder, Geo. Wm..........St. Paul
§ Sohlberg, Olof I..........St. Paul
Sommers, Ben..........St. Paul
§ Sorem, Milton B..........St. Paul
Soucheray, Philip H..........St. Paul
§ Souster, Benj. B..........St. Paul
§ Sowada, Ernest J..........St. Paul
‡ Sprafka, Gregory..........N. Y., N. Y.
†§Sprafka, Joseph L..........St. Paul
§ Sprafka, Joseph M..........St. Paul
§ Steinberg, Chas. L..........St. Paul
§ Sterner, Donald C..........St. Paul
†§Sterner, Ernest G..........St. Paul
§ Sterner, E. R..........St. Paul
§ Sterner, John J..........St. Paul
Stewart, Alexander..........St. Paul
§ Stolpestad, Armer H..........St. Paul
§ Stolpestad, Herbert L..........St. Paul
§ Strate, Gordon E..........St. Paul
Straus, M. L..........St. Paul
§ Strem, Edward L..........St. Paul
§ Sturley, Rodney F..........St. Paul
Swanson, John A..........St. Paul
‡ Swanson, Lawrence J..........St. Paul
§ Swendson, James J..........St. Paul
† Swenson, Donald B..........Minneapolis
§ Teisberg, John E..........St. Paul
Thompson, Floyd A..........St. Paul
Thoreson, M. C. Bernice....So. St. Paul
§ Tifft, Cyril R..........St. Paul
§ Tongen, Lyle A..........St. Paul
§ Tracht, Robert R..........St. Paul
Travis, James S..........St. Paul
§ Tregilgas, Harold R..........So. St. Paul
Van Bergen, F. H..........Minneapolis
Varco, Richard L..........St. Paul
Veirs, Dean M..........St. Paul
Veirs, Ruby J. S..........St. Paul
§ Venables, Alexander E..........St. Paul
‡§von Amerongen, W. W..........St. Paul
§ Waas, Chas. W..........St. Paul
§ Walker, Arthur E..........St. Paul
§ Wallinga, Jack..........St. Paul
§ Walsh, Edward F..........St. Paul
§ Walter, Clarence W..........St. Paul
†§Ward, Peter D..........St. Paul
Warren, Cecil A..........St. Paul
Watson, P. Theodore..........St. Paul
§ Watson, W. H. A..........St. Paul
§ Watson, Wm. J..........Newport
Watz, Clarence E..........St. Paul
§ Webber, Fred L..........St. Paul
§ Weis, Benjamin A..........St. Paul
§ Weisberg, Maurice..........St. Paul
§ Wenzel, Gilbert P..........St. Paul
Wesolowski, Stanley P..........St. Paul
‡ Westover, D. E..........St. Paul
† Wheeler, Merrit W..........Stillwater
† Wilkinson, Stella L..........St. Paul
† Williams, Arthur B..........St. Paul
§ Williams, Clayton K..........St. Paul
§ Williams, John A..........St. Paul
Wilson, J. Allen..........St. Paul
§ Wilson, James V..........St. Paul
† Winnick, Jos. B..........St. Paul
§ Wolff, Herman J..........St. Paul
Wolkoff, H. J..........St. Paul
Word, Harlan L..........St. Paul
Wurdemann, Alma L. White Bear Lake
‡ Yamamoto, Joe..........Fort Lewis, Wash.
Youngren, E. R..........St. Paul
Zachman, Leo L..........St. Paul
§ Zimmermann, H. B..........St. Paul

RED RIVER VALLEY MEDICAL SOCIETY

Kittson, Mahnomen, Marshall, Norman, Pennington, Polk, Red Lake and Roseau Counties
Regular meetings, three—plus extras
Annual meeting, first week in December
Election, January
Number of Members—67

President
McKaig, A. M..........Red Lake Falls

Secretary
Sather, Russell O..........Crookston

† Adkins, C. M..........Thief River Falls
§ Anderson, W. E..........Clearbrook
‡ Behling, Fredk. L..........Rochester
§ Behr, Orlo K..........Crookston
§ Berge, David O..........Roseau
Berlin, A. S..........Hallock
† Biedermann, Jacob..........Thief River Falls
§ Boyer, G. S..........Crookston
† Bratrud, Edward..........Thief River Falls
Bratrud, Theo..........Minneapolis
§ Bretzke, Carl O..........Greenbush
Brink, Adlai A..........Baudette
Burnett, C. F., Jr..........Crookston
§ Cameron, J. H..........Crookston
§ Covey, Kenneth W..........Mahnomen
§ Dale, L. N..........Red Lake Falls
§†Delmore, John L..........Roseau
§ Delmore, J. L., Jr..........Roseau
Delmore, Robert J..........Roseau
§ Erickson, Eskil..........Halstad
§ Feigal, W. M..........Thief River Falls
Flancher, Leon H..........Crookston
§ Greene, D. E..........Thief River Falls
§†Henney, Wm. H..........McIntosh
Hirsch, Stanton A..........Crookston
† Hollands, Wm. H..........Fisher
§ Holmstrom, C. H..........Warren
Janecky, A. G..........Baudette
‡ Johnson, H. C..........San Diego, Calif.
§ Kinkade, Byron R..........Ada
† Kirk, G. P..........East Grand Forks
§ Klefstad, L. H..........Greenbush
* Knutson, G. A..........Hallock
§ Kostick, Wm. R..........Fertile
§ Loken, Theodore..........Ada
Lund, Anthony J..........Cleveland, Ohio
Martin, George..........Thief River Falls
§ McDonald, W. J..........Crookston
McHardy, Bryson R..........Karlstad
§ McKaig, A. M..........Red Lake Falls
*†Mellby, O. F..........Thief River Falls
§ Mercil, William..........Crookston
† Nelson, Henry E..........Crookston
Nelson, Kenneth..........Warroad
§ Norman, John F..........Crookston
§ Oppegaard, Chester..........Crookston
Parker, Philip..........Thief River Falls
§ Pumala, Erven E..........Warren
† Quigley, Walter..........Brooklyn, N. Y.
§ Reff, Alan R..........Crookston
§ Roholt, Christian..........McIntosh
‡ Roholt, H. B..........Fosston
‡ Rydland, Arne D..........Minneapolis
Sather, Edgar L..........Fosston
§ Sather, George A..........Fosston
§ Sather, Richard..........Fosston
§ Sather, Russell O..........Crookston
§ Skogerboe, Rudolph..........Karlstad
Stadem, Clifford..........Twin Valley
§ Starekow, Milton..........Thief River Falls
§ Steward, Donald..........Crookston
‡ Stone, Norman F..........Chicago, Ill.
§ Thysell, Harold..........Crookston
§ Uhley, Charles G..........Crookston
Van Rooy, George T...Thief River Falls
Wendt, H. P..........Thief River Falls
Wheeler, G. S..........Roseau
§ Wikoff, Howard M..........Crookston
† Wiltrout, Irving G..........Oslo

RENVILLE-REDWOOD COUNTY MEDICAL SOCIETY

Regular meeting, second Tuesday of each month
Annual meeting, November
Number of Members—25

President
Hinderaker, Harris..........Bird Island

Secretary
Diessner, A. W..........Redwood Falls

§ Alcorn, William J..........Wabasso
§ Anderson, Chester A..........Hector
‡ Anderson, Donald C...New York, N. Y.
Bessesen, Daniel H..........Buffalo Lake
§ Billings, Ralph E..........Franklin
† Brand, William A..........Redwood Falls
§ Ceplecha, S. F..........Redwood Falls
§ Cosgriff, James A..........Olivia
Cosgriff, James A., Jr..........Olivia
§ Diessner, A. W..........Redwood Falls
§ Dordal, John..........Sacred Heart
§ Fawcett, Arthur M..........Renville
† Flinn, Thomas E..........Remer
§ Haas, J. F..........Fairfax
§ Hinderaker, Harris..........Bird Island
Johnson, Orville H..........Omaha, Nebr.
§ Johnson, William E..........Morgan
§ Knoche, Harvey A., Jr..........Morgan
§ Lenz, Joseph R..........Morton
† Mesker, George H..........Olivia
§ Nelson, Glenn E..........Fairfax
§ Pierce, Robert B..........Renville
Potthoff, Carl J..........Rochester
† Preisinger, J. W..........Dallas, Texas
§ Strauchler, Jona..........Belview

RICE COUNTY MEDICAL SOCIETY

Regular meeting, third Thursday of every month
Annual meeting, October
Number of Members—33

President
Lende, Norman....Faribault

Secretary
Orr, Burton A....Faribault

Beaton, J. Gordon....Northfield
§ Bruhl, Heinz H....Faribault
Buesgens, Ralph H....Waterville
†§Dungay, Neil S....Northfield
§ Engberg, Edward J....Faribault
Francis, David W....Morristown
§†Hanson, Adolph M....Faribault
† Huxley, Fredk. R....Faribault
§ Kennedy, George L....Faribault
§ Kolars, J. J....Faribault
§ Kucera, L. B....Lonsdale
§ Lende, Norman....Faribault
† Lexa, F. J....Lonsdale
*§Maertz, R. W....Faribault
§ Mears, Robert F....Northfield
Meyer, Frederick C....Kenyon
Meyer, Paul F....Faribault
Meyer, Robert P....Faribault
§ Moses, Royal R....Kenyon
§ Nelson, Ernest J....Lonsdale
§ Nielson, Alvin M....Northfield
§ Nuetzman, A. W....Faribault
§ Orr, Burton A....Faribault
Petersen, D. H....Northfield
§ Robilliard, C. M....Faribault
† Rohrer, C. A....Des Moines, Ia.
Rumpf, Carl W....Faribault
§ Rysgaard, George M....Northfield
§ Stevenson, Frank W....Faribault
§ Street, Bernard....Northfield
Studer, Donald J....Faribault
§ Traeger, Carl A....Faribault
§ Weaver, Paul H....Faribault
§ Wilson, Warren E....Northfield

ST. LOUIS COUNTY MEDICAL SOCIETY

Carlton, Cook, Itasca, Lake and St. Louis Counties
Regular meetings, second Thursday in each month
Annual meeting, second Thursday in January
Election, second Thursday in December
Number of Members—258

President
Boyer, Samuel H., Jr....Duluth

Secretary
Van Ryzin, D. J....Duluth

§ Abraham, Arden L....Duluth
§ Adams, Bertram S....Hibbing
Addy, Edward R....Gilbert
Ahola, Kenneth E....Hibbing
§ Ahrens, Curtis F....Duluth
Arko, J. L....Hibbing
§ Athens, Alvin G....Duluth
§ Aufderheide, Arthur....Duluth
§ Backus, Reno W....Nopeming
§ Bagley, Charles M....Duluth
Bagley, Elizabeth C....Duluth
§ Bagley, Wm. R....Duluth
Baich, Velemir M....Bovey
§ Bakkila, Henry E....Duluth
§ Bardon, Richard....Duluth
§ Barker, John D....Duluth
Barney, Leon A....Duluth
§ Barrett, Earl E....Duluth
§ Becker, Fredk. T....Duluth
§ Benell, Otto E....Virginia
Bepko, Marie K....Cloquet
Berdez, George L....Duluth
§ Bergan, R. O....Duluth
§ Bianco, Anthony J., Sr....Duluth
‡§Bianco, Anthony J., Jr....Duluth
† Binet, Henry E....Grand Rapids
§ Blackmore, Sidney C....Biwabik
§ Bloom, Joseph....Duluth
‡ Bolz, J. A....Camp Carson, Colo.
Boman, Paul G....Duluth
Bouchelle, McLemore....Virginia
Bowen, Robert L....Hibbing
† Boyer, Samuel H., Sr....Duluth
§ Boyer, Samuel H., Jr....Duluth
§ Braun, Ohrmundt C....Grand Rapids
§ Bray, Philip N....Duluth
§ Brooker, Warren J....Duluth
Buchanan, Gerald S....Deer River
§ Buckley, Robert P....Duluth
§ Butler, John K....Cloquet
Cantwell, Wm. F....International Falls
Chapman, Theodore L....Duluth
§ Chermak, F. G....International Falls
§ Christensen, Clarence H....Duluth
§ Clark, Clarence L....Duluth
Clark, Elizabeth A....Duluth
§ Clark, Ivan T....Duluth
§ Coll, Joseph J....Duluth
† Collins, Arthur N....Duluth
‡§Conley, F. W....Ft. Hood, Texas
† Cope, Hershel E....Virginia
§ Coventry, Wm. D....Duluth
Cowan, George M....Duluth
Detjen, Edward D....Bigfork
§ Dickson, F. H., Jr....Proctor
Doyle, George C....Duluth
§ Dwyer, John J....Duluth
§ Eckman, Philip F....Duluth
§ Eckman, Ralph J....Duluth
† Ekblad, John Wm....Duluth
§ Eklund, C. D....Duluth
§ Elias, Frank J....Duluth
§ Emmanuel, Karl Wm....Duluth
Eppard, Raymond M....Cloquet
Erickson, George P....Hibbing
Erskine, Gordon N....Grand Rapids
* Estrem, Theo. A....Hibbing
Evensta, John B....Grand Rapids
§ Fawcett, Keith R....Duluth
§ Fellows, Manley F....Duluth
‡ Ferrell, Clarence R....Grand Rapids
§ Feuling, John C....Duluth
§ Fischer, Mario McC....Duluth
§ Fisketti, Henry....Duluth
§ Flynn, Bernard F....Hibbing
§ Fredericks, Merriam G....Duluth
§ Fuller, Josiah....Duluth
§ Gillespie, M. G....Duluth
§ Goldish, Daniel R....Duluth
§ Goldish, Robert J....Duluth
Goodman, Charles....Virginia
§ Gowman, Lawrence R....Duluth
†§Graham, Archibald W....Chisholm
§ Grahek, Jack P....Ely
§ Grinley, Andrew....Grand Rapids
Grohs, William H....Duluth
§ Haavik, John E....Duluth
Halbert, John J....Duluth
† Halliday, P. V....Minneapolis
§ Halper, Bernard....Hibbing
† Haney, Claude L....Duluth
§ Hansen, Robert E....Hibbing
Hanson, Ernest O....Carlton
§ Harris, Carl N....Hibbing
§ Hatch, Walter E....Duluth
† Hayes, Michael F....Moose Lake
§ Hedberg, G. A....Nopeming
Hedenstrom, Paul H....Duluth
Heiam, Wm. C....Cook
§ Hilding, A. C....Duluth
Hill, Fredk. E....Duluth
Hirschboeck, F. J....Duluth
§ Hoff, Herbert O....Duluth
Hoover, Norman W....Virginia
§ Houkom, S. S....Duluth
Hutchinson, Henry....Moose Lake
‡ Indihar, John E....Virginia
§ Irwin, Homer R....Hibbing
§ Jacobson, Clarence....Chisholm
§ Jacobson, F. C....Duluth
§ Jensen, Thorvold J....Duluth
§ Jeronimus, Henry J....Duluth
Jessico, C. M....Duluth
Joffe, H. H....Virginia
‡ Johnson, Edward A....Virginia
§ Johnson, Karl E....Duluth
§ Johnsrud, L. W....Hibbing
Johnston, Henry W....Virginia
†§Johnston, Rufus O....Hibbing
§ Jolin, Francis M....Bovey
Juntunen, Roy R....Duluth
‡ Karges, L. E....Grand Rapids
§ Kelley, Walter....Duluth
§ Kelly, Albert C....Duluth
Kelly, Robert T....Nashwauk
§ Klein, Harry....Duluth
§ Klein, Wm. A....Duluth
§ Knapp, Frank N....Duluth
§ Knoll, W. V....Duluth
§ Kohlbry, Carl O....Duluth
Koskela, Lauri....Shaker Heights, Ohio
§ Kotchevar, Frank R....Eveleth
Kozberg, Oscar....Moose Lake
§ Krueger, Victor R....Nopeming
§ LaBree, Robert H....Duluth
†§Laird, Arthur T....Duluth
§ Latterell, K. E....Duluth
Law, Harrison E....Virginia
‡ Leek, Joseph H....Duluth
Lepak, Francis J....Duluth
Leppo, N. Erkki A....Duluth
§ Litman, S. N....Duluth
§ MacFarlane, P. H....Chisholm
§ MacRae, G. C....Duluth
§ Magney, F. H....Duluth
‡ Magraw, R. M. San Francisco, Calif.
§ Malmstrom, J. A....Virginia
† Marcley, Walter J....Minneapolis
§ Martin, Wm. B....Duluth
§ Martin, W. C....Duluth
§ Mayne, Roy M....Nopeming
† McCarty, Paul D....Ely
† McCoy, Mary K....Duluth
† McDonald, Archibald....Duluth
§ McDonald, Owen G....Duluth
§ McHaffie, Orval L....Duluth
§ McKenna, M. J....Grand Rapids
*§McLeod, James L....Grand Rapids
§ McNutt, John R....Duluth
Mead, Chas. H....Duluth
§ Merriman, L. L....Duluth
Moe, Thos....Moose Lake
§ Moehring, H. G....Duluth
Mollers, Theodore P....Soudan
Monroe, Paul B....Cloquet
§ Monserud, N. O....Cloquet
§ Morsman, L. W....Hibbing
§ Moyer, John B....Duluth
Munson, Martin S....Barnum
Murray, Robert A....Hibbing
Nakamura, James....Deer River
§ Neff, Walter S....Virginia
§ Nelson, Robert L....Duluth
§ Nicholson, M. A....Duluth
§ Nisius, George F....Duluth
§ Norberg, Carl E....Cloquet
§ Nutting, Roland E....Duluth
§ Olson, Archie....Duluth
§ O'Neill, John C....Duluth
Paciotti, V. J....Minneapolis
§ Papermaster, Ralph....Two Harbors
† Parker, Owen W....Minneapolis
§ Parker, Wilbert H....Chisholm
§ Parson, E. I....Duluth
§ Pasek, Antone W....Cloquet
§ Patch, O. B....Duluth
† Pearsall, R. P....Virginia
§ Pedersen, R. C....Duluth
Pennie, D. F....Duluth
Peterson, Edward N....Virginia
§ Peterson, John H....Duluth
§ Pierce, Jack R....Virginia
Power, John E....Duluth
Power, John E., Jr....Duluth
Puumala, R. H....Cloquet
Raadquist, C. S....Hibbing
Raattama, John....Keewatin
Rajala, Arnold....Grand Rapids
Reed, Paul....Virginia
§ Rokala, Henry E....Virginia

†§Rood, Dana C. Santa Barbara, Calif.
Rowe, Olin W.....Duluth
§ Rowles, E. K.....Coleraine
§ Rudie, Peter S.....Duluth
§ Runquist, John M.....Duluth
Ryan, Wm. J.....Duluth
§ Sach-Rowitz, Alvan.....Moose Lake
§ Salter, R. A.....Virginia
Sandell, S. T.....Nopeming
‡ Sanford, John B.....Duluth
§ Sarff, O. E.....Duluth
Sax, Milton H.....Duluth
Sax, Simon G.....Duluth
§ Schirber, M. J.....Grand Rapids
§ Schmid, John F.....Duluth
§ Schneider, L. E.....Duluth
† Schroder, C. H.....Pottstown, Penn.
§ Schweiger, T. R.....Hibbing
§ Seashore, R. T.....Duluth
§ Sher, David A.....Virginia
Shirai, Shohei.....Coleraine
§ Siegel, John S.....Virginia
§ Sinamark, Andrew.....Hibbing
Sisler, C. E.....Grand Rapids
§ Smith, Cyril M.....Duluth
Smith, W. R.....Grand Marais
Snyker, Omer E.....Ely
§ Spang, A. J.....Duluth
§ Spang, J. S.....Duluth
§ Spang, Wm. M.....Duluth
Storsteen, Kenneth A.....Duluth
‡§Strandjord, N. M.....New York, N. Y.
†§Strathern, M. L.....Gilbert
§ Strauss, Eugene C.....Duluth
§ Strewler, G. J.....Duluth
§ Strobel, Wm. G.....Duluth
§ Sutherland, H. N.....Ely
§ Swedberg, Wm. A.....Duluth
§ Swenson, Arnold O.....Duluth
‡§Teich, K. W.....Fort Monmouth, N. J.
§ Tetlie, James P.....Duluth
§ Terrell, B. J.....Nopeming
Tingdale, Carlyle.....Hibbing
Tomhave, Wesley G.....Hibbing
§ Tosseland, N. E.....Duluth
Tuohy, Edward L.....Duluth
§ Urberg, Samuel E.....Duluth
Van Ryzin, D. J.....Duluth
§ Walder, H. J.....Duluth
† Walker, A. E.....St. Paul
§ Wallace, M. O.....Duluth
Walter, F. H.....International Falls
§ Wells, Arthur H.....Duluth
§ Wheeler, Danl. W.....Duluth
§ Williams, Bruce F. P.....Duluth
Winter, John A.....Duluth
Wolff, John M.....Duluth
†§Ylitalo, W. H.....Rochester
§ Young, Thos. O.....Duluth
§ Zupanc, Edward.....Duluth

SCOTT-CARVER COUNTY MEDICAL SOCIETY

Regular meetings, second Wednesday of each month
Annual meeting, June
Number of Members—26

President
Olson, C. J.....Belle Plaine

Secretary
Rynda, E. R.....New Prague

† Buck, Fredk. H.....Shakopee
§ Cervenka, Chas. F.....New Prague
‡§Doherty, Elmer M.....New Prague
§ Hebeisen, Milton B.....Chaska
Heinz, Ivy B.....Shakopee
Heinz, Lawrence H.....Shakopee
§ Juergens, Herman M.....Belle Plaine
*§ Kortsch, F. P.....Prior Lake
§ Kucera, Stanley T.....Northfield
Larson, Leighton.....Watertown
§ Martin, T. P.....Arlington
Nagel, Harold D.....Waconia
Ninneman, Newton N.....Waconia
† Novak, Edward E.....New Prague
Olson, Chester J.....Belle Plaine
§ Pearson, Bror F.....Shakopee
† Pearson, Roy T.....Shakopee
Pogue, Richard E.....Watertown
§ Ponterio, James E.....Shakopee
Rieschl, Elizabeth K.....Jordan
§ Rynda, Edwin R.....New Prague
† Sanford, James A.....Savage
Simmonds, H. N.....Prior Lake
Schimelpfenig, Geo. T.....Chaska
§ Simons, Bernard H.....Chaska
Stahler, Paul A.....Jordan
†§Westerman, Alvin.....Montgomery

SOUTHWESTERN MINNESOTA MEDICAL SOCIETY

Cottonwood, Jackson, Murray, Nobles, Pipestone and Rock Counties
Regular meetings, on call
Annual meeting, October. Election, November
Number of Members—59

President
Pierson, Roy F.....Slayton

Secretary
Heiberg, Olaf M.....Worthington

§ Arnold, Elmer W.....Adrian
§ Bader, J. L.....Slayton
§ Basinger, H. R.....Mountain Lake
Basinger, Homer P.....Windom
§ Beckering, Gerrit.....Edgerton
§ Benjamin, W. G.....Pipestone
§ Blattspieler, L. H.....Worthington
Bofenkamp, Ferdinand.....Luverne
§ Boone, Ervin S.....Luverne
§ Bouma, John H.....Edgerton
† Brown, Alexander H.....Pipestone
Buresh, Kenneth L.....Westbrook
Carlson, J. V.....Westbrook
§ Christiansen, H. A.....Jackson
† De Boer, Hermanus.....Edgerton
Dokken, James H.....Windom
Doman, Victor W.....Lakefield
§ Hallin, Roger P.....Worthington
§ Halloran, W. H.....Jackson
§ Halpern, David J.....Brewster
§ Harrison, P. W.....Worthington
§ Heiberg, Olaf M.....Worthington
§ Hoyer, Ludolph J.....Windom
§ Karleen, B. N.....Jackson
§ Keyes, Robert W.....Pipestone
§ Kilbride, Edwin A.....Worthington
§ Koenecke, F. H., Jr.....Lakefield
§ Kotval, Russell J.....Pipestone
§ Laikola, Leslie A.....Adrian
§ Lohmann, John G.....Pipestone
§ Maitland, Edwin T.....Jackson
† Manson, Frank M.....Worthington
§ Martin, Albert C.....Luverne
§ Minge, Raymond K.....Worthington
§ Nealy, Donald E.....Adrian
§ Nelson, C. A.....Worthington
§ Nicholson, R. W.....Heron Lake
§ Nywall, Dean D.....Slayton
§ Pankratz, P. J.....Mountain Lake
§ Patterson, H. D.....Slayton
‡ Philip, David R.....Minneapolis
§ Pierson, Roy F.....Slayton
§ Piper, William A.....Mountain Lake
† Ritzinger, Fredk. R.....Lakefield
§ Robinett, R. W.....Worthington
§ Rose, John T.....Lakefield
§ Sawtell, R. R.....Worthington
§ Schade, F. L.....Worthington
§ Schultz, Elmer S.....Mountain Lake
§ Sherman, Charles L.....Luverne
§ Slater, S. A.....Worthington
§ Sogge, Ludwig L.....Windom
§ Stam, John.....Worthington
Stanley Court R.....Worthington
Stevenson, Basil M.....Fulda
§ Stratte, H. C.....Windom
§ Wells, Walter B.....Jackson
§ Williams, Chas. A.....Pipestone
† Williams, Leon A.....Minneapolis

STEARNS-BENTON COUNTY MEDICAL SOCIETY

Regular meeting, third Thursday of each month
Annual meeting, December
Number of Members—65

President
Kelly, John F.....Cold Spring

Secretary
Anderson, E. M.....St. Cloud

§ Anderson, E. M.....St. Cloud
Andrews, Bernice F.....Holdingford
Autrey, William A.....St. Cloud
§ Baumgartner, F. H.....Albany
§ Beuning, John B.....St. Cloud
† Brigham, C. F., Sr.....St. Cloud
§ Brigham, C. F., Jr.....St. Cloud
† Broker, Henry M.....Minneapolis
§ Buscher, Julius C.....St. Cloud
Cesnik, R. J.....St. Cloud
§ Clark, Harry B.....St. Cloud
Cleaves, W. D.....Sauk Centre
Coseriu, Vasile.....St. Cloud
§ Donaldson, C. S.....St. Cloud
DuBois, J. F., Sr.....Sauk Centre
DuBois, J. F., Jr.....Sauk Centre
Evans, Leslie.....Sauk Rapids
‡ Fidelman, N. E.....Fort Sheridan, Ill.
§ Fleming, Thomas N.....St. Cloud
§ Gaida, Joseph B.....St. Cloud
§ Goehrs, Gilman H.....St. Cloud
† Goehrs, Henry W.....St. Cloud
Grant, John C.....Sauk Centre
§ Halenbeck, P. L.....St. Cloud
Henry, Clarence J.....Milaca
‡ Henry, J. E.....Milaca
Hoehn, David.....Holdingford
§ Jones, Richard N.....St. Cloud
Keith, Paul J.....Milaca
§ Kelly, John F.....Cold Spring
Koenig, Robert P.....Paynesville
† Kohler, Delphin W.....Tacoma, Wash.
§ Kuhlmann, L. B.....Melrose
† Lewis, C. B.....St. Cloud
§ Libert, John N.....St. Cloud
‡ Lindeman, R. J.....Montgomery, Ala.
‡§Luckemeyer, C. J.....Camp Polk, La.
†§McDowell, John P.....St. Cloud
Meyer, Anthony A.....Melrose
§ Milhaupt, E. N.....St. Cloud
§ Musachio, N. F.....Foley
Myre, Clifford R.....Paynesville
§ Neils, Vernon E.....Sauk Rapids
§ Nessa, Curtis B.....St. Cloud

Nietfeld, Aloys....Sauk Centre
§ O'Keefe, James P....St. Cloud
§ Petersen, Robert T....St. Cloud
§ Phares, Otto C....St. Cloud
§ Raetz, Sylvester J....Maple Lake
§ Reif, Henry J....St. Cloud
§ Richards, Wm. B....St. Cloud
§ Sandven, Nels O....Paynesville
Schatz, F. J....St. Cloud
† Schmitz, Everett J....Seattle, Wash.
† Sherwood, G. E....Kimball
‡§Simon, T. R....St. Cloud
Sisk, Harvey E....St. Cloud
Thuringer, Carl B....St. Cloud
Veranth, L. A....St. Cloud
§ Walfred, Karl A....St. Cloud
‡ Wedes, Deno J....Chicago, Ill.
§ Wenner, W. T....St. Cloud
† Wetzel, E. V....St. Paul
§ Wittrock, Louis H....Watkins
Zachman, Albert H....Melrose

STEELE COUNTY MEDICAL SOCIETY

Regular meetings, called by the officers
Annual meeting, February
Number of Members—21

President
McEnaney, C. T....Owatonna

Secretary
Dewey, Donald H....Owatonna

§ Anderson, F. C....Owatonna
§ Arnesen, John F....Owatonna
§ Dewey, Donald H....Owatonna
† Ertel, Edward Q....Ellendale
§ Fischer, J. R....Blooming Prairie
§ Halvorsen, D. K....Owatonna
Hartung, Elmer H....Claremont
Kulstad, O. S....Dodge Center
† Kurtin, H. J....Milwaukee, Wisc.
† Kurtin, J. J....Cudahy, Wisc.
§ Lundquist, C. W....Owatonna
§ McEnaney, C. T....Owatonna
§ McIntyre, John A....Owatonna
†§Melby, Benedik....Blooming Prairie
† Morehead, Dewey E....Owatonna
§ Olson, Albert J....Owatonna
§ Roberts, O. W....Owatonna
§ Schaefer, J. F....Owatonna
† Senn, Edward W....Owatonna
§ Stransky, Theo. W....Owatonna
§ Wilkowske, R. J....Owatonna

UPPER MISSISSIPPI MEDICAL SOCIETY

Aitkin, Beltrami, Cass, Clearwater, Crow Wing, Hubbard, Koochiching, Lake of the Woods, Morrison, Todd, and Wadena Counties
Regular meetings, quarterly
Annual meeting, January
Number of Members—97

President
Vandersluis, C. W....Bemidji

Secretary
Badeaux, G. I....Brainerd

§ Badeaux, George I....Brainerd
§ Beise, Rudolph A....Brainerd
Bender, James H....Brainerd
Benson, Alfred H....Little Falls
Bolstad, Owen C....Little Falls
Borgerson, A. H....Long Prairie
Brabec, Paul F....Bagley
‡ Brown, Hector M....Walker
Cardle, George E....Brainerd
Closuit, F. C....Aitkin
Cook, Jay M....Staples
§ Coombs, Carl H....Cass Lake
*†Corrigan, J. E....Spooner
Craig, Clair C....International Falls
Crow, Earl R....Walker
Cushing, Robert L....Brainerd
Davis, Lloyd T....Wadena
§ Davis, Luther E....Wadena
Davis, Thos. L., Jr....Wadena
DeWeese, W. J....Bemidji
Eiler, John....Park Rapids
§ Erickson, Alvin O....Long Prairie
Fitzsimons, Wm. E....Brainerd
§ Fortier, G. M. A....Little Falls
Franklin, Gordon W....Northome
Gacusana, J. M....Mahnomen
Garlock, A. V....Bemidji
Garlock, D. W....Bemidji
‡ Garlock, G. L....Bemidji
Ghostley, Mary C....Bemidji
† Gilmore, Rowland....Crookston
Griffin, John, Jr....Bemidji
Groschupf, Theo. P....Bemidji
Grose, Fredk. N....Clarrissa
‡ Halme, W. B....San Francisco, Calif.
§ Hanover, Ralph D....International Falls
Hartjen, J. K....Bemidji
Healy, Raymond T....Pierz
‡ Heine, George W....Little Falls
Hendricks, Esten J....Saint Paul
Higgs, Walter W....Park Rapids
Hildebrand, J. E....Bemidji
‡ Hoganson, Donald E....Bemidji
† House, Z. E....Burbank, Calif.
Houston, Donald M....Park Rapids
Hubbard, Otto E....Brainerd
§ Hughes, Bernard J....Brainerd
Janda, George W....
Port Clayton Hospital, Canal Zone
§ Johnson, Douglas L....Little Falls
† Johnson, Einer W....Bemidji
Johnson, Rudolph E....Mankato
§ Kanne, Earl R....Brainerd
Kelly, James H....Wadena
Kilmer, Warren L....International Falls
Kinports, E. B....San Diego, Calif.
§ Knight, Edwin G....Swanville
Larson, LeRoy J....Bagley
Lee, Hubert W....Brainerd
Leggett, Elizabeth....Bemidji
§ Lenarz, Albert J....Browerville
Longfellow, Helen W....Brainerd
Lund, Werner J....Staples
Lundsten, Leslie C....Bemidji
§ MacDonald, Roger A....Little Fork
§ Marshall, C. M....Crosby
McCann, Dan'l. F....Bemidji
‡ McGreary, M. D....Oceanside, Calif.
§ McLane, Wm. O....Brainerd
§ Mosby, Maurice E....Long Prairie
§ Mulligan, Arthur M....Brainerd
Nelson, Nesmith P....Minneapolis
Nixon, James B....Crosby
Olson, Lillian A....Ah-Gwah-Ching
§ Palmer, Harry A....Blackduck
Parker, Charles W....Wadena
Petraborg, Harvey T....Aitkin
Pierce, Chas. H....Wadena
§ Quanstrom, V. E....Brainerd
Ringle, Otto F....Walker
Rozycki, Anthony T....Pine River
Sanderson, A. G....Anoka
Schmitz, Glen P....Little Falls
‡ Schwyzer, Arnold G....Waltham, Mass.
Simons, Edwin J....Edina
Skaife, William F....Little Falls
Spurbeck, George A....Ah-Gwah-Ching
§ Stein, Raymond J....Pierz
‡ Stoy, R. A....Chicago, Ill.
*†Thabes, J. A., Sr....Brainerd
Thabes, J. A., Jr....Brainerd
Vandersluis, C. W....Bemidji
§ Watson, Alexander M....Royalton
Watson, Percy T....Northfield
§ Watson, Sidney W....Royalton
Whittemore, Dexter D....Bemidji
§ Will, Charles B....Bertha
§ Will, Wm. W....Bertha
Williams, M. M....Ah-Gwah-Ching
Zeigler, Charles M....Pine River

WABASHA COUNTY MEDICAL SOCIETY

Annual meeting, first Thursday after first Monday in October
Other meetings, as called by President
Number of Members—13

President
Ochsner, Clarence G....Wabasha

Secretary
Flesche, Bernard A....Lake City

§ Bayley, Emery C....Lake City
§ Bouquet, Bertram J....Wabasha
§ Bowers, Robert N....Lake City
† Collins, Joseph S....Wabasha
Ekstrand, LeRoy M....Wabasha
§ Ellis, Earl W....Elgin
§ Flesche, Bernard A....Lake City
§ Gjerde, Wm. P....Lake City
Glabe, Robert A....Plainview
§ Mahle, Donald G....Plainview
§ Ochsner, Clarence G....Wabasha
† Replogle, W. H....Los Angeles, Calif.
Wellman, T. G....Clinton, Iowa
*†Wilson, W. F....Lake City

WASECA COUNTY MEDICAL SOCIETY

Regular meetings, as decided
Annual meeting, January
Number of Members—10

President
Normann, Stephen T....Waseca

Secretary
Florine, Martin C....Janesville

§ Davis, Raymond D....Waseca
§ Florine, Martin C....Janesville
†§ Gallagher, Bernard J....Waseca
§ Hottinger, Raymond C....Janesville
§ McIntire, Homer M....Waseca
§ Normann, Stephen T....Waseca
§ Oeljen, Siegfried C. G....Waseca
§ Olds, George H....New Richland
† Swenson, Donald B....Minneapolis
§ Swenson, Orvie J....Waseca

WASHINGTON COUNTY MEDICAL SOCIETY

Regular meetings, second Tuesday in each month
Annual meeting, December
Number of Members—21

President
Johnson, Ray G.........Stillwater

Secretary
Carlson, Russel E.........Stillwater

§ Carlson, Russel E.........Stillwater
Fasbender, Herman.........Hastings
†§Haines, James H.........Stillwater
§ Holcomb, Joel T.....Marine-on-St. Croix
Hooper, Worth.........Hastings
Humphrey, Wade R.........Stillwater
§ Jenson, James E.........Stillwater
§ Johnson, Ray G.........Stillwater
Josewski, R. J.........Stillwater
§ Juergens, Manley.........Stillwater
Just, Herman J.........Hastings
Kulzer, Norbert.........Hastings
§ McCarten, F. M.........Stillwater
Mensheha, Nicholas.........Forest Lake
† Poirier, Jos. A.........Forest Lake
Racer, Harley J.........Stillwater
Ruggles, G. M.........Forest Lake
§ Sherman, Carnot H.........Bayport
§ Stuhr, John W.........Stillwater
Thompson, V. C.....Marine-on-St. Croix
Van Meier, Henry.........Stillwater

WEST CENTRAL MINNESOTA MEDICAL SOCIETY

Big Stone, Pope, Stevens, and Traverse Counties
Regular meetings, first Wednesday in March, May, September and November
Annual meeting, November
Number of Members—26

President
Eide, O. A.........Hancock

Secretary
Watson, Robert M.........Morris

§ Arneson, Arthur I.........Morris
§ Behmler, Fredk. W.........Morris
† Bolsta, Charles.........Ortonville
‡§Bucher, Foster D.........N. Y., N. Y.
†§Ebelin, Edward A.........Glenwood
§ Eide, O. A.........Hancock
*§Elsey, E. M.........Glenwood
† Elsey, James R.........Glenwood
† Fitzgerald, E. T.........Rialti, Calif.
§ Giesen, Allan F.........Starbuck
Hedemark, Homer H.........Ortonville
§ Karn, Jacob F.........Ortonville
§ Kooda, Jennings C.........Morris
Lee, G. E.........Glenwood
Letson, R. D.........Glenwood
§ Lindberg, Alfred L.........Wheaton
†§Linde, Herman.........Cyrus
§ Magnuson, Allen E.........Wheaton
* McIver, Bert A.........Lowry
§ Merrill, Robert W.........Morris
Muir, Walter E.........Browns Valley
§ O'Donnell, D. M.........Ortonville
§ Oliver, Irwin L.........Graceville
† Ransom, Matthias L.........Hancock
§ Rossberg, Raymond A.........Morris
‡§Salk, Richard J.........Sauk Rapids
§ Swedenburg, Paul A.........Glenwood
§ Watson, Robert M.........Morris

WINONA COUNTY MEDICAL SOCIETY

Regular meeting, first Monday in January, April, July and October
Annual meeting, first Monday in January
Number of Members—28

President
Boardman, D. V.........Winona

Secretary
Finkelnburg, Wm. O.........Winona

† Benoit, Frank T.........Winona
§ Christensen, Eli E.........Winona
§ Finkelnburg, Wm. O.........Winona
‡ Haesly, Warren W.........Winona
§ Hartwich, Roger F.........Winona
§ Heise, Herbert V. R.........Winona
§ Heise, Paul.........Winona
§ Heise, Phillip.........Winona
*†§Heise, W. F. C.........Winona
Hughes, S .O.........Winona
§ Johnston, L. F.........Winona
§ Loomis, G. L.........Winona
§ Mattison, Percy A.........Winona
† McLaughlin, Edmund M.........Winona
§ Meinert, Albert E.........Winona
§ Page, Raymond L.........St. Charles
†§Robbings, Chas. P.........Winona
§ Roemer, Henry J.........Winona
§ Rogers, Charles W.........Winona
§ Rollins, Pat.........St. Charles
§ Satterlee, Howard W.........Lewiston
§ Schmidt, Hilmar R.........Winona
§ Steiner, Irving W.........Winona
§ Tweedy, John A.........Winona
Vollmer, Fredk. J.........Winona
‡ Wilson, Louis J.........Winona
§ Wilson, Rolland H.........Winona
§ Woltjen, Myron J.........Rushford
§ Younger, Louis I.........Winona

WRIGHT COUNTY MEDICAL SOCIETY

Regular meetings every three months
Annual meeting, first Tuesday in October
Number of Members—17

President
Sandeen, Robert M.........Buffalo

Secretary
Catlin, Theodore J.........Buffalo

§ Anderson, Waldo P.........Buffalo
§ Bendix, Lester H.........Annandale
†§Catlin, John H.........Buffalo
§ Catlin, Theodore J.........Buffalo
§ Ellison, Frank E.........Monticello
§ Greenfield, Wm. T.........Cokato
Grundset, Ole J.........Montrose
§ Guilfoile, Pierre F.........Delano
Hagen, Kristofer.........Cokato
Hall, William E.........Maple Lake
§ Hart, William E.........Monticello
Larson, Loren J.........Minneapolis
§ Sandeen, Robert M.........Buffalo
§ Shragg, Robert I.........Howard Lake
§ Smorszczok, Mitrofan.........Monticello
§ Thielen, Robert D.........Saint Michael
§ Thomas, Wm. H.........Howard Lake

Alphabetic Roster

Key to Symbols: *Deceased; †Affiliate or Life Member; ‡In Service.

Aanes, Almer M.	Red Wing
‡ Abbott, Albert R.	Rochester
*†Aborn, W. H.	Hawley
Abraham, Arden L.	Duluth
Abramson, Milton	Minneapolis
Achor, Richard W. P.	Rochester
Adair, Albert F., Jr.	St. Paul
Adams, Bertram S.	Hibbing
Adams, Richard C.	Rochester
Addy, Edward R.	Gilbert
Adkins, Charles D.	Minneapolis
† Adkins, Charles M.	Thief River Falls
Adkins, Galen H.	Anoka
Affeldt, Danl. E.	Kasson
† Aga, John H.	Rochester
Ager, Ernest A.	Minneapolis
Agustsson, Hreidar	Minneapolis
Ahern, Eugene E.	Minneapolis
Ahlfs, Jacob J.	Caledonia
Ahola, Kenneth E.	Hibbing
† Ahrens, Albert E.	St. Paul
Ahrens, Curtis F.	Duluth
Ahrens, Robert M.	St. Paul
† Aitkens, Herbert B.	LeCenter
Akey, Robert M.	Rochester
Akins, Willard M.	Red Wing
Albrecht, H. H.	Chisago City
Alcorn, Wm. J.	Wabasso
‡ Alden, J. F., Jr.	Ft. Smith, Ark.
Alexander, Harlan A.	Minneapolis
Aling, Charles A.	Minneapolis
Allen, Edgar V. N.	Rochester
Allen, George S.	Cannon Falls
Allen, John H.	Montevideo
Allen, Raymond A.	Rochester
Allison, David D.	Litchfield
Althausen, Theo. L., Jr.	Minneapolis
Altnow, Hugo O.	Coral Gables, Fla.
† Amberg, Saml.	Rochester
Andersen, Howard A.	Rochester
Andersen, Silas C.	Minneapolis
Anderson, Arnold S.	St. Louis Park
Anderson, Chester A.	Hector
Anderson, Chester A.	Madison
Anderson, David P., Jr.	Austin
Anderson, David M.	Minneapolis
‡ Anderson, Donald C.	N. Y., N. Y.
† Anderson, Edward D.	Minneapolis
Anderson, Ernest M.	St. Cloud
Anderson, Ernest R.	Minneapolis
Anderson, Frank J.	Minneapolis
Anderson, Franklin C.	Owatonna
Anderson, Harold J.	Austin
Anderson, James J.	Mankato
Anderson, Karl W.	Minneapolis
Anderson, Margaret C.	Mankato
Anderson, Mark J.	Rochester
‡ Anderson, Milton W.	Rochester
Anderson, Richard E.	Willmar
Anderson, Richard W.	St. Paul
Anderson, Roger L.	Minneapolis
Anderson, U. Schuyler	Minneapolis
Anderson, Waldo P.	Buffalo
Anderson, Wallace E.	Clearbrook
Anderson, Wallace E.	Lakeville
Anderson, Wallace R.	Austin
Anderson, Wm. H.	Minneapolis
Anderson, Wm. T.	Minneapolis
Andreassen, Einar C.	St. Paul
Andreassen, Rolf	Minneapolis
Andresen, K. D'A.	Minneapolis
Andrews, Bernice F.	Holdingford
Andrews, Robert S.	Minneapolis
Andrews, Roy N.	Mankato
Ankner, Frank J.	Minneapolis
Antel, John J.	Rochester
Archer, Willard E.	Anoka
† Ardan, Nicholas, Jr.	Rochester
Arends, Archabald L.	Richfield
Arey, S. Lane	Minneapolis
Arko, Joseph L.	Hibbing
Arlander, Clarence E.	Minneapolis
Arling, Leonard S.	Minneapolis
Arms, James J.	Minneapolis
Armstrong, Ralph S.	Winnebago
Arndt, Harry Wm.	Detroit Lakes
Arnesen, John F.	Owatonna
Arneson, Arthur I.	Morris
Arnold, Anna W.	Minneapolis
Arnold, Elmer Wm.	Adrian
Arnquist, Andrew S.	St. Paul
Arny, Fredk. P.	St. Paul
Arvidson, Carl G.	Minneapolis
Arzt, Philip K.	St. Paul
Athens, Alvin G.	Duluth
Atmore, Wm. G.	Rochester
Aufderheide, Arthur C.	Duluth
† Aune, Martin	Minneapolis
† Aurand, Wm. H.	Minneapolis
Aurelius, J. Richards	St. Paul
Ausman, Duane R.	St. Paul
Autrey, William A.	St. Cloud
Austrian, Sol	Cosmos
‡ Baars, Conrad W.	Camp Cook, Calif.
Babb, Frank S.	St. Paul
Backus, Reno W.	Nopeming
Bacon, Donald K.	St. Paul
Badeaux, George I.	Brainerd
Bader, J. L.	Slayton
Baer, Walter	St. Paul
‡ Bagby, G. W.	Ft. Leonard Wood, Md.
Baggenstoss, Archie H.	Rochester
Baggenstoss, Osmond J.	Minneapolis
Bagley, Charles M.	Duluth
Bagley, Elizabeth C.	Duluth
Bagley, Russell W.	Minneapolis
Bagley, Wm. R.	Duluth
Baich, Velemir M.	Bovey
‡ Bain, Robert C.	Oakland, Calif.
Bair, Hugo L.	Rochester
Baird, Joe W.	Minneapolis
Baird, Raymond L.	Lake Crystal
Baken, Melvin P.	Minneapolis
Baker, A. B.	Minneapolis
Baker, Alfred T.	Minneapolis
Baker, Geo. S.	Rochester
† Baker, Harry R.	Hayfield
‡ Baker, Hillier L., Jr.	West Point, N. Y.
Baker, Jeannette L.	Fergus Falls
† Baker, Looe	Minneapolis
Baker, Milton E.	Minneapolis
Baker, Norman H.	Fergus Falls
Baker, Russell L.	Madison, Wisc.
Bakkila, Henry E.	Duluth
Balcome, Milton M.	St. Paul
Baleisis, Peter	Minneapolis
† Balfour, Donald C.	Rochester
Balfour, Wm. M.	Rochester
Balkin, Saml. G.	Minneapolis
Balogh, Charles J.	Minneapolis
Bank, Harry E.	San Francisco, Calif.
Banner, Edward A.	Rochester
Barber, Tracy E.	Austin
Bardon, Richard	Duluth
Bargen, J. Arnold	Rochester
Barker, John D.	Duluth
Barker, Nelson W.	Rochester
Barnes, Arlie R.	Rochester
Barnett, Joseph M.	St. Paul
Barney, Leon A.	Duluth
‡ Barno, Alex	St. Louis Park
Baronofsky, Ivan D.	Minneapolis
Barr, Lowell C.	Albert Lea
Barr, Maxwell M.	Minneapolis
Barr, Robert N.	Minneapolis
Barr, Ronald W.	Montevideo
Barrett, Earl E.	Duluth
Barron, Jesse J.	Minneapolis
† Barron, Moses	Minneapolis
Barron, S. Steven	Minneapolis
* Barry, Lee W.	St. Paul
† Barsness, Nellie O. N.	St. Paul
Basinger, Harvey R.	Mountain Lake
Basinger, Homer P.	Windom
Bastron, James A.	Rochester
Batdorf, B. Niles	Good Thunder
Bauer, Eugene L.	St. Paul
Baumgartner, Florian H.	Albany
† Baxter, Stephen H.	Minneapolis
Bayley, Emery C.	Lake City
‡ Bayard, Edwin D.	Rochester
Beach, Northrop	Minneapolis
Beahrs, Oliver H.	Rochester
† Beals, Hugh	St. Paul
Beaton, J. Gordon	Northfield
† Becker, Arnetta M.	Minneapolis
Becker, Fredk. T.	Duluth
Beckering, Gerrit	Edgerton
† Beddow, Ralph M.	Rochester
Bedford, Edgar Wm.	Minneapolis
Beech, Raymond H.	St. Paul
Beek, Harvey O.	St. Paul
Beer, John J.	St. Paul
‡ Behling, Fredk. L.	Barston, Calif.
Behmler, Fredk. W.	Morris
Behr, Orlo K.	Crookston
Beirstein, Samuel	Minneapolis
Beise, Rudolph A.	Brainerd
Beiswanger, Richard H.	Minneapolis
Bell, Chas. C.	St. Paul
† Bell, E. T.	Minneapolis
Bellomo, James	St. Paul
‡ Bellomo, John	St. Paul
Bellville, Titus P.	Minneapolis
Belzer, Meyer S.	Minneapolis
Bender, James H.	Brainerd
‡ Bender, Leonard F.	San Diego, Calif.
Bendix, Lester H.	Annandale
Benell, Otto E.	Virginia
Benepe, James L.	St. Paul
Benesh, Louis A.	Minneapolis
*†Benjamin, A. E.	Minneapolis
Benjamin, Edwin G.	Minneapolis
Benjamin, Harold G.	Minneapolis
Benjamin, Walter G.	Pipestone
Bennett, Warren A.	Rochester
† Bennion, P. H.	Isway, Montana
Benoit, Clair	Rochester
† Benoit, Frank T.	Winona
Benson, Alfred H.	Little Falls
Benson, E. S.	Minneapolis
Bentley, Norman P.	St. Paul
Bepko, Marie K.	Cloquet
Berdez, George L.	Duluth
Bergan, R. O.	Duluth
Berge, David O.	Roseau
Berge, Harry L.	Mora
† Berge, Henry B.	Rochester
† Berge, Kenneth	Rochester
Berger, Alex G.	Minneapolis
Bergh, Geo. S.	Miinneapolis
Bergh, Solveig M.	Minneapolis
Berglund, Eldon B.	Minneapolis
Bergman, Oscar B.	St. James
Bergquist, Karl E.	Battle Lake
† Berkman, David M.	Oronoco
Berkman, John M.	Rochester
Berkwitz, Nathaniel J.	Minneapolis
Berlin, Anthony S.	Hallock
Berman, Reuben	Minneapolis
‡ Bernatz, Philip E.	San Francisco, Calif.
Bernier, M. J.	No. St. Paul
Bernstein, Irving C.	Minneapolis
Bernstein, Wm. C.	St. Paul
Bessesen, Alfred N., Jr.	Minneapolis
Bessesen, Dan'l. H.	Buffalo Lake
† Bessesen, Wm. A.	Minneapolis
Beuning, John B.	St. Cloud
‡ Beyer, Eugene	San Francisco, Calif.
Bianco, Anthony J.	Duluth
‡ Bianco, Anthony J., Jr.	Duluth
Bicek, Jos. F.	St. Paul
Bickel, Wm. H.	Rochester
† Biedermann, Jacob	Thief River Falls
Bieter, R. N.	Minneapolis
† Bigelow, Chas. E.	Dodge Center
Bigler, Earl E.	Perham
Bigler, Ivan E.	Perham
Bilka, P. J.	Minneapolis
Billings, Ralph E.	Franklin
Binder, Manuel R.	Minneapolis
† Binet, Henry E.	Grand Rapids
† Binger, Henry E.	Phoenix, Ariz.
Bjornson, Robt. G.	N. Y., N. Y.
Black, B. Marden	Rochester
Black, Earl J.	St. Paul
Black, Wm. A.	New Ulm
Blackburn, Charles M.	Rochester
Blackmore, Sidney C.	Biwabik
Blake, Allen J.	Hopkins
Blake, James A.	Hopkins
Blake, Paul S.	Hopkins
† Blakey, Adam R.	Osakis
Blattspieler, Lucian H.	Worthington
Block, Melvin A.	Detroit, Mich.
Bloedel, Traugott J. G.	Osseo
Blomberg, Wm. R.	Princeton
Bloom, Joseph	Duluth
Bloom, Norman B.	Minneapolis
Blumberg, Henry B.	Fairmont
Blumenthal, Jacob S.	Minneapolis
† Bock, Ronald A.	St. Paul
Bockman, Michael W. H.	Hot Springs Nat'l Park, Ark.

Bodaski, Albert A. ..... Tyler
‡ Bodelson, A. H. ..... Fulda
† Boeckmann, Egil ..... St. Paul
Boehrer, John J. ..... Minneapolis
Bofenkamp, Benjamin ..... Minneapolis
Bofenkamp, F. Wm. ..... Luverne
‡ Bohn, Donald G. ..... Minneapolis
Boies, Lawrence R. ..... Minneapolis
Bolender, Harold L. ..... St. Paul
Boline, Clifford A. ..... Battle Lake
† Bolsta, Chas. ..... Ortonville
Bolstad, Owen C. ..... Little Falls
‡ Bolz, J. A. ..... Camp Carson, Colo.
Boman, Paul G. ..... Duluth
Bonello, Frank J. ..... St. Paul
Bonnet, John D. ..... Rochester
Boone, Ervin S. ..... Luverne
† Booth, Albert E. ..... Minneapolis
*†Boothby, W. M. ..... Albuquerque, N. M.
Boreen, Clifton A. ..... Minneapolis
Borg, Jos. F. ..... St. Paul
* Borgen, Alfred E. ..... St. Paul
Borgerson, Arthur H. ..... Long Prairie
† Borgeson, Egbert J. ..... St. Paul
Borman, C. N. ..... Minneapolis
Borowicz, Leonard A. ..... Minneapolis
Bosland, Howard G. ..... Willmar
† Bossard, John W. ..... Rochester
† Bossert, Clarence S. ..... Mora
‡ Boswell, J. Thornton ..... Portsmouth, Va.
Botham, Richard J. ..... Rochester
Bottolfson, Bottolf T. ..... Moorhead
Bouchelle, McLemore ..... Virginia
Bouma, John H. ..... Edgerton
† Bouma, Lewis R. ..... St. Paul
Bouquet, Bertram J. ..... Wabasha
Bowen, Robt. L. ..... Hibbing
Bowers, G. G. ..... Minneapolis
Bowers, Robt. N. ..... Lake City
† Bowes, Donald E. ..... Rochester
Boyd, David A., Jr. ..... Rochester
† Boyd, George K. ..... Rochester
* Boyd, Leon M. ..... Alexandria
Boyer, George S. ..... Crookston
† Boyer, Saml. H., Sr. ..... Duluth
Boyer, Saml. H., Jr. ..... Duluth
Boynton, Ruth E. ..... Minneapolis
Boysen, Herbert ..... Madelia
† Boysen, Peter ..... Bemidji
Braasch, John W. ..... Rochester
† Braasch, Wm. F. ..... Rochester
Brabec, Paul F. ..... Bagley
† Brackett, Ralph D. ..... Rochester
Bradley, N. J. ..... Willmar
Brady, Joan V. ..... Rochester
Brand, Geo. D. ..... St. Paul
† Brand, Wm. A. ..... Redwood Falls
Brandenburg, John T. ..... Rochester
Brandenburg, Robt. O. ..... Rochester
Branham, Donald S. ..... St. Peter
Bratrud, A. F. ..... Minneapolis
† Bratrud, Edward ..... Thief River Falls
Bratrud, Theo. E. ..... Minneapolis
Bratrude, Earl J. ..... St. James
Braun, Ohrmundt C. ..... Grand Rapids
Braun, Robert A. ..... Rochester
† Bray, Elwyn R. ..... St. Paul
Bray, Philip N. ..... Duluth
* Bregel, Fred L. ..... St. Paul
‡ Breitenbucher, R. B. ..... Columbia, S. C.
Brekke, Harvey J. ..... Minneapolis
Bretzke, Carl O. ..... Greenbush
Brickley, Paul M. ..... Rochester
Bridge, Allyn G. ..... Minneapolis
Bridge, Ezra V. ..... Cannon Falls
Briggs, John F. ..... St. Paul
† Brigham, Chas. F., Sr. ..... St. Cloud
Brigham, Chas. F., Jr. ..... St. Cloud
Brill, Alice K. ..... Minneapolis
Brindley, Clyde O. ..... Rochester
Brink, Adlai A. ..... Baudette
Brink, Donald M. ..... Hutchinson
Broadbent, James C. ..... Rochester
Broadie, Thos. E. ..... St. Paul
‡ Broders, C. W. ..... San Francisco, Calif.
Brodie, Walter D. ..... St. Paul
† Broker, Henry M. ..... Minneapolis
Brooker, Warren J. ..... Duluth
† Brooks, Charles N. ..... Minneapolis
Brown, Alexander ..... Rochester
† Brown, Alexander H. ..... Pipestone
† Brown, Edgar D. ..... Paynesville
‡ Brown, Hector M. ..... Walker
Brown, Henry A. ..... Rochester
Brown, Ian A. ..... Minneapolis
Brown, Joe R. ..... Rochester
† Brown, John C. ..... Los Gatos, Calif.
Brown, Philip W. ..... Rochester
Brown, Roland G. ..... Rochester
Brown, Wm. D. ..... Minneapolis
Bruhl, Heinz H. ..... Faribault
Brunsting, Louis A. ..... Rochester
Brusegard, James F. ..... Red Wing
Buchanan, Gerald S. ..... Deer River
‡ Bucher, Foster D. ..... New York, N. Y.
Buchstein, Harold F. ..... Minneapolis
† Buck, Fredk. H. ..... Shakopee
Buckley, Robt. P. ..... Duluth
Buesgens, Ralph H. ..... Waterville
Buie, Louis A. ..... Rochester
Buirge, Raymond E. ..... Minneapolis
Bulinski, Theo. J. ..... St. Paul
Bulkley, Kenneth ..... Minneapolis
Bunker, B. W. ..... Anoka
Burch, E. P., II ..... St. Paul
† Burch, Frank E. ..... St. Paul
Burchell, Howard B. ..... Rochester
Buresh, Kenneth L. ..... Westbrook
Burke, Edmund C. ..... Rochester
Burklund, Edwin C. ..... St. Paul
Burlingame, David A. ..... St. Paul
Burmeister, Richard ..... St. Paul
Burnett, Clem F., Jr. ..... Crookston
Burnham, Wesley H. ..... Minneapolis
Burns, Catherine ..... Albert Lea
Burns, Floyd M. ..... Milan
Burns, M. Alpheus ..... Milan
† Burns, Robt. M. ..... St. Paul
Burseth, Edgar C. ..... Mora
Burton, Carl G. ..... St. Paul
Buscher, Julius C. ..... St. Cloud
Bush, Robert P. ..... St. Paul
Bushard, Wilfred J. ..... Minneapolis
Busher, Herbert H. ..... St. Paul
Butler, John K. ..... Cloquet
Butt, Hugh R. ..... Rochester
Butturff, Carl R. ..... Freeborn
Butzer, John A. ..... Mankato
Buzzelle, Leonard K. ..... Minneapolis

Cable, Morris L. ..... Minneapolis
Cabot, Clyde M. ..... Minneapolis
* Cabot, V. S. ..... Minneapolis
Cady, Laurence H. ..... Minneapolis
Cain, Clark L. ..... St. Paul
Cain, James C. ..... Rochester
Cain, James H. ..... Hoffman
Cairns, Robert J. ..... Redwood Falls
† Calhoun, F. W. ..... Albert Lea
Callahan, Francis F. ..... St. Paul
Cameron, Isabell L. ..... Minneapolis
Cameron, John H. ..... Crookston
Campagna, Mario J. ..... Rochester
Campbell, Donald C. ..... Rochester
Campbell, Lowell M. ..... Minneapolis
Campbell, Orwood J. ..... Minneapolis
Cantwell, Wm. F. ..... International Falls
Caplan, Leslie ..... Minneapolis
‡ Card, Wm. H. ..... Miineapolis
Cardle, George E. ..... Brainerd
Carey, James B. ..... Minneapolis
Carey, John M. ..... Rochester
Carlander, Lester W. ..... Minneapolis
Carley, Walter A. ..... St. Paul
Carlson, Carl E. ..... Alexandria
Carlson, J. V. ..... Westbrook
Carlson, Lawrence ..... Minneapolis
Carlson, Leonard T. ..... Minneapolis
Carlson, Russell E. ..... Stillwater
Carlson, Vernon J. ..... Moorhead
Caron, Robt. P. ..... Minneapolis
Carr, David T. ..... Rochester
Carroll, John J. ..... Montevideo
Carroll, Wm. C. ..... St. Paul
Carryer, Haddon M. ..... Rochester
Carthey, Frank J. ..... New Ulm
Caspers, Carl G. ..... Minneapolis
† Catlin, John J. ..... Buffalo
Catlin, Theo. J. ..... Buffalo
Caruolo, Joseph E. ..... Rochester
† Cavanor, Frank T. ..... Minneapolis
Cedarleaf, Cherry B. ..... St. Paul
Ceder, Elmer T. ..... Minneapolis
Ceplecha, Stanley F. ..... Redwood Falls
Cervenka, Chas. F. ..... New Prague
Cesnik, R. J. ..... St. Cloud
Chadbourn, Chas. R. ..... St. Paul
† Chadbourn, Wayne A. ..... Minneapolis
Chalgren, Wm. S. ..... Mankato
Challman, Samuel A. ..... Minneapolis
Chambers, Winslow C. ..... Blue Earth
Chance, Donald P. ..... Rochester
Chapman, T. L. ..... Duluth
Chatterton, Carl C. ..... St. Paul
Chavez, Demetrio A. ..... Minneapolis
Chermak, Francis G. ..... Internat'l Falls
Chesler, Merrill D. ..... Minneapolis
Chesley, Albert J. ..... Minneapolis
Childs, Donald S., Jr. ..... Rochester
Chiles, N. H. ..... Rochester
Chisholm, Tague C. ..... Minneapolis
Christensen, Clarence H. ..... Duluth
Christensen, Eli E. ..... Winona
Christensen, L. E. ..... Minneapolis
Christensen, Norman A. ..... Rochester
Christiansen, Andrew ..... St. Paul
Christiansen, Harold A. ..... Jackson
* Christianson, H. W. ..... Minneapolis
Chunn, Stanley S. ..... Willmar
Clagett, Oscar T. ..... Rochester
Clark, Clarence L. ..... Duluth
Clark, Elizabeth A. ..... Duluth
Clark, Harry B. ..... St. Cloud
Clark, Henry B., Jr. ..... Minneapolis
Clark, Ivan T. ..... Duluth
Clark, Leslie Wm. ..... Spring Valley
Clarke, Eric K. ..... Minneapolis
Clay, Lyman B. ..... Minneapolis
† Clayburgh, Bennie J. ..... Rochester
Claydon, Howard F. ..... Red Wing
Cleaves, Wm. D. ..... Sauk Centre
† Clement, John B. ..... Lester Prairie
Clifford, Geo. W. ..... Alexandria
Closuit, Fredk. C. ..... Aitkin
* Clothier, E. F. ..... Elk River
Cochrane, Bryon B. ..... St. Paul
Cochrane, Ray F. ..... Minneapolis
Coddon, Walter D. ..... St. Paul
Coe, John I. ..... Minneapolis
Cohen, B. A. ..... Minneapolis
Cohen, Ellis N. ..... St. Paul
Cohen, Ephraim B. ..... Minneapolis
Cohen, Maynard M. ..... St. Paul
Cohen, Sumner S. ..... Oak Terrace
Colby, M. Y., Jr. ..... Rochester
Cole, Wallace H. ..... St. Paul
Coleman, John B. ..... St. Paul
Coll, James J. ..... Duluth
† Collie, H. G. ..... St. Petersburg, Fla.
† Collins, Arthur N. ..... Duluth
Collins, Joseph S. ..... Wabasha
† Collins, Robert A., Jr. ..... Rochester
Combacker, Leon C. ..... Fergus Falls
Comfort, Mandred W. ..... Rochester
Condit, Wm. H. ..... Minneapolis
‡ Conley, Francis W. ..... Ft. Hood, Texas
Conley, Robert H. ..... Mankato
Connelly, Michael E. ..... Rochester
‡ Connolly, Coleman J. ..... Ft. Belvoir, Va.
Connor, Chas. E. ..... St. Paul
Cook, Carroll K. ..... St. Paul
Cook, Edward N. ..... Rochester
Cook, Jay M. ..... Staples
Cooley, Jack C. ..... Rochester
Coombs, Carl H. ..... Cass Lake
Cooney, James F. ..... Rochester
Cooper, Charles C. ..... St. Paul
Cooper, John P. ..... Excelsior
Cooper, Maurice D. ..... Winnebago
Cooper, Robt. R. ..... Minneapolis
† Cope, H. B. ..... Virginia
Corbin, Kendall B. ..... Rochester
Corniea, Albert D. ..... Minneapolis
Correa, Dale H. ..... Minneapolis
*†Corrigan, J. E. ..... Spooner
Coseriu, Vasile ..... St. Cloud
Cosgriff, James A., Sr. ..... Olivia
Cosgriff, James A., Jr. ..... Olivia
† Costello, Addis ..... Rochester
Coulter, Harold E. ..... Madelia
Counseller, Virgil ..... Rochester
Countryman, Roger S. ..... St. Paul
Coventry, Markham B. ..... Rochester
Coventry, Wm. D. ..... Duluth
Covey, K. W. ..... Mahnomen
Cowan, Donald W. ..... Minneapolis
Cowan George M. ..... Duluth
Craig, Clair C. ..... International Falls
Craig, David M. ..... St. Paul
Craig, Joe D. ..... Rochester
Craig, M. Elizabeth ..... Minneapolis
Craig, W. McK. ..... Rochester
Cranmer, Richard R. ..... Minneapolis
Cranston, Robt. W. ..... Minneapolis
Creevy, Chas. D. ..... Minneapolis
Creighton, Ralph H. ..... Minneapolis
*†Crenshaw, John L. ..... Sanford, Fla.
Critchfield, Lyman R. ..... St. Paul
Cronwell, Bernhard J. ..... Austin
Crow, Earl R. ..... Walker
Crowley, James H. ..... St. Paul
Crudo, Vincent D. ..... St. Paul
† Crumbley, James J. ..... Rochester
Crump, James W. ..... St. Paul
Culligan, John M. ..... St. Paul
Culligan, Leo C. ..... Minneapolis
Culp, Ormond S. ..... Rochester
Culver, L. G. ..... St. Paul
Cundy, Donald T. ..... Minneapolis
Curtis, Rauen A. ..... Le Center
Cushing, Robert L. ..... Brainerd
† Cutts, George ..... Minneapolis

Dady, Elmer E.	Minneapolis
Daehlin, Rolf	Fergus Falls
Daggett, Donald R.	Minneapolis
Dahl, Elmer O.	Minneapolis
*†Dahl, G. A.	Mankato
‡ Dahl, James C.	Minneapolis
Dahl, John A.	Minneapolis
Dahlin, David C.	Rochester
‡ Dahlstet, John J.	San Diego, Calif.
† Daignault, Oscar	Benson
Dale, Lester N.	Red Lake Falls
‡ Daly, David	Rochester
† Daniel, Donald H.	Minneapolis
† Danielson, Karl A.	Litchfield
Danyluk, Michael	Minneapolis
Dargay, Cyril P.	Minneapolis
Daugherty, Guy W.	Rochester
David, Reuben	Minneapolis
Davis, Austin C.	Rochester
† Davis, Donald R.	Rochester
Davis, Edw. V.	St. Paul
Davis, Geo. D.	Rochester
† Davis, Harry L.	Rochester
Davis, Jay C.	Minneapolis
Davis, Lloyd T.	Wadena
Davis, Luther F.	Wadena
Davis, Raymond D.	Waseca
Davis, Robert E.	Rochester
Davis, Thomas L.	Wadena
Davis, Wm. I.	Mound
Dawson, James R.	St. Paul
Dawson, Lorin D.	Minneapolis
Dearing, Wm. H.	Rochester
† DeBoer, Hermanus	Edgerton
Decker, Chas. H.	St. Paul
Decker, David G.	Rochester
DeGeest, James H.	Goodhue
DeKruif, Hendrik	Fergus Falls
† Delmore, J. L., Sr.	Roseau
Delmore, J. L., Jr.	Roseau
Delmore, Robert J.	Roseau
Del Plaine, C. W.	Minneapolis
Demo, Robert A.	Albert Lea
Derauf, Benjamin I.	St. Paul
Deters, Donald C.	St. Paul
Detjen, Edward D.	Big Fork
Deveraux, Thos. J.	Wayzata
Devine, Kenneth D.	Rochester
DeWall, Richard A.	Anoka
DeWeerd, James H.	Rochester
Deweese, Wilford J.	Bemidji
Dewey, Donald H.	Owatonna
Dickson, F. H., Jr.	Proctor
† Dickson, Thos. H.	St. Paul
Diefenbach, E. J., Jr.	Minneapolis
Diehl, Harold S.	Minneapolis
Diessner, A. W.	Redwood Falls
Diessner, Grant R.	Rochester
Diessner, Henry D.	Minneapolis
‡ Dillard, P. G., Jr.	Camp Carson, Colo.
Dille, Donald E.	Litchfield
Dixon, Claude F.	Rochester
Doane, Joseph C.	Rochester
Dobson, M. W.	Mankato
Dobson, Ross A.	Rochester
Dockerty, Malcolm B.	Rochester
Dodds, Wm. C.	Detroit Lakes
Dodge, H. W., Jr.	Rochester
‡ Doherty, Elmer M.	New Prague
Dokken, James H.	Windom
Doman, V. Wm.	Lakefield
Doms, Vernon A.	Elbow Lake
Donaldson, Chas. S.	St. Cloud
Donatelle, E. P.	Minneapolis
† Donin, J. F.	Winnipeg, Can.
Donoghue, Francis E.	Rochester
* Donohue, P. F.	St. Paul
Donovan, Danl. L.	Albert Lea
Dordal, John	Sacred Heart
Dorge, Richard I.	Minneapolis
Dornbach, Robert A.	Excelsior
† Dornblaser, Harry B.	Minneapolis
Dorsey, Cletis S.	Rochester
Dorsey, Geo. C.	Minneapolis
Douglas, Kenneth W.	Willmar
Douglass, Bruce E.	Rochester
‡ Dovenmuehle, Robt. H.	Durham, N. C.
† Doxey, Gilbert L.	Minneapolis
Doyle, Geo. C.	Duluth
Doyle, James R.	Rochester
Doyle, Lawrence O.	Minneapolis
Drake, Carl B.	St. Paul
† Drake, Charles R.	Minneapolis
† Dredge, Homer P.	Sandstone
Dredge, Thomas E.	Minneapolis
Drexler, Geo. W.	Blue Earth
Drill, Herman E.	Hopkins
† Drips, Della G.	Rochester
Dry, Thos. J.	Rochester
Dubbe, Fredk. H.	New Ulm
DuBois, J. F., Sr.	Sauk Centre
DuBois, J. F., Jr.	Sauk Centre
Duff, Edwin R.	Minneapolis
Dummer, Donald J.	Minneapolis
Duncan, James W.	Moorhead
† Dungay, Neil S.	Northfield
Dunlap, Earl H.	Minneapolis
Dunn, Jack, Jr.	Rochester
Dunn, James N.	St. Paul
Dupont, Joseph A.	Excelsior
Duryea, Willis M.	Minneapolis
DuShane, James W.	Rochester
† Dutton, C. E.	Minneapolis
Dvorak, Benj. A.	Minneapolis
Dwan, P. F.	Minneapolis
Dworsky, Saml. D.	Minneapolis
Dwyer, John J.	Duluth
‡ Dykstra, Peter C.	West Point, N. Y.
Dysterheft, Adolf F.	Gaylord

Earl, Geo. A.	St. Paul
Earl, John R.	St. Paul
Eaton, Lealdes M.	Rochester
Eberley, Tobe S.	Benson
† Eberlin, Edw. A.	Glenwood
Eckdale, John E.	Marshall
Eckman, Philip F.	Duluth
Eckman, Ralph J.	Duluth
Edlund, Gustaf	St. Paul
Edwards, Jesse E.	Rochester
Edwards, Jos. W.	St. Paul
Edwards, Lloyd G.	St. Paul
Edwards, Thos. J.	St. Paul
Egge, Sanford G.	Albert Lea
Eginton, Chas. T.	St. Paul
Ehrenberg, Claude J.	Minneapolis
Ehrlich, Sol P.	Minneapolis
Eich, Matthew A.	Minneapolis
Eichhorn, Edmund P.	Minneapolis
Eide, O. A.	Hancock
Eiler, John	Park Rapids
Eisenstadt, David H.	Minneapolis
Eisenstadt, Wm. S.	Minneapolis
Eitel, Geo. D.	Minneapolis
† Ekblad, John W.	Duluth
Eklund, Carl D.	Duluth
Ekstrand, LeRoy M.	Wabasha
Elias, Frank J.	Duluth
Elkins, Earl C.	Rochester
Ellertson, Leonard M.	Albert Lea
Ellinger, Albert J.	Willmar
Elliot, Harold J.	Hayfield
Ellis, Earl Wm.	Elgin
Ellis, F. Henry	Rochester
Ellison, David E.	Minneapolis
Ellison, Ellis	Minneapolis
Ellison, Frank E.	Monticello
* Elsey, Edw. M.	Glenwood
† Elsey, James R.	Glenwood
† Elstner, Howard L.	Rochester
Ely, Orriman S.	St. Paul
Emanuel, Karl Wm.	Duluth
Emerson, Edward C.	St. Paul
Emmett, John L.	Rochester
Emmons, R. W.	St. Paul
Emond, Albert J.	Farmington
Emond, J. S.	Farmington
Endress, Edward K.	St. Paul
Engberg, Edward J.	Faribault
Engel, Joseph P.	Minneapolis
Engelhart, P. C.	Minneapolis
Englund, Elvin F.	Minneapolis
Engstrand, Oscar J.	Minneapolis
Engstrom, Robert	Mankato
Enroth, Oscar E.	St. Paul
Eppard, Raymond M.	Cloquet
Erdal, Ove A.	Albert Lea
Erich, John B.	Rochester
Erickson, Alvin O.	Long Prairie
Erickson, Clifford O.	Minneapolis
Erickson, Donald J.	Rochester
Erickson, Eskil	Halstad
Erickson, George P.	Hibbing
Erickson, John G.	Rochester
‡ Erickson, L. F.	Minneapolis
Erickson, Myron E.	Minneapolis
Erickson, Reuben F.	Minneapolis
Ericson, Reinhold M.	Wayzata
† Ericson, Swan	LeSueur
† Ernest, Geo. C. H.	St. Petersburg, Fla.
Ersfeld, Murray P.	St. Paul
Erskine, Gordon M.	Grand Rapids
† Ertel, Edward Q.	Ellendale
Esensten, Sidney	Minneapolis
† Eshelby, E. C.	St. Paul
† Esser, Robert A.	Rochester
‡ Estes, J. Earle	San Francisco, Calif.
* Estrem, C. O.	Fergus Falls
Estrem, Ralph L.	Fergus Falls
Estrem, Robert D.	Fergus Falls
* Estrem, Theo. A.	Hibbing
† Eusterman, George B.	Rochester
Eustermann, John J.	Mankato
Evans, Edward T.	Minneapolis
† Evans, Eugene G., Jr.	Rochester
Evans, Leslie	Sauk Rapids
Evans, Robert D.	Minneapolis
† Evarts, Arrah B.	Rochester
Evensta, John B.	Grand Rapids
‡ Evert, John A., Jr.	St. Paul

Faber, John E.	Rochester
Fahr, George	Minneapolis
Fansler, Walter A.	Minneapolis
Farkas, John V.	St. Paul
Fasbender, Herman T.	Hastings
*†Farrish, R. C.	Sherburn
Faulconer, Albert, Jr.	Rochester
Fawcett, Arthur M.	Renville
Fawcett, Keith R.	Duluth
Fee, John G.	St. Paul
Feeney, John M.	Minneapolis
‡ Feigal, David	A.F.B., Wyoming
Feigal, William M.	Thief River Falls
Feinberg, Philip	Minneapolis
Feinstein, Julius Y.	Minneapolis
Felder, Davitt A.	St. Paul
Felion, Arthur J.	St. Paul
Fellows, Manley F.	Duluth
Fenger, E. P. K.	Oak Terrace
Fergeson, James	Rochester
† Ferguson, James C.	St. Paul
Ferguson, Wm. C.	Walnut Grove
‡ Ferrell, Clarence R.	Grand Rapids
Ferris, Deward O.	Rochester
Fesenmaier, Otto B.	New Ulm
Fesler, Harold H.	St. Paul
Feuling, John C.	Duluth
‡ Fidelman, N. E.	Ft. Sheridan, Ill.
Field, Anthony H.	Farmington
Field, Chas. W.	Minneapolis
† Fieldman, E. Jay	Rochester
Figi, Fredk. A.	Rochester
Fingerman, David L.	Minneapolis
Fink, Daniel L.	St. Paul
Fink, Leo W.	Minneapolis
Fink, Walter H.	Minneapolis
Finkelnburg, Wm. O.	Winona
Fisch, Herbert M.	Austin
Fischer, John R.	Blooming Prairie
Fischer, Marion McC.	Duluth
Fisher, Dan W.	St. Paul
Fisher, Isadore I.	Minneapolis
Fisketti, Henry	Duluth
† Fitzgerald, D. F.	Wayzata
† Fitzgerald, E. T.	Rialto, Calif.
Fitzsimons, Wm. E.	Brainerd
Fjeldstad, Christian A.	Minneapolis
Flanagan, Harold F.	St. Paul
Flanagan, Leonard G.	Austin
Flancher, Leon H.	Crookston
Flannery, Hubert F.	St. Paul
Fleeson, Wm. H.	Minneapolis
† Fleming, A. S.	Madison, N. J.
Fleming, Dean S.	Hopkins
Fleming, Thos. N.	St. Cloud
Flesche, Bernard A.	Lake City
Fliehr, Richard R.	Minneapolis
Flink, Edmund B.	Minneapolis
Flinn, James B.	Redwood Falls
† Flinn, Thomas E.	Remer
Flom, Martin G.	Zumbrota
Flom, R. P.	St. Paul
‡ Flom, Robert S.	Echo
Florine, Martin	Janesville
† Fly, Orceneth A., Jr.	Rochester
Flynn, Bernard F.	Hibbing
Flynn, L. L., Jr.	St. Paul
† Fogarty, C. Wm., Sr.	St. Paul
Fogarty, C. Wm., Jr.	St. Paul
Fogelberg, Emil J.	St. Paul
Foker, Leslie W.	Minneapolis
Foley, Fredk. E. B.	St. Paul
Folken, Frank G.	Albert Lea
Folsom, Louis B.	Minneapolis
Ford, Burton C.	Marshall
Ford, Wm. H.	Minneapolis
Forsythe, James R.	St. Paul
Fortier, G. M. A.	Little Falls
Foss, Edward L.	Rochester
Foster, Orley W.	Minneapolis
Fowler, Lucius H.	Miinneapolis
Fox, James R.	Minneapolis
† Franchere, F. Wm.	Lake Crystal
Francis, David W.	Morristown
Frane, Donald B.	Minneapolis
Franklin, Gordon W.	Northome
† Frear, Rosemary	Minneapolis
Fredericks, Geo. M.	Minneapolis
Frederickson, Alice C.	Willmar
Fredricks, Merriam G.	Duluth
Freedman, Marshall A.	Rochester
Freeman, C. D., Jr.	St. Paul

Freeman, Craig..........Minneapolis
Freeman, Donald W.....St. Louis Park
‡ Freeman, Gerald I..........St. Paul
Freeman, John G.....Jamestown, N. D.
† Freeman, John P..........Glenville
Freidman, Louis L..........St. Paul
* Freligh, W. P..........Albert Lea
French, Lyle A..........Minneapolis
Frerichs, Cletus T..........Rochester
Frey, Richard J..........Minneapolis
† Freymann, John G..........Boston, Mass.
Friberg, Jos..........Minneapolis
Fricke, Robt. E..........Rochester
Fried, Louis A..........Minneapolis
Friedell, Aaron..........Minneapolis
Friedell, George..........Ivanhoe
Friedman, Harry S..........Minneapolis
Friedman, Jack..........Minneapolis
Friend, Charles A..........Minneapolis
Fritsche, Albert..........New Ulm
Fritsche, Carl J..........New Ulm
Fritsche, Theo. R..........New Ulm
Fritz, Wallace L..........St. Paul
Froats, Chas. W..........St. Paul
† Frost, Edward H..........Willmar
Frost, John B..........Minneapolis
Frost, Russell H..........Minneapolis
Frykman, Howard M..........Minneapolis
Fugina, Geo. R..........Mankato
Fuller, Alice H..........Minneapolis
Fuller, Benjamin F..........St. Paul
Fuller, Josiah..........Duluth
Funk, Victor K..........Oak Terrace
Furman, Lucie C..........Minneapolis
Furnell, Dale..........St. Paul

Gaard, Richard C..........Minneapolis
Gacusana, J. M..........Mahnomen
Gaida, Jos. B..........St. Cloud
† Gallagher, Bernard J..........Waseca
Gallett, L. E..........Minneapolis
Galligan, John J..........St. Paul
† Galligan, Margaret M..........Minneapolis
Galloway, John B..........Minneapolis
† Gambill, Carl M..........Rochester
Gambill, Earl E..........Rochester
Gammell, John H..........Minneapolis
Garbrecht, A. Wm..........St. Paul
Gardner, Victor H., Sr..........Fairmont
Gardner, Walter P..........St. Paul
Garlock, Arthur V..........Bemidji
Garlock, Dewitt H..........Bemidji
‡ Garlock, Grant L..........Bemidji
† Garrett, Chas., Jr..........Rochester
Garrow, David M..........St. Paul
Garske, George L..........Minneapolis
Garten, Joseph L..........Minneapolis
‡ Gastineau, Clifford F..........Rochester
‡ Gatchell, Frank G..........Savannah, Ga.
‡ Gaunt, Wm. D..........N. Y., N. Y.
Gaviser, David..........Minneapolis
Gehlen, Jos. N..........St. Paul
Geib, Marvin J..........Moorhead
Geiser, Peter M..........Alexandria
George, Robert P..........Fergus Falls
Geraci, Jos. E..........Rochester
Geurs, Benjamin R..........Mankato
Ghent, Chas. H..........St. Paul
Ghormley, Ralph K..........Rochester
Ghostley, Mary C..........Bemidji
Gibb, Robert P..........Rochester
Gibbs, Edward C..........St. Paul
Gibbs, Robert W..........Minneapolis
Giberson, Raymond G..........Rochester
Giebenhain, John N..........Minneapolis
Giere, Jos. C..........Minneapolis
Giere, Richard W..........Minneapolis
Giere, Silas W..........Benson
Giesen, Allan F..........Starbuck
† Giffin, Herbert Z..........Rochester
Giffin, Mary E..........Rochester
Gifford, R. W., Jr..........Rochester
Gilbert, Maurice G..........Minneapolis
* Gilkey, S. E..........St. Paul
Gillespie, Delmar R..........St. Paul
Gillespie, Malcolm G..........Duluth
Gilman, Lloyd C..........Willmar
† Gilmore, Rowland..........Crookston
Gingold, Benj. A..........Minneapolis
† Ginsberg, James P..........Rochester
Ginsberg, Robert L..........Rochester
*†Giroux, A. A..........Mankato
Girvin, Richard B..........Minneapolis
Gjerde, Wm. P..........Lake City
Glabe, Robert A..........Plainview
† Glaeser, John H..........Minneapolis
Gleason, Wallace A..........St. Paul
Goblirsch, Andrew P..........Sleepy Eye
Goehrs, Gilman H..........St. Cloud
† Goehrs, Henry W..........St. Cloud
Goldberg, Isadore M..........Minneapolis
Goldish, Danl. R..........Duluth
Goldish, Robert J..........Duluth
Goldman, Theo. I..........Minneapolis
Goldner, Meyer Z..........Minneapolis
Goldsmith, Joseph W..........St. Paul
‡ Goldstein, N. P..........Bethesda, Md.
† Goltz, Edward V..........St. Paul
‡ Goltz, Neill F..........St. Paul
Goltz, Robert W..........Minneapolis
Good, C. Allen, Jr..........Rochester
Good, Hoff D..........Minneapolis
Goodman, Chas..........Virginia
† Gordon, Donald L..........Rochester
Gordon, Philip E..........Minneapolis
Gordon, Sewell S..........St. Louis Park
Gossel, John D..........Rochester
Gowan, Lawrence R..........Duluth
† Graham, Archibald W..........Chisholm
‡ Graham, G. G..........Millington, Tenn.
Grahek, Jack P..........Ely
‡ Grais, Melvin L.....Colo. Springs, Colo.
Grant, Hendrie W..........St. Paul
Grant, John C..........Sauk Centre
Grant, Suzanne..........Minneapolis
Grattan, Robert T..........Rochester
Gratzek, F. R. E..........Minneapolis
Gratzek, Thos..........St. Paul
Grau, R. K..........St. Paul
Graves, Richard B..........Red Wing
† Gray, Frank D..........Marshall
Gray, Howard K..........Rochester
Gray, Royal C..........Minneapolis
Green, Paul A..........Rochester
Green, Robert A..........St. Louis Park
† Green, Robert C., Jr..........Rochester
Greenberg, Albert J..........Minneapolis
Greene, Daniel E..........Thief River Falls
Greene, Laurence F..........Rochester
Greenfield, Irving..........Minneapolis
Greenfield, Wm. T..........Cokato
† Greishiemer, E. M.....Philadelphia, Pa.
Gridley, John W..........Arlington
Griebe, Grant L..........Brownton
Griffin, George D., Jr..........Rochester
Griffin, John W., Jr..........Bemidji
Griffin, R. P..........Benson
Grimes, Burton P..........St. Peter
Grimes, Marian..........Minneapolis
Grindlay, John H..........Rochester
Grinley, Andrew V..........Grand Rapids
Grise, Wm. B..........Austin
Grohs, William H..........Duluth
Gronvall, Paul R..........Minneapolis
Groschupf, Theo P..........Bemidji
Grose, Fredk. N..........Clarissa
‡ Gross, J. B..........N. Y., N. Y.
Grotting, J. K..........Minneapolis
† Gruenhagen, Arnold P..........St. Paul
Grundset, Ole J..........Montrose
Guilbert, G. D..........Waukesha, Wisc.
Guilfoile, Pierre J..........Delano
‡ Gullickson, Glenn, Jr.....Bethesda, Md.
† Gullixson, Andrew..........Longmont, Colo.
Gully, Raymond J..........Cambridge
Gunlaugson, Fredk. C..........Minneapolis
Gushurst, Edward G..........Minneapolis
Gustason, Harold T..........Minneapolis
‡ Guy, Jack A..........New London
Gwinn, John L..........Rochester

Haas, J. F..........Fairfax
Haavik, John E..........Duluth
Habein, Harold C., Sr..........Rochester
Habein, Harold C., Jr..........Rochester
Haberer, Helen R..........Minneapolis
Haes, Julius E..........Mankato
‡ Haesly, Warren W..........Winona
‡ Hagedorn, Albert B..........N. Y., N. Y.
Hagen, Kristofer..........Cokato
† Hagen, Olaf J..........Moorhead
Hagen, W. S..........Minneapolis
† Haggard, G. D..........Minneapolis
† Haigler, Frank H., Jr..........Rochester
† Haines, James H..........Stillwater
Haines, Saml. F..........Rochester
Halbert, John J..........Duluth
Halenbeck, Philip L..........St. Cloud
Hall, Barnard..........St. Paul
Hall, Harry B..........Minneapolis
Hall, Wendell..........Minneapolis
Hall, Wm. E..........Maple Lake
Hallberg, Chas. A..........Minneapolis
Hallberg, Olav E..........Rochester
† Hallenbeck, D. F..........LaJolla, Calif.
Hallenbeck, George A..........Rochester
† Halliday, Phillip V..........Minneapolis
Hallin, Roger P..........Worthington
Halloran, Walter H..........Jackson
‡ Halme, W. B..........San Francisco. Calif.
Halper, Bernard..........Hibbing
Halpern, David J..........Brewster
Halpin, Jos. E..........Rush City
Halvorsen, Daniel K..........Owatonna
Hamlon, John S..........Fergus Falls
Hammar, Lawrence M..........Mankato
Hammes, Ernest M., Sr..........St. Paul
Hammes, Ernest M., Jr..........St. Paul
† Hammond, James F..........St. Paul
† Haney, Claude L..........Duluth
Hankerson, Robt. G.....Minnesota Lake
Hanlon, David G..........Rochester
Hannah, Hewitt B..........Minneapolis
Hanover, Ralph D.....International Falls
Hansen, Cyrus O..........Minneapolis
Hansen, Erling W..........Minneapolis
Hansen, Olga S..........Minneapolis
Hansen, Robert E..........Hibbing
‡ Hansen, Rollin M.....Falls Church, Va.
Hansen, Theo. M..........Albert Lea
† Hanson, Adolph M..........Faribault
Hanson, Ernest O..........Carlton
Hanson, Everett C..........New York Mills
Hanson, Harlow J..........Minneapolis
Hanson, Harold B..........St. Paul
Hanson, Henry V..........Minneapolis
Hanson, LeRoy W..........Pelican Rapids
Hanson, Lewis..........Frost
Hanson, Malcolm B..........Minneapolis
Hanson, Mark C..........Minneapolis
Hanson, Norbert O..........Rochester
Hanson, Wm. A. H..........Minneapolis
Happe, Lawrence J..........Minneapolis
Harbaugh, John T..........St. Paul
Harmon, Gaius E..........St. Paul
Harrington, Stuart W..........Rochester
Harris, Carl N..........Hibbing
† Harris, Evelyn S..........Seattle, Wash.
Harris, L. D..........Minneapolis
Harris, Lloyd E..........Rochester
Harrison, Percy W..........Worthington
Hart, Wm. E..........Monticello
Hartfiel, Herbert A..........Montevideo
Hartfiel, Wm. F..........St. Paul
Hartig, Marjorie..........St. Paul
Hartjen, Jason K..........Bemidji
Hartley, Everett C..........St. Paul
Hartman, Evelyn..........Minneapolis
Hartman, Howard R..........Rochester
Hartnagel, G. F..........Red Wing
Hartung, Elmer H..........Claremont
Hartwich, Roger F..........Winona
Hass, Fredk. M..........Minneapolis
† Hassett, Roger G..........Mankato
Hastings, DeForest R..........Minneapolis
Hastings, Donald W..........Minneapolis
Hatch, Walter E..........Duluth
Hauge, Erling T..........Minneapolis
Hauge, Malvin I..........Clarkfield
‡ Haugen, G. W.....San Francisco, Calif.
Haugen, John A..........Minneapolis
Hauser, George W..........Minneapolis
Hauser, Victor P..........St. Paul
‡ Havel, Robert J..........Minneapolis
Haven, Walter K..........Minneapolis
Havens, Fred Z..........Rochester
† Havens, J. G. W..........Austin
Hawkinson, R. P..........Minneapolis
Hawley, G. M. B., II..........Red Wing
Hayes, Albert F..........St. Paul
Hayes, James M..........Minneapolis
† Hayes, M. F..........Moose Lake
Hayles, Alvin B..........Rochester
Hays, Albert T..........Minneapolis
Head, Douglas..........Minneapolis
Healy, John R..........Rochester
Healy, Raymond T..........Pierz
Hebbel, Robt..........Minneapolis
Hebeisen, Milton B..........Chaska
Heck, Frank J..........Rochester
Heck, Wm. W..........St. Paul
Hedberg, Gustaf A..........Nopeming
Hedemark, Homer H..........Ortonville
Hedemark, T. A..........Ortonville
Hedenstrom, Frank G..........St. Paul
Hedenstrom, Paul H..........Duluth
Hedenstrom, Philip C..........Marshall
Hedin, Raymond F..........Red Wing
Hedlund, Chas. J..........St. James
† Hegge, Olav H..........Austin
Hegge, R. S..........Austin
Heiam, Wm. C..........Cook
Heiberg, Emmett A..........Fergus Falls
Heiberg, Olaf M..........Worthington
Heilig, Wm. R..........St. Paul
† Heilman, D. M. H.....San Diego, Calif.
Heilman, Fordyce R..........Rochester
† Heimark, John J..........Rochester
Heimark, Julius J..........Fairmont
‡ Heine, Geo. W..........Little Falls
Heinz, Ivy B..........Shakopee
Heinz, Lawrence H..........Shakopee
Heise, Herbert V. R..........Winona

	Heise, Paul	Winona
	Heise, Philip R.	Winona
*†	Heise, Wm. F. C.	Winona
‡	Heisler, John J.	Minneapolis
	Helden, Rudolph A.	Rochester
†	Helland, Gustav M.	Spring Grove
†	Helland, John W.	Spring Grove
	Heller, Edgar E.	Mankato
†	Helmholz, Henry F.	Rochester
	Helseth, H. K.	Pelican Rapids
	Hempel, Dean J.	Minneapolis
†	Hempstead, B. E.	Rochester
	Hench, Philip S.	Rochester
	Henderson, A. J. G.	No. St. Paul
	Henderson, Edward D.	Rochester
	Henderson, John W.	Rochester
	Henderson, Lowell L.	Rochester
†	Henderson, Melvin S.	Rochester
	Hendricks, Esten J.	St. Paul
†	Hendricks, Everett D.	Rochester
†	Hendrickson, John F.	Minneapolis
	Hengstler, Wm. H.	St. Paul
	Henn, Mary J.	Rochester
†	Henney, Wm. H.	McIntosh
	Henrikson, Earl C.	Minneapolis
	Henry, C. J.	Milaca
†	Henry, C. E.	Kirksville, Mo.
‡	Henry, H. W.	Benton, Ill.
‡	Henry, J. E.	Milaca
*	Henry, M. O.	Minneapolis
	Henry, M. R.	St. Peter
†	Hensel, C. N.	St. Paul
	Hepper, Norman G.	Rochester
	Herbert, Willis	Minneapolis
	Herbst, Richard F.	Willmar
	Herman, Samuel M.	St. Paul
	Hermann, H. W.	Minneapolis
	Hermanson, Peter E.	Hendricks
	Heron, Roy C.	St. Paul
	Herrmann, Edgar T.	St. Paul
†	Hertel, Garfield E.	Austin
	Hertz, Myron J.	St. Paul
	Hesla, Inman A.	Austin
†	Hewitt, Edith S.	Rochester
†	Hewitt, Richard M.	Rochester
†	Heyerdale, O. C.	Rochester
†	Higgins, John H.	Minneapolis
	Higgs, Walter W.	Park Rapids
	Hildebrand, J. E.	Bemidji
	Hilding, Anderson C.	Duluth
†	Hilger, Andrew, Wm.	St. Paul
	Hilger, Jerome A.	St. Paul
	Hilger, Laurence D.	St. Paul
	Hilker, Marcus D.	St. Paul
	Hill, Earl	Minneapolis
	Hill, Elmer M.	Minneapolis
	Hill, Fredk. E.	Duluth
	Hill, John R.	Rochester
†	Hillis, S. J.	East Bradenton, Fla.
	Hinckley, Robert G.	Minneapolis
	Hinderaker, Harris P.	Bird Island
	Hines, E. A., Jr.	Rochester
	Hiniker, Louis P.	St. Paul
	Hiniker, Peter J.	LeSueur
	Hinz, Walter E.	Willmar
	Hirschboeck, Frank J.	Duluth
	Hirsh, Stanton A.	Crookston
	Hirshfield, Frank	Minneapolis
	Hitchcock, Claude R.	Minneapolis
	Hochfilzer, John J.	St. Paul
	Hodapp, Robert V.	Willmar
	Hodgson, Corrin H.	Rochester
	Hodgson, Jane E.	St. Paul
	Hodgson, John R.	Rochester
	Hoehn, David	Holdingford
	Hoeper, Philip G.	Mankato
	Hoff, Herbert O.	Duluth
	Hoffbauer, Fredk.	Minneapolis
	Hoffert, Henry E.	Minneapolis
	Hoffman, Roy A.	Minneapolis
	Hoffman, Walter L.	Minneapolis
‡	Hoganson, D. E.	Bemidji
†	Hoidale, Andrew	Tracy
	Holcomb, Joel T.	Marine-on-St. Croix
†	Holcomb, O. W.	St. Paul
†	Hollands, William H.	Fisher
	Hollenhorst, R. W.	Rochester
	Hollinshead, W. H.	St. Paul
	Holm, Donald F.	Benson
*	Holm, H. H.	Glencoe
	Holman, Colin B.	Rochester
	Holmberg, Conrad J.	Minneapolis
	Holmen, Robt. W.	St. Paul
	Holmes, Alva E.	Rush City
	Holmstrom, Carle H.	Warren
	Holt, Allen H.	Rochester
	Holt, John E.	St. Paul
	Holzapfel, Fred C.	Minneapolis
	Hom, L. Y. W.	Battle Lake
‡	Hood, R. T.	San Francisco, Calif.
	Hooper, Worth A.	Hastings
	Hoover, Norman W.	Virginia
	Hopkins, George W.	St. Paul
‡	Horns, Howard L.	Colo. Springs, Colo.
	Horns, Richard C.	Minneapolis
	Horton, Bayard T.	Rochester
	Hottinger, Raymond C.	Janesville
	Houglum, Arvid J.	Lake Park
†	Houkom, Bjarne	T. T., East Africa
	Houkom, Samuel S.	Duluth
‡	Houle, Rollin J.	New Brighton
†	House, Zachariah E.	Burbank, Calif.
	Houston, Donald M.	Park Rapids
	Houts, Joseph C.	Dassel
	Hovde, Rolf	Winthrop
	Hovland, Melvin L.	Minneapolis
	Howard, Marshall I.	Mankato
	Howard, Merrill A.	St. Paul
	Howard, Robert B.	Minneapolis
	Howard, Solomon	Minneapolis
	Howard, Willard S.	St. Paul
	Howe, Newell W.	St. Paul
	Howell, Carter W.	Minneapolis
	Howell, Llewelyn P.	Rochester
	Howell, Milton M.	Glencoe
	Hoyer, Ludolph J.	Windom
	Hruza, Wm. J.	Madelia
	Hubbard, Otto E.	Brainerd
	Hubin, Edwin G.	Sandstone
	Hudec, Elwyn R.	Echo
	Hudspeth, Wm. T.	Madison
	Huebert, Dan W.	Hutchinson
	Huenekens, E. J.	Minneapolis
†	Huffington, H. L.	Mankato
	Huffington, Herb L.	Waterville
	Hughes, Bernard J.	Brainerd
	Hughes, S. O.	Winona
	Huizenga, Kenneth A.	Rochester
	Hullsiek, Harold E.	St. Paul
	Hullsiek, Richard B.	Ft. Snelling
*†	Hultkrans, J. C.	Minneapolis
	Hultkrans, R. E.	Minneapolis
	Humphrey, E. W., Sr.	Moorhead
	Humphrey, E. W., Jr.	Moorhead
	Humphrey, Wade R.	Stillwater
*†	Hunt, A. F.	Alhambra, Calif.
	Hunt, Arthur B.	Rochester
	Hunt, Roscoe C.	Fairmont
	Hunt, William	Rochester
	Hunter, James S.	Rochester
	Hunter, Murray, H.	Farmington
†	Hurd, Annah	Minneapolis
	Hurwitz, Milton M.	St. Paul
	Hustad, Edward G.	Montevideo
‡	Hutchinson, C. J.	Providence, R. I.
	Hutchinson, Henry	Moose Lake
†	Huxley, Fredk. R.	Faribault
	Hymes, Charles	Minneapolis
†	Hynes, John E.	Minneapolis
†	Ide, Arthur W., Sr.	St. Paul
	Ide, Arthur W., Jr.	Minneapolis
	Idstrom, L. G.	Minneapolis
	Ikeda, Kano	St. Paul
‡	Indihar, John E., Jr.	Virginia
	Ingerson, Carl A.	St. Paul
	Inglis, Wm.	Redwood Falls
†	Irvine, Harry G.	Minneapolis
	Irwin, Homer R.	Hibbing
	Iverson, Rolf M.	Minneapolis
	Ivins, John C.	Rochester
	Jackman, Raymond J.	Rochester
‡	Jackson, Richard L.	Rochester
	Jacobs, Douglas L.	Willmar
†	Jacobs, Johannes C.	Willmar
	Jacobson, Clarence	Chisholm
	Jacobson, C. W.	Breckenridge
	Jacobson, F. C.	Duluth
	Jacobson, Loren J.	Minneapolis
	Jacobson, W. E.	St. Louis Park
	James, Ellery M.	St. Paul
	James, John W.	White Bear Lake
‡	Jamplis, R. W.	San Francisco, Calif.
	Janda, George W.	Port Clayton Hospital, Canal Zone
	Janecky, Allen G.	Baudette
	Janes, Joseph M.	Rochester
	Janssen, Martin E.	St. Paul
	Jarvis, Marilyn A.	St. Paul
	Jay, Alan R.	Minneapolis
‡	Jensen, Adrian R.	Long Island, N. Y.
	Jensen, Alvin M.	Brownton
	Jensen, Harry C.	Minneapolis
†	Jensen, Marius J.	Minneapolis
	Jensen, Nathan K.	Minneapolis
	Jensen, Reynold A.	Minneapolis
	Jensen, Thorvold J.	Duluth
	Jenson, James E.	Stillwater
	Jerome, Bourne	Minneapolis
	Jerome, Elizabeth E.	Minneapolis
	Jeronimus, Henry J.	Duluth
	Jesion, Jos. W.	St. Paul
	Jessico, Charles M.	Duluth
‡	Jeub, Robert P.	San Antonio, Texas
	Joffe, Harold H.	Virginia
	Johanson, Waldemar G.	St. Paul
	Johnson, Adelaide McF.	Rochester
	Johnson, Aldridge F.	Isle
	Johnson, Arthur B.	Minneapolis
	Johnson, August E.	Minneapolis
	Johnson, Carl E.	St. Paul
	Johnson, Carl Eric	Rochester
‡	Johnson, Curtis M.	Dawson
	Johnson, C. Percy	Tyler
	Johnson, Douglas L.	Little Falls
‡	Johnson, Edward A.	Virginia
†	Johnson, Einer W., Sr.	Bemidji
	Johnson, Einer W., Jr.	Rochester
	Johnson, Emil W.	Minneapolis
	Johnson, Frank E.	Minneapolis
‡	Johnson, H. C.	San Diego, Calif.
	Johnson, Harry A.	Minneapolis
	Johnson, Herbert W.	St. Paul
	Johnson, James A.	Minneapolis
	Johnson, John W.	Minneapolis
	Johnson, Karl E.	Duluth
	Johnson, Malcolm R.	Minneapolis
†	Johnson, Morris B.	St. Paul
	Johnson, Norman P.	Minneapolis
	Johnson, Norton T.	Minneapolis
	Johnson, Olga H.	Moorhead
	Johnson, Orville H.	Omaha, Nebr.
	Johnson, R. J.	St. Paul
	Johnson, Ralph B.	Lanesboro
	Johnson, Ray G.	Stillwater
	Johnson, Reinald G.	Minneapolis
	Johnson, Reuben A.	Minneapolis
	Johnson, Robert E.	Minneapolis
	Johnson, Roger S.	Wayzata
	Johnson, Rudolph E.	Mankato
	Johnson, Vilhelm M.	Dawson
	Johnson, Wm. E.	Morgan
	Johnson, Youbert T.	Minneapolis
	Johnsrud, Luverne W.	Hibbing
	Johnston, Edward V.	Rochester
	Johnston, Henry W.	Virginia
	Johnston, Leonard F.	Winona
†	Johnston, Rufus O.	Hibbing
	Jolin, Francis M.	Bovey
†	Jones, Alvah W.	Red Wing
	Jones, David G.	Minneapolis
†	Jones, E. Mendelssohn	St. Paul
†	Jones, Herbert W., Jr.	Minneapolis
	Jones, Orville H.	Mankato
	Jones, Richard F.	Rochester
	Jones, Richard H.	Minneapolis
	Jones, Richard N.	St. Cloud
	Jones, Robcliff V.	Rochester
	Jones, Wm. R.	Minneapolis
†	Jordan, Kathleen B.	Granite Falls
	Jordan, Lewis S.	Granite Falls
†	Jordan, Stanley	Rochester
	Josewich, Alexander	Minneapolis
	Josewski, Raymond J.	Stillwater
	Judd, Allen S.	Minneapolis
	Judd, Edward S., Jr.	Rochester
	Judd, Walter H.	Washington, D. C.
	Juergens, Herman M.	Belle Plaine
†	Juergens, John L.	Belle Plaine
	Juergens, Manley F.	Stillwater
	Juers, Edward H.	Red Wing
	Juhl, John H.	Minneapolis
†	Juliar, Richard O.	Glendale, Calif.
	Juntunen, Roy R.	Duluth
	Jurdy, Mitchell J.	Minneapolis
	Just, Herman J.	Hastings
	Kaasa, Lawrence J.	St. Peter
	Kabrick, Ola A.	St. Peter
	Kadesky, Harold B.	Minneapolis
	Kaiser, Milton L.	New Ulm
	Kalin, Oscar T.	Minneapolis
	Kallestad, Leonard L.	Minneapolis
	Kamman, Gordon R.	St. Paul
†	Kamp, Byron A.	Albert Lea
	Kanne, Earl R.	Brainerd
	Kantar, Bruce L.	Minneapolis
	Kaplan, David H.	St. Paul
	Kaplan, Harold A.	St. Louis Park
	Kaplan, John J.	Minneapolis
	Kapsner, Alfred T.	Princeton
	Karges, L. E.	Grand Rapids
	Karleen, Bernard N.	Jackson
	Karleen, Conrad I.	Minneapolis
	Karlen, Markle	Minneapolis
	Karn, Jacob F.	Ortonville
	Karon, Irvine M.	St. Paul
	Kasper, Eugene M.	St. Paul
	Kath, Reinhard H.	Arlington
	Katz, Louis J.	Long Beach, Calif.
†	Katz, Yale J.	Minneapolis
*	Katzberg, Lewis	Willmar

	Kaufman, Edward J.	Appleton
	Kaufman, Herschel J.	Minneapolis
	Kaufman, Walter B.	Mankato
	Kaufman, Wm. C.	Appleton
	Kearney, Rochfort W.	Mankato
	Kearns, Thomas P.	Rochester
	Keating, F. R., Jr.	Rochester
	Keefe, Rolland E.	St. Paul
	Keil, Marcus A.	Albert Lea
	Keith, Haddow M.	Rochester
†	Keith, Norman M.	Rochester
	Keith, Paul J.	Milaca
	Keithahn, Elmer E.	Sleepy Eye
	Kelby, Gjert M.	Minneapolis
	Kelley, Walter M.	Duluth
	Kelly, Albert C.	Duluth
	Kelly, Chas. F.	Minneapolis
	Kelly, James H.	St. Paul
	Kelly, John F.	Cold Spring
	Kelly, John P.	Minneapolis
†	Kelly, John V.	St. Paul
	Kelly, Robert T.	Nashwauk
†	Kelsey, Carleton G.	St. Paul
	Kelsey, Chauncey M.	St. Paul
	Kemp, Alphonse F.	Mankato
	Kenefick, Emmett V.	St. Paul
	Kennedy, Claude C.	Minneapolis
	Kennedy, George L.	Faribault
†	Kennedy, Jane F.	Minneapolis
	Kennedy, Roger L. J.	Rochester
‡	Kent, Geo. B.	Ft. McPherson, Ga.
	Kenyon, Thomas J.	St. Paul
	Kerkhof, Arthur C.	Minneapolis
	Kernohan, James W.	Rochester
	Kesting, Herman	St. Paul
	Kevern, Jay L.	Henning
	Keyes, Robert W.	Pipestone
‡	Kiely, James P.	San Francisco, Calif.
‡	Kiely, Joseph M.	Joliet, Ill.
	Kierland, Robt. R.	Rochester
	Kiesler, Frank, Jr.	Minneapolis
	Kilbride, Edwin A.	Worthington
‡	Kilby, Ralph A.	Columbia, So. Car.
	Kilmer, Warren L.	International Falls
	Kimmel, George C.	Red Wing
†	King, E. A.	Minneapolis
	King, Frances W.	Oak Terrace
†	King, George L.	St. Paul
	Kinkade, Byron R.	Ada
	Kinports, Edward B.	San Diego, Calif.
	Kinsella, Thos. J.	Minneapolis
	Kirby, Thomas J., Jr.	Rochester
†	Kirk, George P.	East Grand Forks
	Kirklin, Byrl R.	Rochester
	Kirklin, John W.	Rochester
	Kistler, Alvin J.	Minneapolis
	Kitzberger, Peter J.	New Ulm
	Klakeg, Clayton H.	Rochester
†	Klass, Donald W.	Rochester
	Klefstad, Lloyd H.	Greenbush
	Klein, Harry	Duluth
	Klein, Henry N.	St. Paul
	Klein, Wm. A.	Duluth
	Klicka, Karl S.	Minneapolis
	Klima, Wm. W.	Stewart
	Kline, Richard F.	Wayzata
	Knapp, Frank N.	Duluth
	Knapp, Miland E.	Minneapolis
	Knight, Edwin G.	Swanville
	Knight, Ralph T.	Minneapolis
†	Knight, Ray R.	Minneapolis
	Knoche, Harvey A.	Morgan
	Knoll, W. V.	Duluth
	Knudsen, Helen L.	Minneapolis
*	Knutson, G. A.	Hallock
	Knutson, Gerhard E.	St. Paul
	Knutson, Lewis A.	Spring Grove
	Knutson, Robert C.	St. Paul
	Koelsche, Giles A.	Rochester
	Koenecke, Fred H., Jr.	Lakefield
	Koenig, Robert P.	Paynesville
	Koenigsberger, Chas.	Mankato
	Koepcke, Gerald M.	Minneapolis
	Kohlbry, Carl O.	Duluth
†	Kohler, Delphin W.	Tacoma, Wash.
	Kolars, James J.	Faribault
	Kolb, Lawrence C.	Rochester
	Koller, Hermann M.	Minneapolis
	Koller, Louis R.	Minneapolis
	Koller, Robert L.	Minneapolis
	Kooda, Jennings, C.	Morris
	Korchik, John P.	Minneapolis
	Korda, Henry A.	Pelican Rapids
*	Kortsch, Ferdinand P.	Prior Lake
	Kosiak, John	Minneapolis
	Koskela, Lauri E.	Shaker Heights, Ohio
	Kostick, William R.	Fertile
	Kotchevar, Frank R.	Eveleth
	Kottke, Fredk. J.	Minneapolis
	Kotval, Russell J.	Pipestone
	Koucky, Rudolph W.	Minneapolis
	Kovack, Freeman D.	Minneapolis
	Koza, Donald W.	St. Paul
	Kozberg, Oscar	Moose Lake
	Kraemer, George N.	Fairmont
	Kragh, Lyle O.	Braham
	Krause, C. W.	Fairmont
	Kremen, Arnold J.	Minneapolis
	Kreuzer, Titus C.	Marshall
	Krezowski, Thomas K.	St. Paul
	Krieser, Albert E.	Anoka
	Kroboth, Frank J., Jr.	Rochester
‡	Kroll, Harry G.	Rochester
‡	Krout, Robert M.	New Cumberland, Pa.
	Krueger, Victor R.	Nopeming
	Krusen, Frank H.	Rochester
	Kruzick, S. J.	Sleepy Eye
	Krystosek, Lee A.	Clara City
	Kucera, Frank J.	Hopkins
	Kucera, Louis B.	Lonsdale
	Kucera, Stanley T.	Northfield
	Kucera, Wm. J., Sr.	Minneapolis
	Kucera, Wm. J., Jr.	Minneapolis
	Kugler, Alex A.	St. Paul
	Kuhlmann, Lawrence B.	Melrose
	Kulstad, Oscar S.	Dodge Center
	Kulzer, Norbert J.	Hastings
	Kunkel, Wm. M., Jr.	Rochester
†	Kurtin, Henry J.	Milwaukee, Wisc.
†	Kurtin, Jos. J.	Cudahy, Wisc.
	Kuske, Albert W.	St. Paul
	Kusske, Arthur L.	New Ulm
†	Kusske, Bradley W.	St. Louis Park
	Kusske, Douglas R.	St. Paul
	Kusz, Clarence	Minneapolis
	Kvale, Walter F.	Rochester
	Kvitrud, Gilbert	St. Paul
	LaBree, John W.	St. Louis Park
	LaBree, Robert H.	Duluth
†	Lacy, Paul E.	Rochester
	LaGaard, Sheldon M.	Minneapolis
	Laikola, Leslie A.	Adrian
†	Laird, Arthur T.	Duluth
	Lajoie, John M.	Minneapolis
	Lake, Clifford F.	Rochester
	Lamb, H. Douglas	Minneapolis
	Lang, Leonard A.	Minneapolis
	Lange, Robert D.	Minneapolis
	Langhoff, Arthur H.	Mankato
	Lannin, Bernard G.	St. Paul
	Lannin, Donald R.	St. Paul
	Lapierre, Arthur P.	Minneapolis
	Lapierre, Jean T.	Minneapolis
	Larrabee, Walter F.	St. Paul
	Larsen, Frank Wm.	Minneapolis
	Larson, Arnold	Detroit Lakes
	Larson, Clarence M.	Minneapolis
	Larson, Donald M.	Fergus Falls
	Larson, Donald M.	Minneapolis
	Larson, Eva Jane	St. Paul
	Larson, Gerald E.	Cambridge
	Larson, James T.	So. St. Paul
	Larson, Kenneth R.	St. Paul
	Larson, Leighton	Watertown
	Larson, Lawrence M.	Minneapolis
	Larson, Leonard M.	Oak Terrace
	Larson, LeRoy J.	Bagley
	Larson, Loren J.	Minneapolis
	Larson, Martin L.	St. Paul
	Larson, Milo H.	Lake Benton
	Larson, Oliver E. H.	Zumbrota
	Larson, Ralph H.	Anoka
	Larson, Roger C.	Minneapolis
	Latterell, Kenneth E.	Duluth
†	LaVake, Rae T.	Minneapolis
	Law, Harrison E.	Virginia
	Law, William M.	Rochester
	Lax, Morris H.	St. Paul
	Laymon, Carl W.	Minneapolis
†	Lazar, Henry L.	Excelsior
	Lazarte, Jorge A.	Rochester
†	Leahy, Bartholomew	St. Paul
	Leavenworth, Richard O., Sr.	St. Paul
†	Leavenworth, Richard O., Jr.	St. Paul
†	Leavitt, H. H.	Phoenix, Ariz.
	Lebowske, Jos. A.	Minneapolis
	Leck, Paul C.	Austin
	Leddy, Eugene T.	Rochester
	Lee, G. E.	Glenwood
	Lee, Hubert W.	Brainerd
	Lee, Norman J.	Tracy
†	Lee, Philip R.	Rochester
†	Lee, Walter N.	Claremont, Calif.
‡	Leek, Joseph H.	Duluth
	Leemhuis, Andrew J.	Minneapolis
	Leggett, Elizabeth A.	Bemidji
	Leibold, Herbert H.	Parkers Prairie
	Leick, Richard M.	St. Paul
	Leitch, Archibald	St. Paul
*†	Leitschuh, L. F.	Winsted
	Leland, Harold R.	Minneapolis
†	Lemon, Willis S.	Clifton Forge, Va.
	Lenander, Mellvin E.	St. Peter
	Lenarz, Albert J.	Browerville
	Lende, Norman	Faribault
	Lenz, Jos. R.	Morton
	Lenz, Otto A.	Minneapolis
	Leonard, Lawrence J.	Minneapolis
	Leonard, Saml.	Minneapolis
†	Leopard, Brand A.	Brownsville, Texas
	Lepak, Francis J.	Duluth
	Lepak, John A.	St. Paul
	Leppo, N. Erkki	Duluth
†	Lerche, William	Cable, Wisc.
	Lerner, A. Ross	Minneapolis
	Lescoe, Richard J.	Rochester
‡	Lester, Malcolm J., Jr.	Truman
	Letson, R. D.	Glenwood
	Lev, Ralph	Rochester
	Leven, N. Logan	St. Paul
	Leverenz, Carleton W.	St. Paul
	Levin, Bert G.	St. Paul
	Levitt, George X.	St. Paul
	Lewis, Arthur J.	Henning
	Lewis, Barton L.	Rochester
†	Lewis, Claude B.	St. Cloud
	Lewis, F. John	Minneapolis
‡	Lewis, J. S., Jr.	Minneapolis
†	Lexa, Frank J.	Lonsdale
	Libert, John N.	St. Cloud
	Lick, Chas. L.	St. Paul
‡	Lick, Louis C.	San Francisco, Calif.
	Lick, Wm. J., Jr.	St. Paul
†	Liedloff, Adolph G.	Mankato
	Lien, Richard J.	St. Paul
	Liffrig, Wm. W.	Red Wing
	Lilleberg, Norbert J.	St. Paul
	Lillehei, C. Walton	Minneapolis
	Lillehei, Elmer J.	Robbinsdale
	Lillie, Harold I.	Rochester
	Lillie, John C.	Rochester
	Lima, Ludvig R.	Montevideo
	Limbeck, Donald A.	Le Sueur
‡	Lind, C. J., Jr.	Ft. Sam Houston, Texas
	Lindahl, Merlyn J.	Sherburn
	Lindberg, Alfred L.	Wheaton
	Lindberg, Arvid C.	Minneapolis
	Lindberg, Vernon L.	Minneapolis
	Lindberg, Winston R.	Minneapolis
	Lindblom, Alton E.	No. Mankato
†	Linde, Herman	Cyrus
	Lindell, Robert	St. Paul
‡	Lindeman, R. J.	Montgomery, Ala.
	Lindemann, Charles E.	Minneapolis
	Lindgren, Russell C.	Minneapolis
	Lindner, Janus C.	Minneapolis
	Lindquist, Richard H.	Minneapolis
	Linner, Gunnar	Minneapolis
	Linner, Henry P.	Minneapolis
	Linner, John H.	Minneapolis
	Linner, Paul W.	Minneapolis
	Lippman, Emanuel S.	Minneapolis
	Lippman, Hyman S.	St. Paul
	Lippmann, Elmer W.	Hutchinson
	Lipschultz, Oscar	Minneapolis
	Lipscomb, Paul R.	Rochester
	Litchfield, John T.	Minneapolis
	Litin, Edward M.	Rochester
	Litman, Abraham B.	Minneapolis
	Litman, Saml. N.	Duluth
	Lober, Paul H.	Minneapolis
	Lofgren, Karl A.	Rochester
	Lofsness, Stanley V.	Oak Terrace
†	Logan, Archibald H.	Rochester
	Logan, George B.	Rochester
†	Logefeil, Rudolph C.	Minneapolis
	Lohmann, John G.	Pipestone
	Loken, Selmur M.	St. Paul
	Loken, Theodore	Ada
	Lommen, Peter A.	Austin
	Longfellow, Henry W.	Brainerd
	Loomis, Earl A.	Minneapolis
	Loomis, G. L.	Winona
	Lorentzen, Ernest S.	Detroit Lakes
‡	Lorton, Wm. L.	Rochester
	Lott, Fredk. H.	Minneapolis
	Love, Fredk. A.	Carlos
	Love, J. Grafton	Rochester
	Lovett, Beatrice R.	Oak Terrace
	Lowe, Earl R.	So. St. Paul
	Lowe, Thos. A.	So. St. Paul
	Lowry, Elizabeth C.	Minneapolis
	Lowry, Paul	Minneapolis
	Lowry, Thomas	Minneapolis
	Luck, Hilda	Mankato
‡	Luckemeyer, C. J.	Camp Polk, La.
	Lueck, Wallace W.	Minneapolis

Lufkin, Nathaniel H..........Minneapolis
Lund, Anthony J..........Cleveland, Ohio
Lund, Carl J. T..........Fergus Falls
Lund, George W..........Minneapolis
Lund, Werner L. J..........Staples
Lundberg, Ruth I..........Minneapolis
Lundblad, Robert M..........Clara City
Lundblad, Roy A..........Minneapolis
Lundblad, S. W..........Minneapolis
Lundell, Carl L..........Granite Falls
* Lundgren, A. C..........Minneapolis
Lundholm, Arthur M..........St. Paul
Lundquist, Curt W..........Owatonna
Lundquist, V. J. P..........Minneapolis
Lundsten, Leslie C..........Bemidji
Lundy, John S..........Rochester
Lynch, Francis W..........St. Paul
† Lynch, Matthew J..........Minneapolis
† Lynn, Thomas E..........Rochester
Lysne, Henry..........Minneapolis
Lysne, Myron..........Minneapolis
Lysyj, Anatol..........Minneapolis

MacCarty, Collin S..........Rochester
† MacCarty, W. C..........Rochester
MacDonald, Danl. A..........Minneapolis
MacDonald, John W..........Minneapolis
MacDonald, Roger A..........Little Fork
MacFarlane, P. H..........Chisholm
Mach, Frank B..........Minneapolis
‡ Mach, Ralph F..........Minneapolis
MacKinnon, D. C..........Minneapolis
Macklin, W. E., Jr..........Wilmar
‡ Mackoff, S. M..........Scott Field, Ill.
MacRae, Gordon C..........Duluth
Madden, John F..........St. Paul
Madison, Mitchell S..........Rochester
Madland, Robert S..........St. Paul
Maeder, Edward C..........Minneapolis
* Maertz, R. W..........Faribault
Maertz, Wm. F..........Anoka
Magath, Thos. B..........Rochester
Magney, Fredolph H..........Duluth
Magnuson, Allen E..........Wheaton
‡ Magnuson, R. C..........Cambridge
‡ Magraw, R. M..........Minneapolis
Mahle, Donald G..........Plainview
Mahowald, Aloys..........Fergus Falls
Maitland, Edwin T..........Jackson
† Maland, Clarence O..........Minneapolis
Malerich, J. A..........St. Paul
Malmstrom, John A..........Virginia
Maloney, William F..........Minneapolis
Mandel, Sheldon L..........Minneapolis
Manger, Wm. M..........Rochester
Mankey, James C..........Minneapolis
Mankin, Harold T..........Rochester
Mann, Frank D..........Rochester
Mann, Joseph D..........Rochester
‡ Manning, Phil R..........Rantoul, Ill.
† Manson, Frank M..........Worthington
March, Kenneth A..........Cambridge
† Marcley, Walter J..........Minneapolis
Marking, George H..........Minneapolis
‡ Markle, G. B., IV....Ft. Monroe, Va.
Marks, Roger W..........St. Paul
Marr, Norval M., Jr..........Rochester
Marshall, C. M..........Crosby
Martens, Theo. G..........Rochester
Martin, Albert C..........Luverne
Martin, Dwight L..........St. Paul
Martin, George B..........Thief River Falls
Martin, Geo. R..........Minneapolis
Martin, Gordon M..........Rochester
Martin, Thos. P..........Arlington
Martin, Webster C..........Duluth
Martin, William B..........Duluth
Martin, William J..........Rochester
Martineau, Jos. L..........St. Paul
Martinson, Carl J..........Wayzata
Martinson, Elmer J..........Wayzata
Masson, Duncan M..........Rochester
† Masson, James C..........Rochester
Masson, James K..........Rochester
*†Matchan, Glenn R...........St. Paul
Mathieson, Don R..........Rochester
Matthews, James H..........Minneapolis
Mattill, Peter M..........Oak Terrace
Mattison, Percy A..........Winona
Mattson, Albert D..........St. James
Mattson, H. A. N..........Minneapolis
Maxeiner, Stanley R., Sr.....Minneapolis
‡ Maxeiner, Stanley R., Jr.......... Ft. Monroe, Va.
† Maxfield, Robert G..........Rochester
Mayne, Roy M..........Nopeming
Mayo, Chas. W..........Rochester
McBean, J. B..........Rochester
McCabe, James S..........St. Paul
McCaffrey, Fabian J..........Minneapolis

McCain, Donovan L..........St. Paul
McCann, Danl. F..........Bemidji
McCann, Eugene J..........Minneapolis
McCannel, Malcolm A..........Minneapolis
McCarran, Samuel P..........Rochester
McCarten, Francis M..........Stillwater
McCarthy, Austin M..........Willmar
McCarthy, Donald..........St. Paul
McCarthy, Jos. J..........St. Paul
* McCarthy, W. R..........St. Paul
McCartney, James S..........Minneapolis
† McCarty, Paul D..........Ely
† McClanahan, J. H.....White Bear Lake
McClanahan, T. S..........White Bear Lake
McCloud, C. N., Jr..........St. Paul
‡ McClure, Rensselaer, Jr.......... Fairmont, W. Va.
McConahey, W. M., Jr..........Rochester
McCormick, D. P..........Minneapolis
† McCoy, Mary K..........Duluth
† McDaniel, Orianna..........Minneapolis
† McDonald, A. L..........Duluth
McDonald, John R..........Rochester
McDonald, Owen G..........Duluth
McDonald, W. J..........Crookston
† McDowell, John P..........St. Cloud
McEnaney, C. T..........Owatonna
McEwan, Alexander..........St. Paul
McFarland, Arthur H..........Minneapolis
McFarlane, John A..........Rochester
McGandy, Robt. F..........Minneapolis
McGeary, Geo. E..........Minneapolis
McGeary, M.D..........Oceanside, Calif.
McGroarty, Brian J..........St. Paul
McGroarty, John J..........Easton
McHaffie, Orval L..........Duluth
McHardy, Bryson R..........Karlstad
McInerny, Maurice W..........Minneapolis
McIntire, Homer M..........Waseca
McIntyre, John A..........Owatonna
*†McIver, Bert A..........Lowry
McKaig, A. M..........Red Lake Falls
McKaig, Carle B..........Pine Island
‡ McKee, Robert E.....Homestead, Penn.
McKelvey, John L..........Minneapolis
McKenna, Elizabeth M..........Austin
McKenna, Jay K..........Austin
McKenna, Maurice J..........Grand Rapids
McKenzie, Chas. H..........Minneapolis
McKenzie, Eva E..........St. Paul
McKinlay, C. A..........Minneapolis
McKinney, Frank S..........Minneapolis
McLane, W. O..........Brainerd
McLaughlin, B. H..........Minneapolis
† McLaughlin, E. M..........Winona
* McLeod, J. L..........Grand Rapids
McManus, William F..........Princeton
McMurtrie, W. B..........Minneapolis
McNear, Geo. R., Jr..........Mankato
McNeil, John J..........Minneapolis
McNeill, J. A..........St. Paul
McNutt, John R..........Duluth
McPheeters, H. O..........Minneapolis
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McQuay, N. W..........Rochester
McWhorter, Henry E..........Rochester
Mead, Chas. H..........Duluth
Meade, John R..........St. Paul
Mears, Burtis J..........St. Paul
Mears, Robert F..........Northfield
Mears, Thomas W..........Rochester
Medelman, John P..........St. Paul
Medoff, Alvin S..........Rochester
Meinert, Albert E..........Winona
Melancon, Jos. F..........St. Paul
† Melby, Benedik..........Blooming Prairie
*†Mellby, O. F..........Thief River Falls
Meller, Robert L..........Minneapolis
† Melzer, G. R..........Lyle
Menges, Chas. G. H..........Rochester
Menold, Wm. F..........St. Paul
Mensheha, Nicholas..........Forest Lake
Mercil, Wm. F..........Crookston
Merkert, Chas. E..........Minneapolis
Merkert, Geo. L..........Minneapolis
Merner, Thomas B..........Faribault
† Merrick, Charlotte T..........St. Paul
Merrick, Robert L..........St. Paul
Merrill, Robt. W..........Morris
Merriman, Lloyd L..........Duluth
Merritt, Wallace A..........Rochester
† Mesker, George H..........Olivia
‡ Metcalf, Norman B..........Princeton
Meyer, Anthony A..........Melrose
Meyer, Alvin J..........Minneapolis
† Meyer, Ette L..........Minneapolis
Meyer, Fredk. C..........Kenyon
Meyer, Paul F..........Faribault
Meyer, Robert J..........Minneapolis
Meyer, Robert P..........Faribault

† Meyerding, Edward A..........St. Paul
† Meyerding, Henry Wm..........Rochester
Michael, Jos. C..........Minneapolis
Michel, Henry H..........Minneapolis
Michels, Roger P..........Willmar
Michelson, Henry E..........Minneapolis
Michienzi, Leonard J..........St. Paul
† Mickelsen, Emma F..........Minneapolis
Mickelson, John C..........Mankato
Midboe, Gilbert T..........St. Paul
Midthune, A. S..........Lake Park
Milhaupt, E. N..........St. Cloud
Millakan, Clark H..........Rochester
Miller, Arder L..........Minneapolis
Miller, Harold E..........Minneapolis
Miller, Herman..........Grand Meadow
Miller, Hugo E..........Minneapolis
Miller, John C..........Minneapolis
‡ Miller, J. M..........Detroit, Mich.
Miller, Roland D..........Rochester
Miller, Ross H..........Rochester
Miller, Samuel..........Albert Lea
† Miller, Victor I..........Mankato
Miller, William J..........Rochester
Miller, W. R..........Red Wing
‡ Miller, W. T..........St. Paul
Miller, Z. R..........St. Paul
Millett, D. K..........Minneapolis
Mills, John L..........Winnebago
Mills, Stephen D..........Rochester
Milnar, Frank J..........St. Paul
Milton, John S..........Minneapolis
Minge, Raymond K..........Worthington
Minsky, Armen A..........Minneapolis
Mintz, Charles M..........Minneapolis
Misbach, Wm. D..........Fairmont
Mishek, Charles J..........St. Paul
Mitby, Irving L..........Minneapolis
Mitchell, Berton D..........Minneapolis
Mitchell, E. C..........Minneapolis
Mitchell, Mancel T..........Minneapolis
‡ Mixer, Harry W..........Ft. Hood, Texas
Moberg, Clarence W..........Detroit Lakes
Moe, John H..........Minneapolis
Moe, Thos..........Moose Lake
Moehn, John T..........Minneapolis
Moehring, Henry G..........Duluth
Moen, J. K., Jr..........Minneapolis
Moersch, Herman J..........Rochester
Moga, John A..........St. Paul
Molander, Herbert A..........St. Paul
Molenaar, Robert E..........Cannon Falls
Mollers, Theo. P..........Soudan
† Monahan, Elizabeth S..........Minneapolis
Monahan, Robert H..........St. Paul
Monroe, Paul B..........Cloquet
Monserud, Nels O..........Cloquet
Monson, Einer M..........Minneapolis
Monson, Leonard J..........Canby
Montgomery, Hamilton..........Rochester
Mooney, Robert P..........St. Paul
Moore, Irvin H..........Minneapolis
† Moores, Kenneth D..........Rochester
Moorhead, Marie..........Minneapolis
Moos, Daniel J..........Minneapolis
† Moquin, Marie A..........St. Paul
† Morehead, D. E..........Owatonna
† Moren, J. Adelaide..........St. Paul
Morgan, Hugh O..........Amboy
Moriarty, Berenice..........St. Paul
Moriarty, Cecile R..........St. Paul
Mork, Arthur H..........Anoka
Mork, Frank E..........Anoka
Morlock, Carl G..........Rochester
Morris, Kenneth L..........Rochester
Morrison, Charlotte J..........Minneapolis
Morse, Morton P..........LeRoy
Morsman, L. W..........Hibbing
Mortensen, J. D..........Rochester
Mortensen, Nels, G..........Minneapolis
Mosby, Maurice E..........Long Prairie
Moses, Royal R..........Kenyon
Mosser, Donn G..........Minneapolis
Mouritsen, Glenn J..........Fergus Falls
Moyer, John B..........Duluth
Moyer, Leonard B..........Minneapolis
Muir, Walter F..........Brown Valley
‡ Mulder, Donald W..........Rochester
Mulholland, Wm. M..........Minneapolis
Muller, A. E..........No. St. Paul
Mulligan, Arthur M..........Brainerd
Mundahl, Harold R..........St. Paul
Munson, Martin S..........Barnum
Murphy, E. P..........Minneapolis
Murphy, Jack T..........St. Paul
Murphy, Joseph E..........Marshall
† Murray, Elizabeth..........Minneapolis
Murray, Robert A..........Hibbing
Musachio, Nicholas F..........Foley
Muske, Marvin..........Minneapolis

Mussey, Mary E. .... Rochester
† Mussey, Robt. D. .... Rochester
Musty, Nicholas J. .... Minneapolis
Myers, Cortland .... Rochester
Myers, Jay A. .... Minneapolis
† Myers, Richard L. .... Rochester
Myers, Thomas T. .... Rochester
Myhre, James A. .... Minneapolis
Myre, Clifford R. .... Paynesville
Myre, Theo. T. .... Rochester

† Naegeli, Frank .... Fergus Falls
Nagel, Harold D. .... Waconia
Nakamura, James Y. .... Deer River
Nash, Leo A. .... St. Paul
Naslund, Ames W. .... Minneapolis
Nauth, Bernard S. .... Minneapolis
Navratil, Donald R. .... Montgomery
Neal, Joe M. .... St. Paul
Nealy, Donald E. .... Adrian
† Neary, Angus J. .... Rochester
Neary, Richard P. .... Minneapolis
Neel, Harry B. .... Albert Lea
Neff, Walter S. .... Virginia
Nehring, Jesse P. .... Preston
Neibergs, Lidija .... St. Paul
Neibergs, Pauls .... St. Paul
Neils, Vernon E. .... Sauk Rapids
Nelson, Bernette G. .... Minneapolis
Nelson, Bernice A. .... Minneapolis
Nelson, Carleton A. .... Worthington
Nelson, C. Barton .... Minneapolis
Nelson, Clayton E. J. .... Albert Lea
Nelson, Edward N. .... Minneapolis
Nelson, Ernest J. .... Lonsdale
Nelson, Glenn E. .... Fairfax
Nelson, Harvey .... Minneapolis
† Nelson, Henry E. .... Crookston
Nelson, Kenneth L. .... Warroad
Nelson, Lloyd S. .... Minneapolis
† Nelson, Louis A. .... St. Paul
Nelson, Maxine O. .... Minneapolis
Nelson, Maynard C. .... Minneapolis
Nelson, Melvin S. .... Granite Falls
† Nelson, Nesmith P. .... Minneapolis
Nelson, O. L. N. .... Minneapolis
Nelson, Robert H. .... International Falls
Nelson, Robert L. .... Duluth
Nelson, Roy A. .... Fergus Falls
Nelson, Wallace I. .... Minneapolis
Nelson, Wilburn O. B. .... Fergus Falls
‡ Nelson, William C. .... Rochester
Nerenberg, Samuel T. .... Minneapolis
Nesbitt, Samuel .... Minneapolis
Nesheim, Martin O. .... Emmons
Nessa, Curtis B. .... St. Cloud
Nesse, J. A. .... Austin
Nesset, Lawren B. .... Minneapolis
Nesset, William D. .... Minneapolis
Neumaier, Arthur .... Glencoe
Neuman, Harold W. .... Rochester
Neumeister, Charles A. .... Minneapolis
Nice, Charles M. .... Minneapolis
Nichols, Donald R. .... Rochester
Nicholson, Murdoch A. .... Duluth
Nicholson, Richard W. .... Heron Lake
Nickerson, John R. .... Fairmont
Nickerson, Neil D. .... Fairmont
Nielson, Alvin M. .... Northfield
Nietfeld, Aloys B. .... Sauk Center
Nilson, Helmer J. .... No. Mankato
Nimlos, Kenneth O. .... St. Paul
† Nimlos, Lenore O. .... St. Paul
Ninneman, Newton N. .... Waconia
Nisius, George .... Duluth
Nixon, James B. .... Crosby
Noble, John F. .... St. Paul
Noble, John L. .... St. Paul
Noonan, William J. .... Minneapolis
Noran, Axel S. N. .... Minneapolis
Noran, Harold H. .... Minneapolis
Norberg, Carl E. .... Cloquet
Nord, Robert E. .... Edina
Nordin, Gustaf T. .... Minneapolis
Nordland, Martin, Sr. .... Minneapolis
Nordland, Martin, Jr. .... Minneapolis
Nordman, Willard F. .... Mora
Norman, John F. .... Crookston
Normann, Stephen T., Jr. .... Waseca
Norris, Neil T. .... Caledonia
Norum, Henry A. .... Minneapolis
Norval, Mildred A. .... Minneapolis
† Noth, Henry W. .... Minneapolis
† Novak, Edward E. .... New Prague
Nuessle, Walter G. .... Springfield
Nuetzman, Arthur W. .... Fairbault
Nutting, Roland E. .... Duluth
Nydahl, Malvin J. .... Minneapolis
Nye, Katherine A. .... St. Paul
Nye, Lillian L. .... St. Paul
Nygren, Wm. T. .... Braham
Nylander, E. G. .... Minneapolis
Nywall, Dean D. .... Slayton

O'Brien, J. C. .... St. Paul
O'Brien, Louis T. .... Breckenridge
† O'Connor, Danl. C. .... Eden Valley
† O'Connor, Loren J. .... St. Paul
O'Donnell, D. M. .... Ortonville
O'Donnell, James E. .... Minneapolis
O'Hanlon, John A. .... Minneapolis
O'Kane, Thomas W. .... St. Paul
O'Keefe, James P. .... St. Cloud
‡ O'Keefe, Mathew .... Rochester
† O'Leary, John B. .... Minneapolis
O'Leary, J. H. .... Thief River Falls
O'Leary, Paul A. .... Rochester
‡ O'Malley, Valentine .... St. Paul
O'Neil, Richard L. .... Rochester
O'Neill, John C. .... Duluth
O'Phelan, E. Harvey .... Minneapolis
O'Reilley, Bernard E. .... St. Paul
‡ O'Shaughnessy, E. J. .... Camp Kilmer, N. J.
† Oberg, Carl M. .... Minneapolis
Ochsner, Clarence G. .... Wabasha
Ockuly, Orville .... St. Paul
Odel, Howard M. .... Rochester
Odland, Mark E. .... Detroit Lakes
Odland, Olin .... Granite Falls
Oeljen, S. C. G. .... Waseca
Ogburn, Paul L. .... Rochester
Ogden, Warner .... St. Paul
Ohage, Justus .... St. Paul
Olavs, Olga .... Minneapolis
Olds, Geo. H. .... New Richland
‡ Olive, John T., Jr. .... Ft. Riley, Kan.
Oliver, Irwin L. .... Graceville
Oliver, James .... Moorhead
Olmanson, Edmund G. .... St. Peter
Olsen, Arthur M. .... Rochester
Olsen, Edward G. .... Minneapolis
Olsen, Ralph L. .... St. Paul
Olson, Albert J. .... Owatonna
Olson, Alton C. .... Minneapolis
Olson, Archibald O. .... Duluth
Olson, Carl J. .... Minneapolis
Olson, C. Kent .... Minneapolis
Olson, Charles A. .... St. Paul
Olson, Chester J. .... Belle Plaine
Olson, Detlof, M. .... Minneapolis
Olson, Duane O. C. .... Gaylord
Olson, Ernest A. .... Pine Island
Olson, Grant E. .... West Concord
Olson, Lillian A. .... Ah-Gwah-Ching
† Olson, Olof A. .... Minneapolis
Onsgard, L. Kenneth .... Houston
‡ Openshaw, Calvin R. .... Salt Lake City, Utah
Oppegaard, Chester L. .... Crookston
Oppen, E. Gerhard .... Minneapolis
Oppen, M. G. .... Wayzata
Opsahl, Lawrence J. .... Willmar
Opstad, Earl T. .... Oak Terrace
Orr, Burton A. .... Faribault
‡ Orwoll, Harold S. .... Japan
Osborn, Donald O. .... Austin
Osborn, John E. .... Rochester
Ostergaard, Erling .... Evansville
Ostergren, Edward W. .... St. Paul
Ostling, Burton C. .... Kerkhoven
Otto, Henry C. .... Frazee
Ouellette, Alfred J. .... St. Paul
Ourada, Anthony L. .... Ceylon
Owen, Charles A., Jr. .... Rochester
Owens, F. M., Jr. .... St. Paul
Owens, William A. .... Montevideo

† Paciotti, V. J. .... Minneapolis
Page, Raymond L. .... St. Charles
Palen, Benjamin J. .... Minneapolis
Palmer, Clinton F. .... Albert Lea
Palmer, Harry A. .... Blackduck
Palmerton, E. S. .... Fergus Falls
Pankratz, Peter J. .... Mountain Lake
Papermaster, Ralph .... Two Harbors
Papermaster, Theo. C. .... Minneapolis
Park, Wilford E. .... Minneapolis
Parker, Charles W. .... Wadena
Parker, Harry L. .... Rochester
† Parker, O. W. .... Minneapolis
Parker, Philip J. .... Thief River Falls
Parker, Robert L. .... Rochester
Parker, Wilbert H. .... Chisholm
Parkhill, Edith M. .... Rochester
Parson, E. J. .... Duluth
Parson, E. Lillian B. .... Elbow Lake
Parson, Lester R. .... Elbow Lake
Parsons, Ralph L. .... Monterey
‡ Parsons, W. B., Jr. .... San Francisco, Calif.
Pasek, Antone W. .... Cloquet
Patch, Orien B. .... Duluth
Patrick, Robert T. .... Worrington, Fla.
Patterson, Hugh D. .... Slayton
† Patterson, W. L. .... Fergus Falls
Paulson, John A. .... Rochester
† Paulson, Theo S. .... Fergus Falls
Paulson, Wallace .... St. Paul
Paynter, Camen R. .... Chicago, Ill.
† Pearsall, R. P. .... Virginia
Pearson, Bror F. .... Shakopee
Pearson, Fritz R. .... St. Paul
Pearson, Malcolm M. .... St. Paul
† Pearson, Roy T. .... Shakopee
Pedersen, Arthur H. .... St. Paul
Pedersen, Robert L. .... Brainerd
Pedersen, Roy C. .... Duluth
Peltier, Leonard F. .... Minneapolis
‡ Peluso, Chas. R. .... Phoenix, Ariz.
‡ Pemberton, A. H. .... San Francisco, Calif.
† Pemberton, John .... Rochester
Pender, John W. .... Rochester
† Penhall, Fletcher W. .... Willmar
Penk, Engward L. .... Springfield
Penn, George E. .... Mankato
Pennie, Danl. F. V. .... Duluth
Peppard, Thos. A. .... Minneapolis
Perkins, Douglass .... Alexandria
Perlman, Everett C. .... Minneapolis
Perlman, Herschel L. .... Minneapolis
Perry, Harold .... Rochester
Person, John P. .... Albert Lea
Person, Waldemar N. .... Rochester
Pertl, Albert L. .... Canby
Peteler, Jennings C. .... Minneapolis
Peters, Gustavus A. .... Rochester
† Peters, Myra A. .... Rochester
Petersen, Arthur B. .... Rochester
Petersen, D. H. .... Northfield
Petersen, Glenn L. .... Minneapolis
† Petersen, John R. .... Minneapolis
Petersen, Magnus C. .... Rochester
Petersen, Peter C. .... Minneapolis
Petersen, Robert T. .... St. Cloud
Petersen, William E. .... Minneapolis
Peterson, David B. .... St. Paul
Peterson, Donald H. .... St. Paul
Peterson, Edward N. .... Virginia
Peterson, Harold O. .... St. Paul
Peterson, Herbert W. .... Minneapolis
Peterson, Joel L. E. .... St. Paul
Peterson, John H. .... Duluth
Peterson, Kenneth A. .... Marshall
Peterson, Kenneth H. .... Hutchinson
Peterson, Nordahl P. .... Minneapolis
Peterson, Oliver H., Sr. .... Minneapolis
† Peterson, Oliver H., Jr. .... Minneapolis
Peterson, Palmer A. .... Minneapolis
Peterson, Peter E. .... Minneapolis
Peterson, Roy A. .... Vesta
Peterson, Roy L. .... White Bear Lake
Peterson, Stanley C. .... Austin
Peterson, Willard C. .... Minneapolis
Peterson, Willard E. .... Willmar
Peterson, W. H. .... Spring Valley
Peterson, Henry .... Minneapolis
Petit, Julien V. .... Minneapolis
Petit, L. J. .... Minneapolis
Petraborg, Harvey T. .... Aitkin
Pettersen, George R. .... Mabel
Pettet, John .... Rochester
Pewters, John T. .... Minneapolis
Peyton, Wm. T. .... Minneapolis
Phares, Otto C. .... St. Cloud
Phelps, Kenneth A. .... Minneapolis
Phillips, John W. .... Rochester
‡ Philp, David R. .... Minneapolis
Pierce, Chas. H. .... Wadena
Pierce, Jack R. .... Virginia
Pierce, Robert B. .... Renville
Pierson, Roy F. .... Slayton
† Piper, Monte C. .... LaCanada, Calif.
Piper, Wm. A. .... Mountain Lake
† Place, Virgil A. .... Rochester
Plasha, Matthew K. .... Anoka
Plass, Herbert F. R. .... Minneapolis
† Platou, Erling S. .... Minneapolis
Plimpton, N. C., Jr. .... Minneapolis
† Plondke, Fred J. .... St. Paul
Plotke, Harry L. .... St. Paul
Plucker, Milton W. .... Clarkfield
Pogue, Richard E. .... Watertown
Pohl, John F. M. .... Minneapolis
† Poirier, Jos. A. .... Forest Lake
Polley, Howard F. .... Rochester
† Pollock, David K. .... Minneapolis
Polski, Paul G. .... So. St. Paul
Polzak, Jacob A. .... Minneapolis

Pone, John....Silver Lake
Ponterio, James E....Shakopee
Pool, Thomas L....Rochester
† Poppe, Fredk. H....Minneapolis
Potek, David....Minneapolis
Potter, R. B....Minneapolis
Potthoff, Carl J....Rochester
Powelson, Myron H....Rochester
Power, John E., Sr....Duluth
Power, John E., Jr....Duluth
*†Powers, Fred H....Rochester
* Prangen, A. DeH....Rochester
† Pratt, F. J., Sr....Minneapolis
‡ Pratt, F. J., Jr....New York, N. Y.
‡ Pratt, George F....San Francisco, Calif.
Pratt, J. H., Jr....Rochester
Preine, Irving A....Osseo
† Preisinger, Jos. W....Dallas, Texas
Preston, Paul J....Minneapolis
Prickman, Louis E....Rochester
Priest, Robt. E....Minneapolis
Priestley, James T....Rochester
† Prim, Jos. A....Minneapolis
Prins, Leo R....Albert Lea
Proeschel, Ray K....Willmar
Proffitt, William E....Minneapolis
Proshek, Chas. E....Minneapolis
Pruitt, Raymond D....Rochester
Pugh, David G....Rochester
Pumala, Erven E....Warren
Purves, George H....Hendricks
Puumala, Reino H....Cloquet

Quanstrom, V. E....Brainerd
Quattlebaum, Frank....St. Paul
Quello, Robt. O. B....Minneapolis
Quer, Erich A....Kansas City, Mo.
† Quigley, Walter P....Brooklyn, N. Y.
† Quist, Henry W., Sr....Minneapolis
Quist, Henry W., Jr....Minneapolis

Raadquist, Chas. S....Hibbing
Raattama, John....Keewatin
Rabens, Robert A....Rochester
Racer, Harley J....Stillwater
Raetz, Sylvester J....Maple Lake
Rajala, Arnold I....Grand Rapids
Ralph, James R....St. Paul
Ralston, Donald E....Rochester
† Ramsey, Walter R....St. Paul
* Randall, A. M....Ashby
Randall, Lawrence M....Rochester
Randall, Raymond V....Rochester
Ransom, H. R....Osseo
† Ransom, Matthias L....Hancock
Rasmussen, Ramby C....St. Paul
Rasmussen, W. C....Rochester
Ravits, Harold G....St. Paul
Rayner, Ralph R....Gibbon
Rea, Chas. E....St. Paul
Reader, Donald R....Minneapolis
Reed, Paul....Virginia
* Reeve, E. T....Elbow Lake
Reff, Alan R....Crookston
Regnier, Edw. A....Minneapolis
Reid, James W....St. Paul
Reid, Lewis M....Minneapolis
Reif, Harold A....Minneapolis
Reif, Henry J....St. Cloud
Reiley, Richard E....Minneapolis
† Reineke, George F....New Ulm
Reitemeier, Richard J....Rochester
Reitmann, John H....Anoka
ReMine, W. H., Jr....Rochester
Remole, William D....Minneapolis
Remsberg, R. R....Tracy
† Replogle, Wm. H....Los Angeles, Calif.
Resch, Jos. A....Minneapolis
Rice, Carl O....Minneapolis
Rice, Frank B....Minneapolis
Rice, Fred A....Minneapolis
Rice, Hagbart G....Moorhead
Rice, Roberta G....Grand Island, Neb.
Richards, Ernest T. F....St. Paul
Richards, Wm. B....St. Cloud
Richardson, Edward J., Jr....St. Paul
Richardson, Robert J....St. Paul
Richdorf, Lawrence F....Minneapolis
Rick, Paul F. W....St. Paul
Ridley, Roger W....Rochester
Rieke, Wellington W....Wayzata
Rieschl, Elizabeth K....Jordan
Rigler, Leo G....Minneapolis
Rigler, R. G....Rochester
Ringle, Otto F....Walker
Rinkey, Eugene....St. Paul
Riordan, Elsie M....Minneapolis
‡ Ripepi, James D....New York, N. Y.
Risch, Ronald E....Minneapolis
Risser, Alden F....Stewartville
Ritchie, Wallace P....St. Paul
Ritt, Albert E....St. Paul
† Ritzinger, Fredk. R....Lakefield
Rizer, Dean K....Minneapolis
Rizer, Robt. I....Minneapolis
Robb, Edwin F....Minneapolis
† Robbins, Chas. P....Winona
Robbins, Owen F....Minneapolis
Roberts, Byron H....Minneapolis
Roberts, Lewis J....Minneapolis
Roberts, Oliver W....Owatonna
Roberts, Stanley W....Minneapolis
† Roberts, W. B....Minneapolis
*†Robertson, J. B....Minneapolis
Robertson, Paul A....Austin
Robilliard, Chas. M....Faribault
Robinett, Robert W....Worthington
Robinson, C. O....Minneapolis
Rocknem, Robert E....Minneapolis
Rockwell, C. V....Minneapolis
Rockwood, Philo H....Fergus Falls
† Rodda, Fredk. C....Minneapolis
Rodgers, Richard S....Minneapolis
Roehlke, Arthur B....Elk River
Roemer, Henry J....Winona
Rogers, Arnold G....Rochester
Rogers, Chas. W....Winona
Rogers, Sidney F....St. Paul
† Rogin, Norton....St. Louis Park
Rogne, Wm. G....Spring Grove
Roholt, Christian L....McIntosh
‡ Roholt, H. B....Fosston
† Rohrer, Christian....Des Moines, Iowa
Rokala, Henry E....Virginia
Rolig, David H....St. Paul
Rollie, O. O....St. Paul
Rollins, Pat....St. Charles
‡ Rollins, Troy G....Hoopa, Calif.
Rome, Howard P....Rochester
Romness, Joseph O. Schenectady, N. Y.
‡ Romness, Kenneth....Mound
† Rood, D. C....Santa Barbara, Calif.
Rooke, E. D....Rochester
Rorem, Jos. A....Appleton
† Rosander, Phyllis....Minneapolis
Rose, John T....Lakefield
Rosenbaum, David L....Minneapolis
Rosendahl, Fredk. G....Minneapolis
Rosenfield, Abraham B....Minneapolis
Rosenow, John H....Minneapolis
Rosenthal, F. Harold....Austin
Rosenthal, Robert....St. Paul
† Rosenwald, Reuben M....Anoka
Ross, Alexander J....Minneapolis
Rossberg, Raymond A....Morris
Rossen, Ralph....Hastings
Roth, Fredk. D....Mankato
Roth, George C....St. Paul
Rothnem, Morris....Minneapolis
† Rothschild, Harold J....St. Paul
Rothwell, Walter S....Rochester
Roust, Henry A....Montevideo
Rovelstad, R. A....Rochester
Rowe, Olin W....Duluth
Rowe, William H....Fairmont
Rowles, Everett K....Coleraine
Roy, Phil C....St. Paul
Rozycki, Anthony....Pine River
Rucker, Chas. W....Rochester
Rucker, Wm. H....Minneapolis
Rud, Norman E....Minneapolis
Rudell, Gustave....Minneapolis
Rudie, Clifford N....St. Peter
Rudie, Peter S....Duluth
Ruggles, Geo. M....Forest Lake
† Ruhberg, Geo. M....Tarzona, Calif.
Rumpf, Carl W....Faribault
Runquist, John M....Duluth
Rushton, Joseph G....Rochester
Russ, Homer H....Blue Earth
Russeth, Arthur N....Minneapolis
Rusten, Elmer M....Minneapolis
Rusterholz, Alan P....St. Paul
Rutledge, Lloyd H....Detroit Lakes
Ryan, John J....St. Paul
Ryan, Jos. M....St. Paul
Ryan, Wm. J....Duluth
Rydburg, Wayne C....Minneapolis
‡ Rydland, Arne D....Foley, Ala.
Rygh, Harold N....Atwater
Rynda, Edwin R....New Prague
Rynearson, Edward H....Rochester
Rysgaard, George M....Northfield

St. Cyr, Harry M., Jr....Robbinsdale
St. Cyr, Kenneth J....Robbinsdale
Sach-Rowitz, Alvan....Moose Lake
Sadler, Wm. P., Jr....Minneapolis
Saffert, Cornelius A....New Ulm
Sahr, Walter G....Hutchinson
Salassa, Robert M....Rochester
Saliterman, Bernard I....Minneapolis
‡ Salk, Richard J....Sauk Rapids
Salter, Reginald A....Virginia
Samuelson, L. G....Mankato
Samuelson, Samuel....Minneapolis
Sandeen, Robert M....Buffalo
Sandell, Samuel T....Nopeming
Sanderson, A. G....Anoka
Sanderson, David J....Fergus Falls
Sandt, Karl E....Minneapolis
Sandven, Nels O....Paynesville
† Sanford, Arthur H....Rochester
† Sanford, James A....Savage
† Sanford, John B....Duluth
Sanford, Raymond A....Minneapolis
Sarff, Oliver E....Duluth
Sargent, Edward C., Jr....Austin
Sarnecki, M. M....St. Paul
† Satersmoen, Theo....Pelican Rapids
Sather, Edgar L....Fosston
Sather, Edgar R....Alexandria
Sather, George A....Fosston
Sather, Richard N....Fosston
Sather, Russell O....Crookston
Satterlee, Howard W....Lewiston
† Satterlund, Victor L....St. Paul
Sauer, William G....Rochester
† Savage, Francis J....St. Paul
Sawatzky, W. A....Minneapolis
Sawtell, Robert R....Worthington
Sax, Milton H....Duluth
Sax, Simon G....Duluth
Saxman, Gertrude O....Georgetown
Sayre, George P....Rochester
Sborov, A. M....Minneapolis
Scanlon, Paul W....Rochester
Schaaf, F. H. K....Minneapolis
Schaar, Frances E....Minneapolis
Schade, Fredk. L....Worthington
Schaefer, Jos. F....Owatonna
Schaefer, Wesley G....Minneapolis
Schamber, Walter F....Parkers Prairie
Schatz, Francis J....St. Cloud
Scheidel, A. McK....Rochester
Scheifley, Charles H....Rochester
† Scheldrup, N. H....Minneapolis
Scherer, Leslie R....Minneapolis
Scherling, Sidney S....Minneapolis
Schiele, Burtrum C....Minneapolis
Schimelpfenig, George....Chaska
Schirber, Martin J....Grand Rapids
Schissel, Gregory A....Minneapolis
* Schlesselman, G. H....Minneapolis
Schmid, John F....Duluth
Schmidt, Herbert W....Rochester
Schmidt, Hilmar R....Winona
† Schmidt, P. A....Aurora, Ill.
Schmidt, P. G., Jr....Granite Falls
Schmidt, Ruben F....Alden
Schmidt, W. R....Minneapolis
Schmidtke, R. L....St. Paul
† Schmitt, S. C....El Cajon, Calif.
Schmitz, Anthony A....Mankato
† Schmitz, Everett J....Seattle, Wash.
Schmitz, Glenn P....Little Falls
Schneck, Jack I....Waukesha, Wisc.
Schneider, Lawrence E....Duluth
Schneider, Paul J....Adams
Schnell, Fredk. S....Litchfield
† Schoch, Robert B. J....St. Paul
Schoeneberger, P. B....Perham
† Scholpp, Otto W....Hutchinson
Schons, Edward....St. Paul
Schottler, Max E....Minneapolis
† Schroder, Chas. H....Pottstown, Pa.
Schroeckenstein, H. F....St. Paul
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Van Herik, Martin	Rochester
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PROBLEMS AND OBLIGATIONS OF ORGANIZED MEDICINE

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I HAVE BEEN asked to discuss some aspects of organized medicine, particularly its problems, its obligations and its relationship to teaching institutions. I suspect that I have been asked to do this because my background should enable me in some measure to bridge the gap in point of view between those in private practice and those who are full-time teachers. While this task has not been easy, I have found it a pleasure because it has offered an opportunity to express some deep convictions.

The future of medicine, I believe, is inseparably bound to a wise, healthy, farseeing, over-all organization which considers the rights and interests of all branches of medicine and which is at once receptive to change, resistant to exploitation, a stimulant to scientific progress and the better utilization of that progress, a monitor of the competency and behavior of its membership, a trusted and respected interpreter of medical thought to the public and all this for the betterment of medical care. The realization of this is a larger order which we have not yet fully achieved but it is a goal toward which we must direct our thoughts and energies.

I am sure that the great majority of us believes in a type of medical practice which is compatible with a free enterprise system. I am sure that the great majority is friendly to the American Medical Association which, as you know, is only the aggregate of all state associations which in turn are the aggregate of all county associations. Trained as you are to challenge existing practices and ideas in the hope of improving them, it would not be unusual if a few entertained ideas which are quite at variance with the views of most of us. If there are such, they are considerate to the wishes of the majority and reasonable enough to know that were they in different positions, active practice for example, they might think differently.

I would like to express some of my own ideas concerning our problems and obligations as a medical organization, which ideas I have reason to believe are not too far removed from those held by the many able men who are giving their time, thought and energies to medicine's needs, who can visualize our goal, and who hold no illusions regarding the pitfalls and road blocks to be overcome.

Our most serious problem, and I should add "in my opinion," for I have rarely heard this thought expressed, is to resist the forces which threaten to divide us. Our recent struggle to escape compulsory health insurance, which was actually a closer call than many realize, indicates what the socializers would do to the practice of medicine were we to become split into factions.

Segmentation and division along lines of special interests assume a constantly increasing menace. Specialization in which whole groups with little knowledge of, or interest in, the problems of other groups fosters the illusion of self-sufficiency.

The general practitioners, who make up well over one-half of all practicing physicians and without whom we could not, for the foreseeable future, serve the American public, create a special problem. Today, the general practitioner feels threatened, suffers from loss of prestige and a sense of insecurity. Especially this is true in the cities where he is in competition with the specialist

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and where everything he does is measured in terms of specialist performance. In rural areas, he fares better and in most cases measures up to the finest traditions of "the family doctor."

Today the general practitioner has his own organization and operates it from the praiseworthy standpoint of education of its membership and from the understandable viewpoint of self-protection. How tragic for medicine if the general practitioners and specialists would engage in a bitter competition for public favor!

There is urgent need today for organized medicine to summon all of its divergent elements and to define and spell out a reasonable relationship, especially between general practitioners and specialists.

To illustrate, the American College of Surgeons has declared it a breach of ethics for a surgeon to use a referring general practitioner as an assistant. Granting that on occasions such assistance may be but a subterfuge for a split fee and, granting that the general practitioner has no vested interest in his patient, he, nevertheless, often by being the family doctor, holds a trusteeship toward his patients' health, which would be poorly served were he forced off the case when surgery is involved. Most of us who are members and strong supporters of the College believe this to be wrong, believe it will discourage requests for consultation and reference of cases and will serve to isolate general practitioners into a resentful and dissident group.

The principle which I wish to emphasize is that questions as basic as that of ethics and that of all-important relationships is a matter to be settled by organized medicine as a whole and not by a special group, no matter how sincere the motives or how great the qualifications to advise.

Thinking further along this line, there is urgent need for organized medicine to gather its resources and courage and to clean up unwholesome and immoral practices within the confines of its own doors. It is, in my opinion, to our everlasting discredit that we did not stop rebates for optical goods and all other forms of rebates until forced to do so by governmental action.

The American College of Surgeons has undertaken a crusade against fee-splitting, ghost surgery, and excessive fees, all reprehensible practices which should have been eliminated long ago. It should not have been necessary for the college to do this, and it is particularly unfortunate that they should have had so little confidence in the help that organized medicine would give them that they took their crusade out of medicine's lap and put it before the public. Again, most of us who will be glad to support any campaign to eradicate such practices, believe that the first step should have been to seek an agreement regarding a definition of fee splitting. All branches of medicine should have participated in reaching such an agreement. After full agreement, the whole-hearted co-operation of the American Medical Association should have been sought and should have been given.

Articles such as appeared in a recent issue of Colliers might not then be so disturbing to the public, so damaging to the confidence we seek to inspire, and so misleading about the extent of the evils described. The article did, indeed, admit that the majority of practitioners do not indulge in such misconduct, but it says so in fine print, figuratively speaking.

Closely allied to the eradication of gross and blatant evils is medicine's obligation to examine and to pass judgment upon the quality of service rendered by its members. Up to this time, it has been difficult if not impossible to do this. It has always been easier to avoid accepting a man into organized medicine if there is reason to doubt his worthiness than to extrude him after he has become a member. The courts have made it so difficult to discipline a member that his offense must be great before he can be expelled. Court action which is pending in Minnesota may be helpful in defining our rights in legal terms.

As many of you know, the Two Harbors Co-operative medical group is suing certain members of the St. Louis County Medical Society for the large sum of over $300,000, claiming conspiracy. Out of this suit, it is to be hoped the rights of medicine will be so clearly established that we may be able to limit our organization to men who foster the type of medicine in which we believe and who maintain the highest ethical and scientific standards.

A study of the problem of monitoring the quality and excellence of practice of the individual member leads inevitably to the conclusion that the place to regulate is at the hospital-staff level. I believe that the recently promulgated rules applying to staff organization and staff obligations which have been issued by the joint Accrediting Committee and which, as you know, require a critical review of every patient admitted to a hospital, clearly indicate such a viewpoint. These edicts are creating a furor, not only because of the added time and responsibility they demand, but more importantly because they require an adjustment in our thinking. Accustomed to freedom of thought and action in the practice of medicine, it is hard for us to accept the idea of supervision by our confreres. However, it is, in my opinion, an important move toward improvement of the quality of service which the public receives. With certain necessary modifications, some of the inconveniences may be eased. The hot breath of censure will most certainly put wings on the feet of the laggard and fear in the heart of the dishonest.

Organized medicine is very much opposed to federal aid to the medical schools. Perhaps some of you may not agree with this position, but you will respect the motives. We feel that support will eventually lead to control, and we believe it to be to the best interests of medicine and of the public to keep our medical schools free of governmental dictation or control of any kind or degree.

The American Medical Association, through a foundation established for the purpose, is attempting to raise substantial funds from its own membership to aid medical colleges. They have fostered other foundations and agencies also seeking funds for our schools. Admittedly, at this time, the moneys which have been raised fall short of the needs, but with more time the accomplishment will be greater. This country which raises so much money for privately endowed colleges will not desert its medical schools.

To stay strong, medicine must remain sensitive and responsive to public needs and demands. Never again will we withdraw from and neglect our public relations. Review but briefly our response to the public's reasonable demand: All over the country, implementing the request of the American Medical Association, county medical societies have established grievance committees to which any person who feels that he has been misused by a member of our profession may bring his complaint and receive satisfaction through explanation or redress.

We have made it possible for anyone to secure a physician within a reasonable time. Different mechanisms operating in various ways are employed over the whole country to assure availability of medical care to the public.

We have recognized the public's complaint of high costs of medical care, and have fostered and promoted the sale of voluntary prepayment forms of insurance, such as the Blue Shield plans, and also the direct and indirect encouragement of the sale of commercial contracts covering surgical and medical expenses.

Today, according to testimony of major insurance companies at hearings before a House Committee, 91,000,000 men, women, and children are covered by hospital insurance, 73,000,000 have surgical-expense coverage, and 36,000,000 are protected against medical expense.

Policies covering catastrophic illness are making their appearance. Two years ago California initiated this in conjunction with their standard Blue Shield policies; others, including private insurance companies, are following. Edmund B. Whittaker, vice-president of Prudential Insurance Company, estimates that already one million people are covered by this form of policy.

The voluntary insurance method of attacking the problem of high costs will never satisfy those who believe that people have a divine right to good health without personal responsibilities for it. It is their contention that under a voluntary prepayment insurance system, man cannot afford his medical care along with food, clothing, shelter and recreation. They completely abandon the spirit of independence, self reliance and free enterprise in favor of governmental paternalism, socialism or some of the other "isms."

Furthermore, I believe that, to earn the respect to which it is entitled, organized medicine has the duty of fostering within its membership a stronger sense of obligation toward citizenship and toward civic responsibility. Perhaps it is because our old tradition taught us to be self-effacing as far as the public is concerned that doctors feel detached from civic activities. Regardless of the cause, it is harmful to us to shirk either the financial demands of our communities or the demands they may make upon our time and energies. When other groups are doing a good job, can we afford to do otherwise?

In the minds of many, medicine should shun politics. What is politics but the business of operating our civilization? We can no more shun politics than we can waste our vote by boycotting the polls.

In this phase, I have followed a rather philosophical, and, some may believe, unrealistic approach. I am quite ready to admit that organized medicine has made many mistakes and will unfortunately make more. I would not have you believe that I consider our need for unity transcends the right of any individual or any group to disagree, to debate and to dissent. You have only to attend a meeting of the House of Delegates to hear very vocal dissension. I ask only that these differences and debates be held within our own portals, and that when the vote is cast the will of the majority shall not only prevail but be freely adopted.

I wish to reiterate my belief that medicine shares in the unrest and the uncertainty afflicting this country and the entire world, that medicine as we would have it is still in danger and that, just as the free world needs a strong America, so free medicine needs a strong American Medical Association.

I would rather skip the next phase but to do so would be to shun an obligation to help smooth out a totally unnecessary feeling of unfriendliness which seems to have crept between our medical school and some of the doctors in our state. It is hard to evaluate this feeling, its origin, its extent or its intensity, but perhaps it is only necessary to know that it exists in order to move to correct it.

Last year, Dr. Charles Sheppard of Hutchinson, speaker of the House of Delegates of the Minnesota State Medical Association, ably represented the point of view of the rural general practitioner and the views he expressed must be considered as held by many of the men in rural practice. The faculty may believe that many of the direct and implied criticisms were unjust, some of the suggestions impractical or outweighed by other considerations. Nevertheless, right or wrong, just or unjust, it was good that he expressed his views.

I am somewhat mollified to think that at the state level our relationship with organized medicine has been excellent. Has not Dr. H. S. Diehl been a faithful attendant at council meetings, given hours of study on important committee assignments and has he not done as much or more on a National level? As a matter of fact, many of our members, including some heads of departments, have worked for organized medicine: Drs. Howard, Spink, Keys, Bieter, Wangensteen, McQuarrie, to mention only a few of them.

Finally, I reflect on the fact that our school cannot grow greater in an atmosphere of professional suspicion and disapproval. Especially this is true when the financial support comes for the most part from taxes appropriated by the state legislature, and our clinical material from the bailiwicks of both friends and critics. I now, therefore, try to evaluate what part of our critics' complaints is valid and how much of it is based on misinformation or misunderstanding.

Let me revert to my role as friendly critic and advisor and attempt to assay the situation in the hope that there may be an easy correction to whatever valid basis of ill feeling may be found to exist.

I believe that the medical school may feel most secure in its relations with the specialists in Minnesota. Within this group criticism and ill feeling are least likely to exist; rather the faculty is held in highest esteem and is called upon frequently for consultation and advice.

Within the Twin Cities, however, there is a tiny fear, a smoldering apprehension, that in years to come, with its source of teaching material from rural areas drying up, a gradual emphasis in private practice among the faculty will result in a large private clinic maintained by taxes and in competition with the private physician.

Let me hasten to add that I am not aware of antagonism to private practice on the part of full-time men when done on a limited basis. The doctors, proud of and ambitious for their medical school, know that we cannot keep good men as heads of departments if they are limited to the same remuneration as a professor of Romance Languages. It is true that those of us who lose private patients to the University actually contribute more to the support of the medical school than do laymen who merely pay taxes. We do not object to the situation as it now exists, but would certainly not willingly accept the presence of a large private clinic. But why worry about something that does not exist?

There is far less ill-feeling among general practitioners in cities than in urban areas. In the matter of sampling, I have found very little antagonism. A general practitioner friend of mine who is very active in the Academy of General

Practice in Hennepin County told me only a week ago that since Dr. Howard had assumed his position, relations between the general practitioner of his area and the medical school had improved greatly.

We come finally to the rural general practitioner, where our real problems lie. These men have been for years feeders of the University Hospital. These are the men who have been most seriously handicapped and frustrated by failure to obtain assistants and replacements in their areas. These men also are allergic to the attractions of specialization for the younger men. Happy in their work and in their positions in their communities, they cannot understand why new graduates in medicine should hesitate to enter general practice in the "country." To add to their frustration, they cannot obtain nurses or even the right to train the nurses needed to operate at their hospitals. This may explain why at times they may appear unreasonable.

I am convinced that our medical faculty does nothing deliberately to discourage our graduates from becoming general practitioners. Our offense, if there is an offense, is one of omission and not commission. In other words, the only question is as to whether we as a faculty are doing all we can to encourage graduates up to the point where the needs of the state are met. After that we have no obligation to influence. Some of us may feel we have no right to attempt to influence.

I believe that we can obtain enough information pertaining to student motivation to clear us of any suspicion of influencing the student toward specialization. I believe also that such information should be gathered, analyzed, and transmitted to the medical profession of the state. For the sake of harmony, we should defend ourselves vigorously in this respect.

I do not believe that medicine would want its medical schools staffed exclusively by teachers whose sole aim and qualification is to turn out good practitioners of medicine. It is good that some teachers find their greatest satisfaction in the superb training of men who are thereby made capable of supplanting or excelling them and that through this and through research they make their greatest contributions to medicine and mankind. We ask only that they not neglect the requirements of providing the needed practicing physicians for the people whose taxes support our school.

We come now to what I consider the only justification for the resentful attitude of these men. Over a period of years in which they have been sending their low income or indigent patients to the University Hospital, a number of irritating incidents have occurred either to them individually or to a confrere whose experience they embrace as their own. A report is not received. It is very tardy. It is sketchy. It is enormously detailed but not summarized. A patient referred to the hospital is sent back without treatment and without explanation. The patient hears or thinks he has heard remarks criticizing the referring doctor. These and a host of other complaints are offered by the rural doctor to explain his reluctance to send his patient to the University Hospital. Add to this the fact that local commissioners charged with the financial responsibilities of the patient's care have found that it is less expensive to use the new hospital in the community than to use the University Hospital, and the patient would rather stay home within easy reach of his family. You will then understand why clinical material is becoming harder to get.

May I offer a suggestion? Complaints on the part of referring doctors are far less common when the patient is a private patient referred to a specific member of the staff. This is because the staff man immediately establishes a consultant relationship with the referring doctor.

Why not the same attitude toward every patient and every referring doctor? Let the code of ethics of the American Medical Association pertaining to consultations and referrals apply in these cases insofar as it is possible to apply it. This simple adjustment in thinking and the mechanics to operate it are in my opinion all that is needed to start our hospital and staff toward a fine relationship with the doctors of the state who will then see that the medical school does not suffer for want of clinical material.

One other suggestion which I have already made to Dr. Diehl: the University Hospital and the Medical School should have a professional relations man, who will visit the county medical societies, and even the doctors' offices to acquaint the

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NEUROGENETIC INHIBITORS FOR FUNCTIONAL DISORDERS

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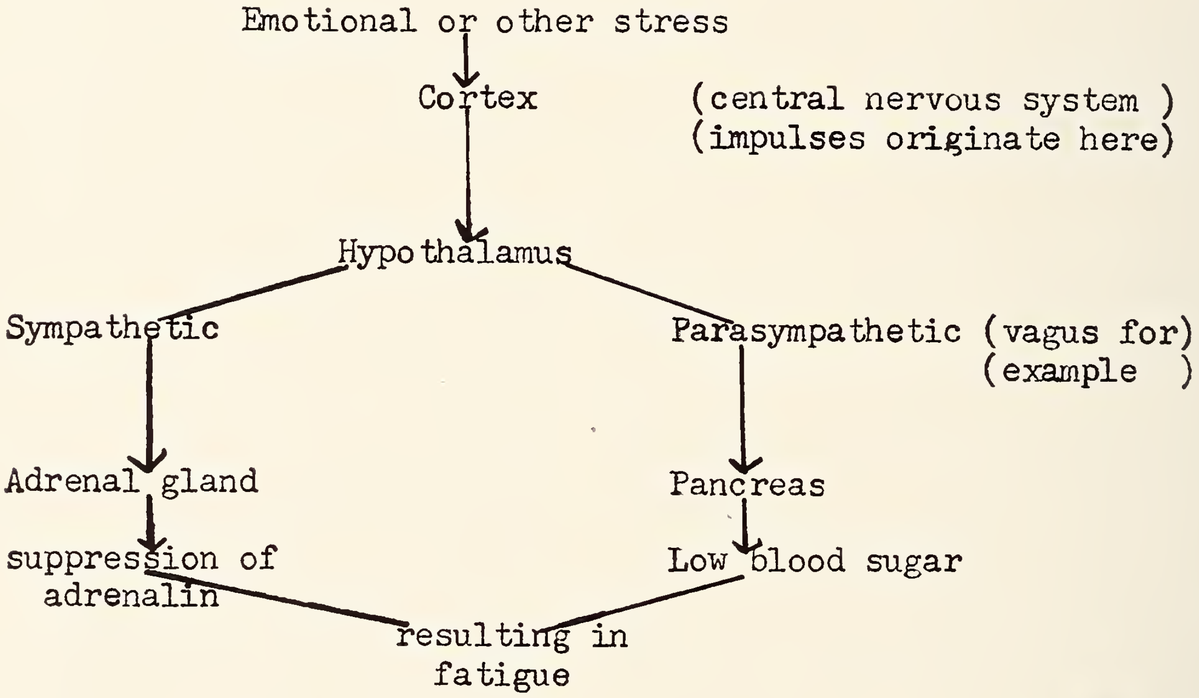
Whether or not an illness is purely a functional disturbance or an organic lesion, the disease pattern is always modified by emotional factors. Evidence today points out that the autonomic nervous system is the main link in transforming emotional stimuli into somatic responses. Corroborative evidence is provided by the fact that the influence of emotional stress is particularly marked where a disease mainly affects functions under autonomic control, and by the fact that great benefit results from treatment with drugs acting on the autonomic nervous system.

Basis of Therapy

In view of our experience with a large number of patients presenting functional ills, we agree with the present-day theory that autonomic imbalance is largely responsible for the development of such conditions. The following diagram explains briefly how the various nerve pathways play a mechanistic role in the development of such symptoms.



The above sketch shows the result of emotional impulses on two particular organs leading to the symptom—fatigue. It must be pointed out that every organ is innervated by these pathways and that any change in the number of nerve impulses will produce a variation in organ function. These changes are manifested by such symptoms as sweating, palpitation, diarrhea, constipation, et cetera.

Treatment

It is often difficult to pin-point the exact nature of functional gastrointestinal symptoms or that of any functional ill for that matter. Oftentimes, a particular individual who has worked himself into a key job in a large organization and who may be under constant tension and pressure develops symptoms of gastrointestinal distress because economically he knows he cannot quit his job for which he has worked so hard and long, and yet truthfully realizes his inadequacies (Case 2). Medication in this type of individual is difficult. One cannot rightfully employ large doses of barbiturates because, actually, this will not cure him and, in addition, will produce drowsiness and slowing of his thinking which will make the handling of his tasks even more difficult.

Most physicians realize that one cannot state a fast and hard rule which will cover medications for every patient. It is necessary to remember that every patient is an individual and that for every individual a specific regime should be organized which will be most effective for him. For example, in a proven case of duodenal ulcer (in which tension state is present), immediate relief is not always obtained with such standard accepted anticholinergic drugs (Banthine®, Pro-Banthine®, Prantal®, Antrenyl®), in addition to antacids and a proper diet. We have found that the addition of a complete autonomic stabilizer (1 tablet b.i.d., or t.i.d) may often be sufficient to make therapy successful. When gastrointestinal disease exists in older individuals—during the climacteric phase—one may often find that the addition of estrogens or androgens may be desired in order to convert ineffective therapy into beneficial therapy. One must be careful, however, not to employ drugs which may be contraindicated or harmful.

From the above diagram, one quickly sees that if the excessive impulses set forth by the central nervous system, and both the sympathetic and parasympathetic activity can be reduced, autonomic imbalance can thus be restored and symptomatic relief eventually achieved. Basic pharmacologic studies by Rothlin[5] demonstrated that drugs inhibiting either the parasympathetic or sympathetic nervous system could be given together without balancing out their effect. In other words, ergotamine tartrate continued to inhibit sympathetic impulses, and belladonna alkaloids continued to inhibit parasympathetic impulses even though administered together. Addition of phenobarbital did not diminish activity of any of the three drugs. Administration of this combination restores autonomic balance because it acts on all three nervous pathways involved in the development of functional symptoms. Karnosh and Zucker[2] have previously suggested that "probably the best medication for all neurovegetative disorders is a combination of bellafoline (vagal sedative), ergotamine tartrate (sympathetic sedative), and phenobarbital (cerebral sedative)." Recent papers[1,3,4,6] indicate the therapeutic trend toward the control of such conditions by means of controlling autonomic activity.

Case Histories

The following case histories demonstrate our results with this agent.*

Case 1.—W. S., a white, forty-seven-year-old milkman, presented himself with a typical history of duodenal ulcer of six weeks' duration. He complained of severe epigastric pain, relieved by food, but which developed two to three hours after eating. He had severe heartburn and spasm. There was no history of previous illness, but the patient had been operated on several years before for an inflamed appendix. Family and marital history were negative.

Physical examination revealed no abnormalities. The man was of normal weight for his height. Upon palpitation the epigastric area was found to be tender. A laboratory examination was completely negative. This included a complete blood chemistry, sedimentation rate, etc. The x-ray studies revealed a typical, small but active duodenal ulcer.

He was placed on a typical ulcer regime, starting out with milk for a few days and gradually increasing non-acid forming foods. He was given antacids and anticholinergic agents. At the same time he went on vacation in order to completely follow the regime and get needed rest.

He returned after one week on the ulcer regime with very little improvement in his symptoms. The pain was as severe as previously. Interestingly enough he complained of severe fatigue and lassitude, although he was not working, and was supposedly on a restful vacation. The above regime was continued but in addition he was prescribed one tablet t.i.d.-a.c., of Bellergal (complete autonomic stabilizer), and one b.i.d.-a.c., of therapeutic B-Complex capsules. He was seen three days later and appeared completely relieved of all symptoms. He stated he no longer felt so tired and was able to enjoy his vacation, and was completely free of his gastric symptoms. He has been on this regime for over a year with no further complaints. X-ray studies show healing of the ulcer crater.

The above case history shows the effectiveness of the addition of a complete neurovegetative sedative in the treatment of duodenal ulcer. This organic lesion often has with it an emotional overlay which may prevent prompt healing. The symptom of fatigue is typical of a functional disorder and as previously discussed will only improve with restoration of autonomic balance.

Case 2.—This thirty-seven-year-old male executive who presented a history of severe atypical pain in the epigastrium region and left upper quadrant of the abdomen, of one and one-half years' duration. This individual assumed a great deal of responsibility in his executive capacity and he took his position and work most seriously. He attained this position at a young age which resulted in a feeling of insecurity for fear that he was unable to live up to the demands made on him.

He apparently had no family or marital problems and had had no history of previous illness. The physical examination was negative except that on palpitation there was tenderness in the epigastrium and upper left quadrant. Laboratory examination was entirely negative. Two complete sets of gastrointestinal series were made, as well as one gall-bladder x-ray study. All three of these were negative except that they revealed a spastic colon. This finding is entirely in keeping with the history of nervousness and feeling of insecurity due to his position. He was placed on a bland roughage diet and prescribed anticholinergic agent and sedatives (barbiturates), with no relief.

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*Bellergal (Sandoz).

After several trials with a variety of drugs he was finally placed on one tablet t.i.d.-a.c. of Bellergal and 0.5 grams of Mephenesin t.i.d. He was seen two weeks later and had shown much improvement. The Bellergal was reduced to one tablet a.c. and 0.5 grams of Mephenesin b.i.d. He has been on this dosage for six months and has been completely relieved of his gastric pain and distress.

This case history is a typical example of a functional disorder. It is easier to diagnose, for the simple reason that the emotional cause of the symptoms are obvious upon discussion. There are two problems with such a patient, for besides being able to control the symptomatology, one must also help him to adjust to his environmental stresses. As previously discussed, the proper use of a complete autonomic sedative aids in producing symptomatic relief. By stopping the pains the patient naturally feels better and can begin to learn to adjust himself emotionally. In this particular instance the cause of his problems were discussed with him and after several sessions, he began to understand how his symptoms resulted from the stresses under which he was living. He was advised to try and get more relaxation, particularly out-door exercise, and to try and develop some interesting hobby in order to have something, other than his work, on his mind. Many of these executives fail to "leave their work in the office," and must be educated not to take it home with them. Under a modified regime this patient has continued to show marked improvement.

Conclusions

We have presented two examples of case histories which demonstrate a method of treating gastrointestinal disease of a functional or organic nature, or, in which a combination exists. We recommend a drug which contains sympathetic, parasympathetic and central inhibitors, for this particular combination appears to work more rapidly and appears to necessitate smaller doses than any drug previously used. Other adjuncts such as Mephenesin or hormones are employed as indicated. The emotional aspect of these conditions must be considered and adequate psychotherapy instituted in order to help the patient readjust himself to his environmental stresses.

References

1. Cerulli, F.: Am. J. Psychiat., 108:799 (April) 1952.
2. Karnosh, L. J., and Zucker, E. M.: A Handbook of Psychiatry. p. 248.
3. Kavinoky, N. R.: J. Am. M. Women's A., 7:294 (Aug.) 1952.
4. MacFadyen, B. V., Am. Pract. & Digest Treat., 2:1028 (Dec.) 1951.
5. Rothlin, E.: Schweiz. med. Wchnschr., 64:188 (March 3) 1934.
6. von Witzleben, H. D.: J. Missouri M. A., 49:486 (June) 1952.

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PROBLEMS AND OBLIGATIONS OF ORGANIZED MEDICINE

(Continued from Page 411)

doctors with University problems and in turn to learn the doctors' problems and what the staff can do to help them.

It has been my very great pleasure and privilege to try to serve both organized medicine and the medical school of this state. I have tried to interpret to each the attitude of the other. There is no incompatibility. There is no clash of interests. There should be no friction.

I am sure that the physicians of this state are very proud of their school and wish to see it continue to climb in stature and recognition.

CHANGES IN BONE AFTER EXPOSURE TO ULTRASONIC ENERGY

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INTEREST in the medical application of ultrasonic energy gained initial momentum in Germany, in 1932.[3] Since then, many widely varying conditions have been considered to be indications for ultrasonic therapy. In an effort to establish a basis for the clinical application of ultrasound, workers at this institution have studied various experimental aspects of this problem during the past six years. The outstanding conclusion to date is that no other known agent can heat bone selectively in so effective a fashion.[1,4]

Ultrasonic waves are those in the acoustic spectrum that are beyond the range of the human ear. The Standards Committee on Electroacoustics has set the lower limit of ultrasonic frequency at about 15 kilocycles per second.[5] The frequencies used most commonly in treatment are 800 and 1,000 kilocycles per second. The respective wave lengths in muscle at these two frequencies are 1.92 and 1.54 mm.

Histologic studies have revealed the production of osteogenesis in the bones of normal dogs after exposure to certain amounts of ultrasonic energy.[2] Subsequently, it was considered advisable to test the reproducibility of this phenomenon. We also wished to study the animals for a longer period than the twenty-one weeks used in previous studies and to add roentgenologic evaluation of any changes. This paper is a preliminary report of the study just suggested.

Equipment and Methods

The equipment necessary for production of ultrasonic energy includes a radio-frequency oscillator that generates alternating current of the desired frequency. This current is transmitted through a long coaxial cable to the sound head, or transducer. The long cable permits freedom of motion of the sound head, which is essential for clinical use. Housed in the sound head is a quartz crystal that has been cut so that its natural vibratory frequency coincides with the frequency produced by the particular generator associated with it. This crystal has piezoelectric properties that cause it to change dimensions when an alternating electric current is applied to it. These changes, occurring with high-frequency alternating current, result in vibrations of the crystal of the same high frequency. This is the source of the ultrasonic waves.

Ultrasonic power can be regulated through the generator, and can be measured by means of a power meter calibrated to read in watts representing the total output of the radiating surface.

In most of our work we used the Siemens Sonostat generator, which had a frequency of 800 kilocycles per second; the attached sound head had a radiating surface of 10 sq. cm. We also used the Ultrasonor generator, a product of the Ultrasonic Medical Equipment Corporation. This generator produced a frequency of 1,000 kilocycles per second and had a sound head with a radiating surface of 5 sq. cm.

During exposure to ultrasonic energy, the animals were anesthetized by intravenous injection of 25 mg. of pentobarbital sodium per kilogram of body weight.

Both femora of a series of sixteen dogs were exposed through the lateral aspect of the thigh; a stationary technique was used. Either 15 watts of power with a frequency of 1,000 kilocycles per second or 20 watts with a frequency of 800 kilocycles per second was applied for three periods of five minutes each, with a five-minute interval for cooling between exposures. In four of these animals, a needle thermocouple was inserted into the thigh so that its tip (the site of one thermojunction) was directly over the middle of the femoral shaft. The position of the sound head

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Abridgement of thesis submitted by Dr. Ardan to the Faculty of the Graduate School of the University of Minnesota in partial fulfillment of the requirements for the degree of Master of Science in Orthopedic Surgery.

The Mayo Foundation is a part of the Graduate School of the University of Minnesota.

Dr. Ardan is a Fellow in Orthopedic Surgery at the Mayo Foundation; Dr. Janes is in the Section of Orthopedic Surgery, and Dr. Herrick is from the Biophysics section of the Mayo Clinic.

that gave the most rapid heating of the thermocouple was determined, and the sound head was clamped in this position. The maximal increase of local temperature reached in these four dogs during single exposures ranged from 11.35° to more than 33.55° C. During several exposures the calibrated scale of the meter was exceeded.

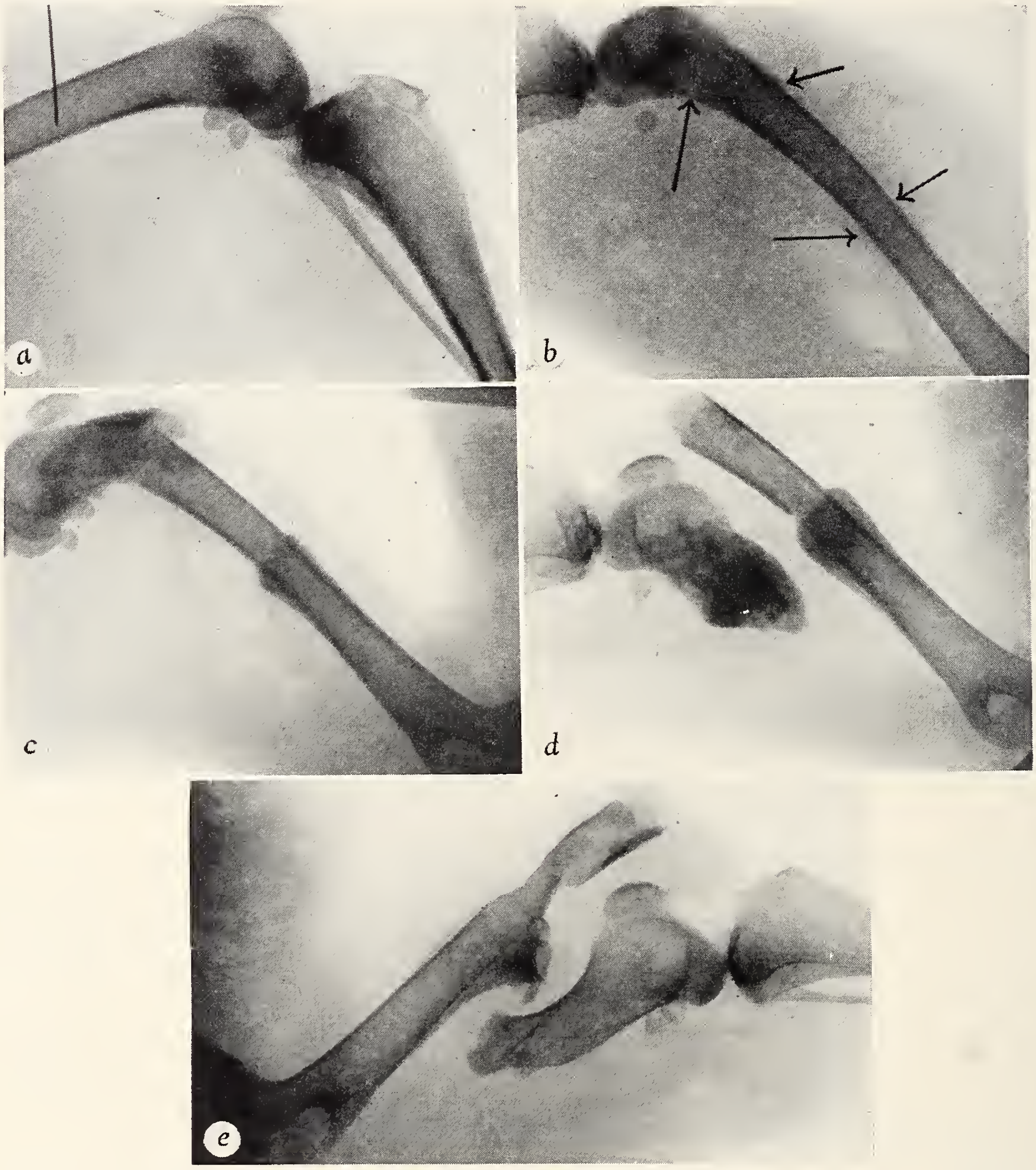


Fig. 1. *(a.)* Femur of dog prior to exposure to ultrasound. Needle thermocouple in position. *(b.)* Same femur ten days after three exposures to 20 watts of ultrasonic power for five minutes. Early periosteal reaction is seen at each end of a segment of bone thought to be necrotic. The field of sound struck the center of this segment. Areas of liquefied fat are seen. *(c.)* Same femur two weeks after application of ultrasound. Note progression of periosteal reactions, the appearance of rarefied bands at each end of the necrotic segment and the fracture through the distal rarefied band. *(d.)* Same femur one month after exposure. Note complete displacement of fractured fragments and no evidence of healing. *(e.)* Same femur five months after exposure. Note resorption of periosteal reactions and necrotic segment. Little effort at healing is present.

The remaining twelve dogs were exposed blindly by attempts to aim the field of sound at the middle of the femoral shaft at a given distance above the lateral femoral condyle.

A coupling medium of mineral oil was used to eliminate any air, which prevents the transmission of ultrasonic energy.

Results

We have evaluated results of a twelve-month roentgenologic and a three-month histologic follow-up in these animals.

Clinically, all animals exposed showed a limp in one or both hind legs for twelve to twenty-four hours after exposure. Other than this, only three dogs showed any early adverse reactions; these were all from the group of four in which the sound had been guided by the thermocouples. In these dogs, small, slowly healing, third-degree burns appeared in the skin of the medial aspect of the thigh directly opposite the sound head, that is, at the point of exit of the ultrasonic energy. In addition, massive edema and apparent paralysis of both hind legs developed in one of these dogs on the fifth day after exposure. This edema and paralysis subsided during a period of fourteen days.

However, other changes were noted in the interim. Figure 1 shows these changes through the stages of periosteal reaction, sequestration, complete dissociation of the fractured fragments with nonunion and, finally, progressive resorption of the initial periosteal reaction and of the necrotic segment of bone. The rarefactions in soft tissue seen in these roentgenograms were interpreted to represent liquefied fat resulting from the intense heat produced during exposure to ultrasound. These changes disappeared in about three weeks. It should be noted that no other dog exposed in this or in any other series in which we have had roentgenologic controls has shown these changes in the soft tissue.

The roentgenologic follow-up demonstrated that of the thirty-two femora exposed, eleven appeared to be completely normal, and thirteen showed minimal evidence of mottled densities with occasional erosions of the cortex. The remaining eight femora showed changes similar to but, for the most part, less extensive than those just illustrated.

All of the twenty-two femora obtained from study of the first eleven dogs showed some gross changes at the site of ultrasonic incidence. Those animals in which x-ray evidence of periosteal reaction had been present showed such reaction grossly (Fig. 2*a*), along with zones of eburnation and zones of pink or brownish discoloration. These discolorations and the eburnation also were apparent in all eleven of those femora which had appeared normal roentgenologically (Fig. 2*b*). We have not made extensive microscopic studies of these latter changes as yet, but the importance of this finding lies in the evaluation of reports of ultrasonic treatment in which the claim that no changes in bone were produced is based on the normal appearance of roentgenograms.

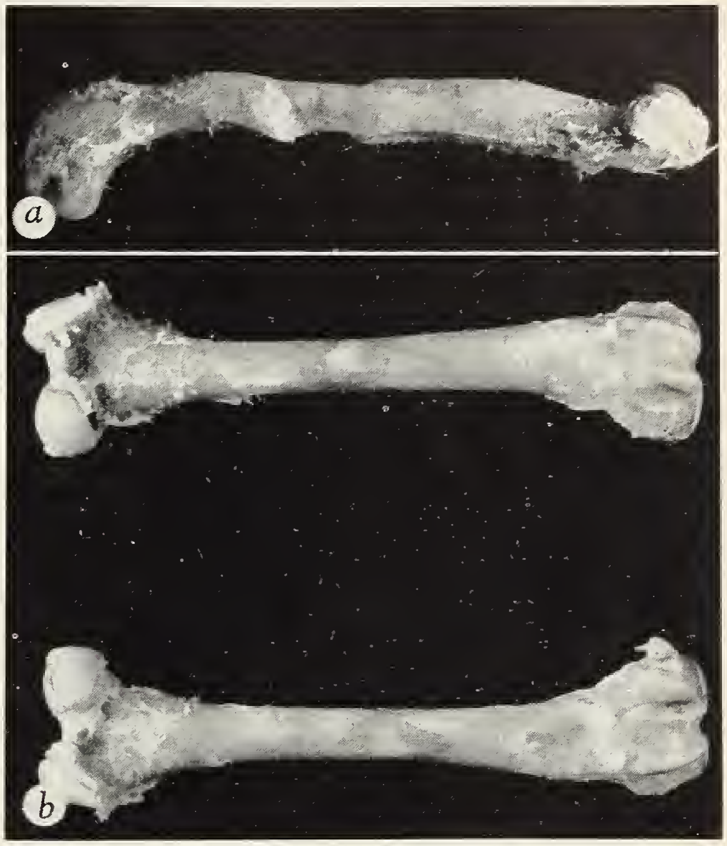


Fig. 2 *(a)*. Femur removed from a dog three months after three exposures to 20 watts of ultrasonic power for five minutes. Note periosteal reactions, with segment of bone between. *(b)* Anterior aspects of femora removed from a dog six months after three exposures to 20 watts of ultrasonic power for five minutes. Note brown discoloration in the region of ultrasonic incidence and the targetlike zone of eburnated bone near the center of these discolorations.

We have surveyed microscopic sections from three femora which exhibited changes similar to those seen in Figure 1. An entire cross section through one of the zones of periosteal reaction is shown in Figure 3*a* for purposes of orientation. The light area at the right end of this section represents a mass of cartilaginous cells undergoing endochondral formation of bone. The slightly darker mass near the left end of the section represents the original medullary cavity that has become filled with dense fibrous tissue. Around this mass can be made out the remains of the original cortex, which has become necrotic and

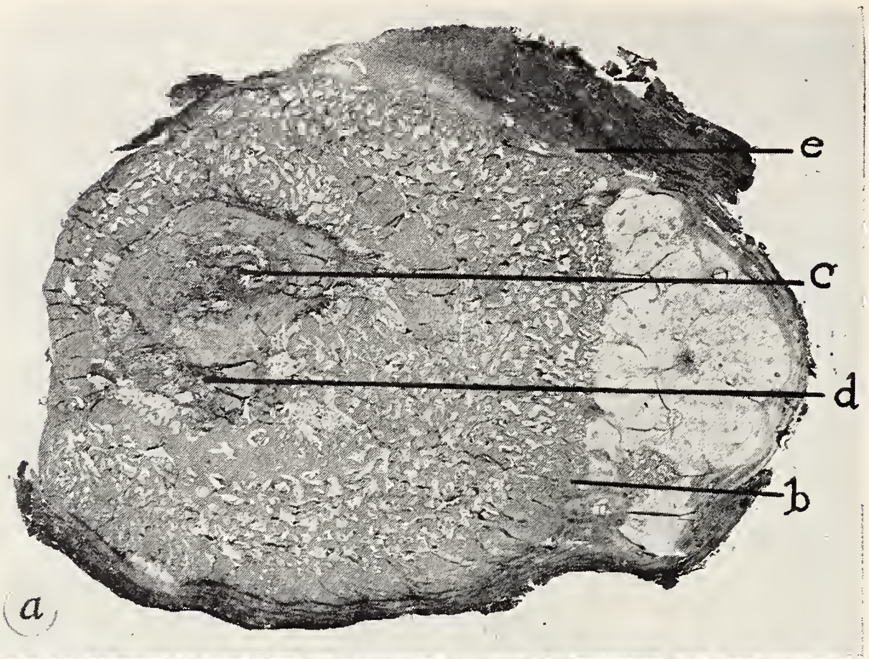
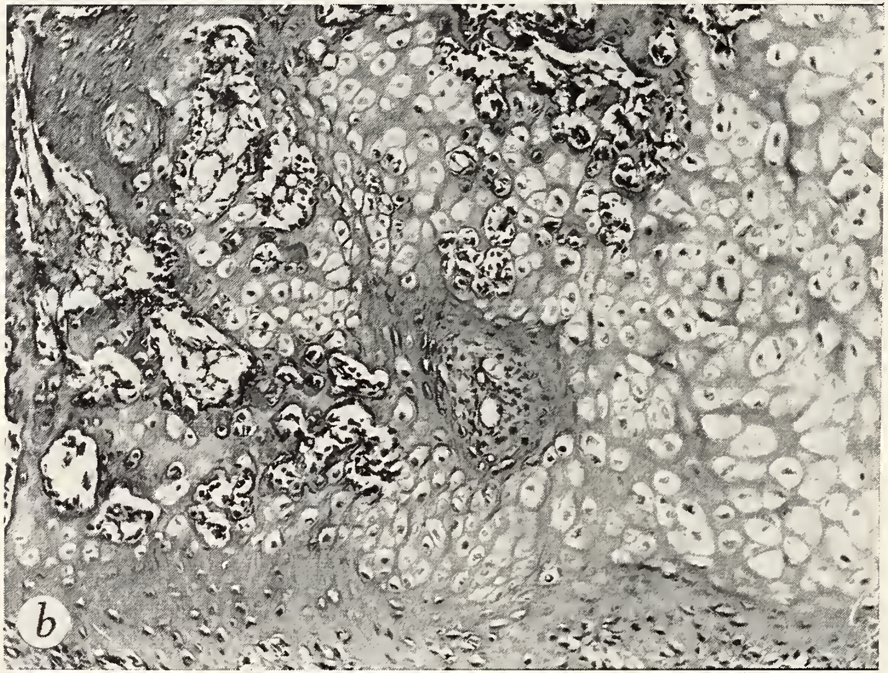
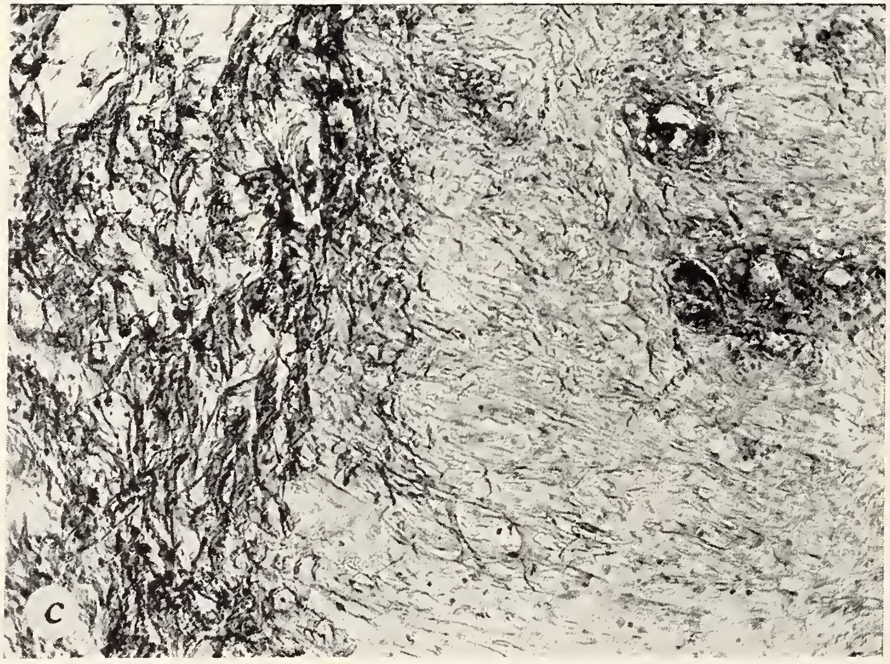


Fig. 3.

(a.) Cross section of femoral shaft through a zone of periosteal reaction three months after three exposures to 20 watts of ultrasonic power for five minutes. The origins of the next four sections are indicated (hematoxylin and eosin; x4).



(b.) Endochondral formation of bone occurring subperiosteally.



(c.) Dense fibrous tissue that has replaced the original marrow contents.

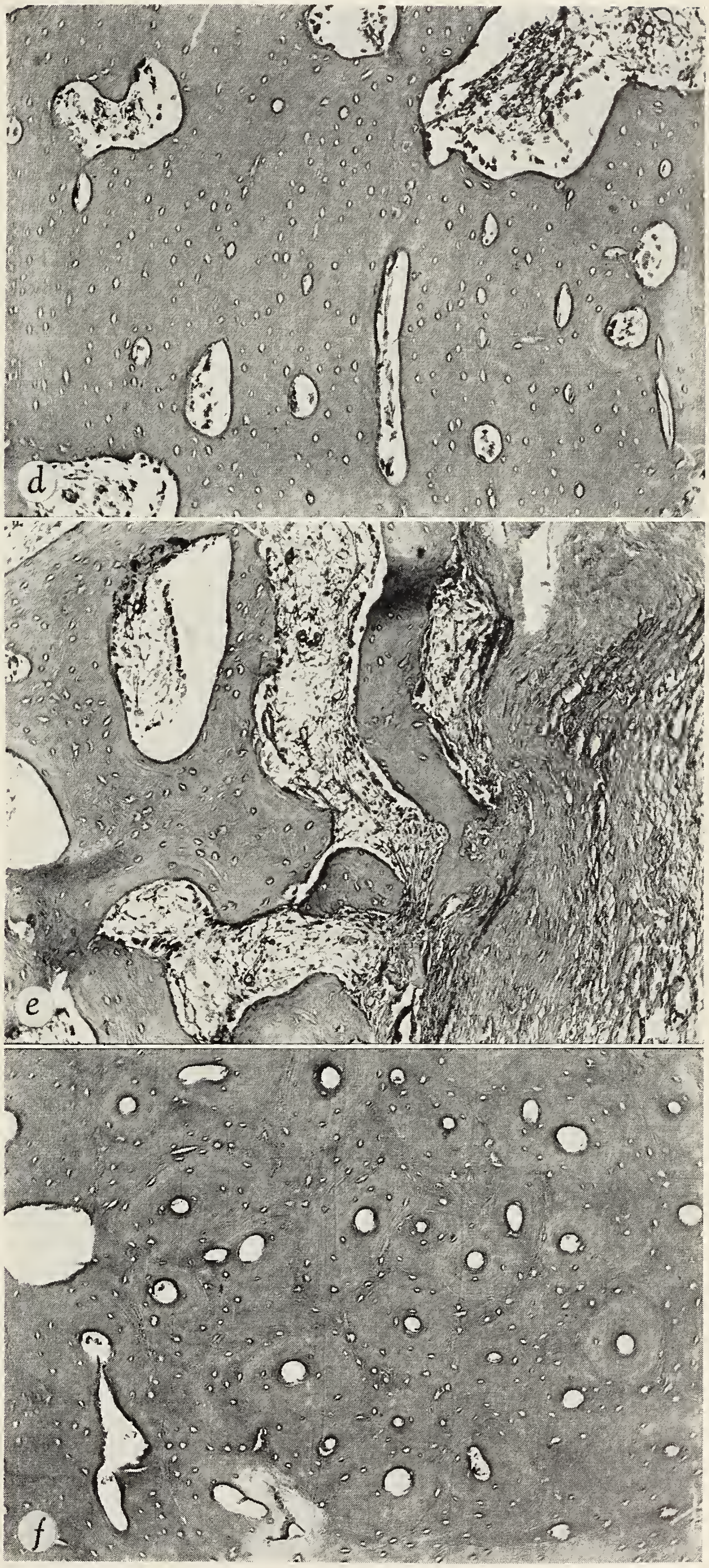


Fig. 3.

(d.) Remains of the original cortex showing loss of normal architecture of cortical bone and loss of osteocytic nuclei.

(e.) Thickened periosteum with subperiosteal formation of new bone.

(f.) Cortex of the necrotic segment between two areas of periosteal reaction. Note loss of osteocytic nuclei but retention of haversian architecture (all hematoxylin and eosin; x95).

broken up. The remainder of the section is comprised of a growth of new bone occurring under an elevated and thickened periosteum. Figure 3*b, c, d* and *e* show higher magnifications of the areas just described. Figure 3*f* is a section through the necrotic segment of the same bone seen roentgenologically to lie between the two zones of periosteal reaction. The loss of osteocytic nuclei can be seen, although the haversian units have remained fairly intact.

Conclusions

1. The response to apparently uniform amounts of ultrasonic energy is variable. All eight femora showing extensive changes occurred in dogs receiving twenty watts of ultrasonic power. However, of the eleven femora that appeared normal roentgenologically, five received 15 watts and six received the larger dose of 20 watts.

2. As in many diseases, negative results of roentgenographic study after exposure to ultrasonic energy do not rule out changes in bone.

3. Ultrasonic energy is capable of dramatic destructive changes in bone, as well as of osteogenesis. It should be noted that the technique of application of ultrasonic energy used in this study is not that advised for clinical use. Although the wattage used was within the limits considered safe for human therapy, the stationary technique concentrated this energy at one spot. With the stroking or massaging technique used clinically, the heating effect is not allowed to become cumulative.

4. Although our results should not be used to condemn the clinical use of ultrasonic energy, they should warn against promiscuous application of this form of energy by untrained personnel.

References

1. Bender, L. F.; Herrick, J. F., and Krusen, F. H.: Temperatures produced in bone by various methods used in ultrasonic therapy; an experimental study. Arch. Phys. Med., 34:424-433 (July) 1953.
2. Bender, L. F.; Janes, J. M., and Herrick, J. F.: Histologic Studies Following Exposure of Bone to Ultrasound. Arch. Phys. Med. & Rehab. (In press.)
3. Freundlich, H.; Söllner, K., and Rogowski, F.: Einige biologische Wirkungen von Ultraschallwellen. Klin. Wchnschr., 11:1512-1513 (Sept. 3) 1932.
4. Nelson, P. A.; Herrick, J. F., and Krusen, F. H.: Temperatures produced in bone marrow, bone and adjacent tissues by ultrasonic diathermy; an experimental study. Arch. Phys. Med., 31:687-695 (Oct.) 1950.
5. Standards Committee, 1950-1951: Standards on electroacoustics: definitions of terms, 1951. Proc. Inst. Radio Engineers, 39:509-532 (May) 1951.

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PRESCRIPTION WRITING

Careless prescription writing may give the patient the impression that there is careless examination and diagnosis. Whether the prescription be written in English or Latin, the important thing is to so write it that it will not be all Greek or Sanskrit to the pharmacist, and that he will know exactly what the prescriber wants.

There were many stories about the illegible handwriting of Horace Greeley, and we just heard a revised up-to-date version of one of these, but it had to do with a physician's prescription.. It concerns a man who got a prescription which was described as having been written in the doctor's usual illegible scrawl. It seems the man recovered before he got around to having the prescription compounded, so he put it in his card case and forgot about it. Later he found a slip of paper in the card case and could not figure out what it was. So he tried it in different ways. For two years he used it as a railroad pass; several times he gained admission to Rockefeller Center Music Hall; it got him into the Yankee Stadium and an exclusive club; he used it as a note from his boss to the cashier for a raise in salary; and finally he brought it home and gave it to his daughter. She played it on the piano and won a scholarship in a conservatory of music.—From the *Pennsylvania Medical Journal,* February, 1952.

DEGENERATION OF THE KIDNEY TUBULES (LOWER NEPHRON NEPHROSIS) ASSOCIATED WITH ELECTROLYTE IMBALANCE

Preliminary Report

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NOT INFREQUENTLY the pathologist has been unable to account for the cause of death in a patient who has died without adequate organic pathology.

In many of these instances degeneration of the kidney tubules may have been found in varying degrees of severity. This has not usually been considered to be adequate to account for the death of the patient unless it has been associated with anuria.

The finding of degenerative changes in the kidney tubules has been called lower nephron nephrosis,[14] transfusion reaction,[1,6,7,9] renal anoxia,[17] crush syndrome,[2,4,9] traumatic anuria,[3,9,18] septic abortion kidney,[21] pigment nephrosis and a variety of other things.[5,11] In addition it has been found to be associated with burns,[12] severe trauma, toxemia of pregnancy, mushroom poisoning, delirium tremens, black water fever,[9,16] carbon tetrachloride, ethyl glycol poisoning,[10,20] sulphanilamide sensitivity,[8,13] and retroperitoneal hemorrhage associated with obstetrical difficulties.[19,22]

We have reviewed many of the case reports in the medical literature portraying degeneration of the kidney tubules. All of those cases which indicated electrolyte studies revealed an electrolyte imbalance. This finding, however, was given no further consideration.[15] In all of the other cases, where electrolyte studies were not done, the nature of the illness or the symptoms make it appear logical to expect that an electrolyte imbalance could have existed in those cases. The symptoms which suggest this possibility are vomiting, diarrhea, fistula, extensive edema, exudative losses (burns), dehydration,[14] abdominal distension, ileus, septicemia, inadequate nutrition and the common practice of administering glucose and saline. All of these factors are notorious in their ability to cause a loss, or a shift, of the electrolytes.

Complete blood electrolyte studies and water and electrolyte balance studies were done on 102 major surgical cases for other purposes.

TABLE I.

	No Organic Postmortem Findings Group I	Organic Postmortem Findings Group II
Carcinoma g. i. tract	3	2
Obstructive jaundice	1	
Esophageal stricture	1	
Obstructive duodenal ulcer	2	2
Intussusception	1	
Perforation g. i. tract (Appendicitis, ulcer, diverticulitis)		5
Esophageal v.v.		1
Mesenteric thrombosis		1
Small bowel obstruction		1
Total	8	12

Among these were eight patients who died without adequate organic postmortem findings to account for the death and twelve patients who presented adequate pathology. Both of these groups had suffered an electrolyte imbalance and showed tubular degeneration in the kidneys. The nature of their primary disease is shown in Table I.

In Group I, the disease condition was corrected surgically, but in all instances the patients did poorly in the postoperative period and finally died without evidence of organic complications. All of these eight patients presented tubular degeneration in the kidneys as the only significant postmortem finding.

In Group II, three of these patients were not operated upon as the stage of their disease did not seem to warrant it. The others had emergency surgery. All of these patients did poorly and died with sufficient organic pathology to account for their death. In addition tubular degeneration in the kidneys was found at postmortem examination. In Group II, the diseases were, on the whole, more severe than in Group I in that many of these patients suffered initially from peritonitis in addition to the underlying primary disease. All of these patients in Group I and Group II were known to have an electrolyte imbalance. Anuria or oliguria was a finding in four of these cases. It was not a consistent

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Presented at the meeting of the Minnesota Surgical Society, Minneapolis, October 31, 1953.

TABLE II.

	Number Patients Showing Electolyte Abnormalities	
	Group I	Group II
Hyponatremia	0	1
Hypernatremia	3	4
Hypopotassemia	3	7
Hyperpotassemia	1	0
Hypochloremia	3	10
Hyperchloremia	2	2
Acidosis	3	9
Alkalosis	3	5
Hyperglycemia	7	7
Uremia	5	8

finding. Nine of these patients received blood transfusions. Only one mild transfusion reaction was encountered. The nature of their blood electrolyte imbalances is recorded in Table II.

There was no constancy in the abnormal electrolyte pattern. Every patient was found to be abnormal in three or more items. On the whole when the electrolyte pattern was more extensively disturbed, the degree of tubular degeneration was likewise greater.

A characteristic study pattern can be observed in Table III in which it can be seen that the patient suffered a severe electrolyte imbalance in all items. She died twenty-five days after surgical release of common duct stone. During this postoperative period she continued to vomit and was given only glucose and saline until three days before her death when the electrolyte imbalance was recognized and an attempt made to correct it. Postmortem examination revealed no organic cause for death. The kidney tubules showed extensive (4+) tubular degeneration (Fig. 1).

TABLE III.

M. L., Age 74	Blood Plasma		Average Balance for Three Days				Postmortem Findings
			In	Out	Balance	Urine	
Obstructive jaundice	Na	170	386	19	+367		Pulmonary congestion.
	K	2.35	162	44.3	+117.7		Myocardial fibrosis.
25 days—Glucose and saline intravenously	Cl	65.6-122.0	297.7	19.2	+278.5		Tubular degeneration.
	CO_2	50.9-19.8					
	BUN	158					
	Sugar	365					
	Water balance		3366	2284	+1082	1252	4+

TABLE IV.

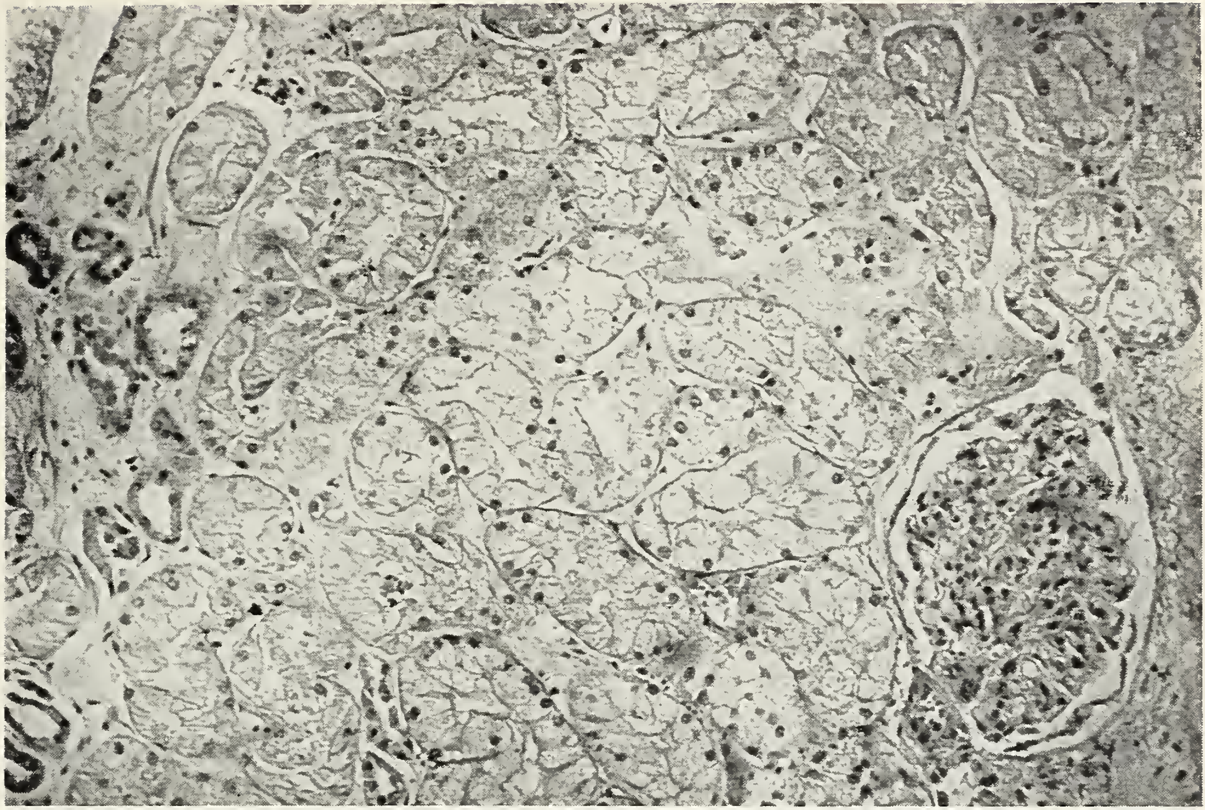
L. M., Age 43	Blood Plasma		Average Balance for Seven Days				Postmortem Findings
			In	Out	Balance	Urine	
Ruptured ileitis	Na	154	24.47	10.97	+13.50		Peritonitis.
Subdiaphragmatic Abscess (Operated)	K	2.2	112.02	87.10	+24.90		Hydrothorax.
	Cl	89.0	149.3	101.1	+48.2		Br. Pneumonia.
	CO_2	36.5-15.9					Tub. Degen. 3+ showing regen. (recovered from electro-imbal. 8 days pre-death.
	BUN	70					
	Sugar	137					
	Water balance		3218	3101	+117	1106	

TABLE V.

E. G.	Blood Plasma		Average Balance for Four Days				Postmortem Findings
			In	Out	Balance	Urine	
Cancer colon resected	Na	148.0	100	152.3	—52.3		Died 48 hrs. postop. Cerebrovasc. Head post. not done. Old subdiaphrag. abscess. PMN infiltra. in spleen. No tubular degeneration.
Old diaphragmatic abscess	K	3.5	45.5	44.8	+ .8		
	Cl	111.0	129.1	143.0	—13.9		
	CO_2	28.1					
	BUN	11.0					
	Sugar	145.0					
	Water balance		2775	2521	+253	1277	

Another instance in which the patient recovered from the electrolyte imbalance with death attributable to peritonitis is shown in Table IV. This patient died from peritonitis eight days after she had recovered from a severe electrolyte imbalance. In this instance evidence of regenerating tubular disease was shown in the kidneys (Fig. 2). This finding was also observed in two other patients who had recovered from their electrolyte imbalance and died eleven days and twenty-four days, respectively, later from other factors.

A third group of patients were studied for control. Among these were serious diseases such as carcinomatosis, diabetic coma, etc., so that if the disease could have caused tubular degenera-



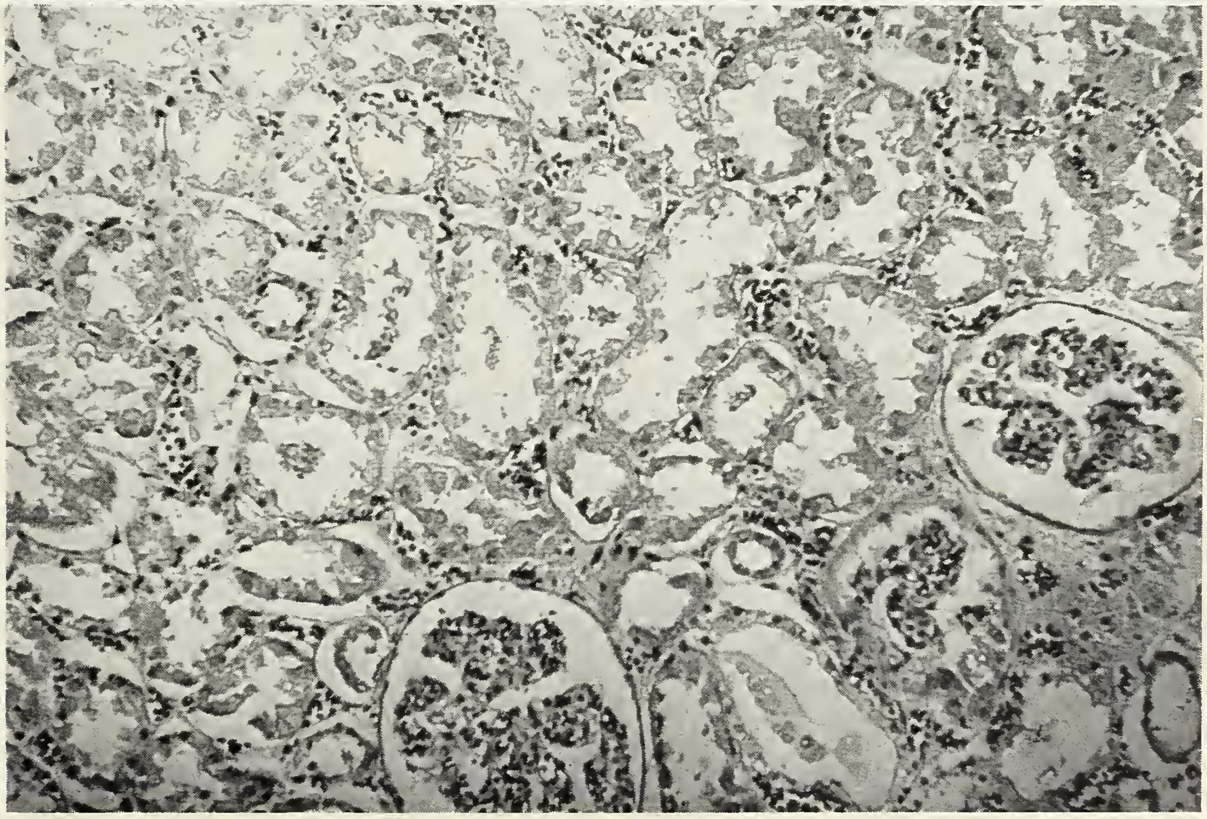


Fig. 1. Obstructive jaundice; operative release. Twenty-five days of glucose and saline. Electrolyte imbalance severe (See Table III). Tubular degeneration 4+ involving all areas of the kidneys. Cells hydropic-glomeruli normal. No oliguria. No organic cause for death found post mortem.

Fig. 2. Ruptured ileitis, peritonitis. Postoperative hypopotassemia, hypochloremia, alkalosis and then acidosis and uremia from which patient recovered eight days before death. Postmortem examination revealed peritonitis and septicemia. Tubular degeneration in scattered areas (2+) showing evidence of regeneration.

tion it should have been present in some of these. Complete studies were not done on all of the control group, but the history and physical findings suggest that an electrolyte imbalance should not have been present. Likewise, an examination of the kidneys obtained postmortem in a series of sudden deaths failed to reveal tubular degeneration in any of them.

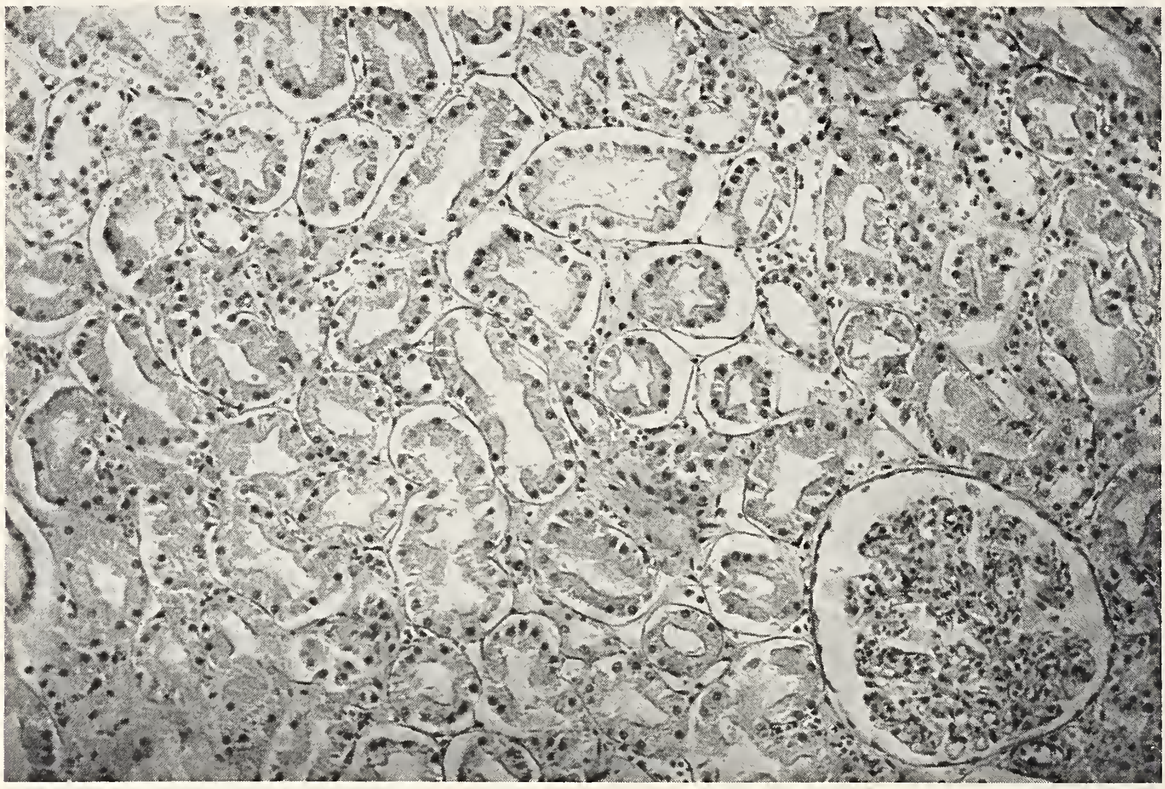


Fig. 3. Normal kidney.

One of the cases in this control group suffered severe disease, but was known definitely not to have an electrolyte imbalance. This case is illustrated in Table V (Fig. 3).

Comments and Conclusions

The blood urea nitrogen was found to be elevated in thirteen of the patients. It was found to be higher in those who presented the greatest degree of tubular degeneration. This is probably merely an index of the inadequacy of the kidney function.

The blood sugar was found to be elevated in all instances, but seemed to have no correlation with the degree of tubular degeneration or the severity of the electrolyte imbalance. The elevated blood sugar was not considered to be due to diabetes, except in two known diabetics, but was thought to represent a manifestation of the metabolic disturbance which is present in some cases of surgical trauma. Tubular degeneration of the kidneys was not found to be present in two diabetic coma cases in the control group and, therefore, it seems that the elevated blood sugar need not be considered a factor in causing the tubular degeneration.

Water balance studies indicate a positive water balance in four cases, negative water balance in one case, water equilibrium in eight cases and insufficient data in seven cases. There appeared to be no correlation between the water balance and the degree of tubular degeneration or the electrolyte imbalance.

The consistency of the known electrolyte imbalance in Group I and Group II associated with the postmortem finding of tubular degeneration, and the absence of an electrolyte imbalance in the control group associated with the finding of normal kidney tubules, suggests that the pathogenesis of tubular degeneration of the kidneys may be related to an electrolyte imbalance.

It is our conclusion from these findings that when tubular degeneration is found in the kidneys at postmortem, a retrospective evaluation of the clinical picture would, in all probability, indicate that an electrolyte imbalance had existed. When microscopic evidence of tubular *regeneration* is evident, it can also be presumed, on the same basis, that the patient was in the process of recovering from an electrolyte imbalance.

Any studies which are done in the future endeavoring to establish the pathogenesis of tubular degeneration of the kidneys must take into consideration the electrolyte and water balance status of the subject before concluding that tubular degeneration of the kidneys is of more specific origin.

References

1. Ager, G. D., and Gould, A. G.: Uremia following blood transfusions; nature and significance of renal changes. Arch. Patho., 33:513-532, 1942.
2. Birnbaum, W.: Crushing injuries with renal failure. Calif. & West. Med., 64:15-18, 1946.
3. Bywaters, E. G. L.: Discussion on effects on kidney of trauma to parts other than the urinary tract—including crush syndrome. Proc. Ray. Soc. Med., 35:321-339, 1942.
4. Bywaters, E. G. L., and Beal, D.: Crush injuries with impairment of renal function. Brit. M. J., 1:427, 1941.
5. Bywaters, E. G. L., Dible, J. H.: Acute paralytic myohemoglobinuria in man. J. Path. & Bact., 55: 7-15, 1943.
6. Daniels, W. B.; Leonard, B. W., and Holtzman, S.: Renal insufficiency following transfusions. Report of thirteen cases. J.A.M.A., 116:1208-1215, 1941.
7. DeNavasquez, S.: Excretion of hemoglobin with special reference to transfusion kidney. J. Path. & Bact., 51:413-425, 1940.
8. French, A. J.: Hypersensitivity in the pathogenesis of the histopathologic changes associated with sulfonamide chemotherapy. Am. J. Path., 22:679-701, 1946.
9. Foy, H.; Altmann, A.; Barnes, H. D., and Kondi, A.: Anuria with special reference to renal failure in black water fever, incompatible transfusions and crush injuries. Tr. Roy. Soc. Tropic. Med. & Hyg., 36:197-238, 1943.
10. Geiling, E. M. D., and Cannon, Paul, R.: Pathologic effects of elixir sulfanilamide (Diethylene glycol). J.A.M.A., 111:919, 1938.
11. Gilligan, D. R., and Blumgart, H. L.: March hemoglobinuria; studies of clinical characteristics, blood metabolism, and mechanism with observations on three new cases and review of literature. Medicine, 20:341-395, 1941.
12. Goodpastor, W. E.; Levenson, S. M., et al: Clinical and pathologic studies of kidney in patients with thermal burns. Surg., Gynec. & Obst., 82:652-670, 1946.
13. Lentscher, J. A., and Blackman, S. S., Jr.: Severe injury to kidneys and brain following sulphathiazol administration, high serum nitrogen and chloride levels and persistent cerebral damage. Am. Int. Med., 18:741-756, 1943.
14. Lucke, Col. Baldwin: Lower nephron nephrosis. Military Surg., 99:371-395, 1946.
15. Lund, Chas. C.; Green, R. W.; Taylor, F. H., and Levenson, S. M.: Burns. Surg., Gynec. & Obst., 82:443, 1946.
16. Maegraith, B. G., and Findlay, G. M.: Oliguria in black water fever. Lancet, 2:403-404, 1944.
17. Maegraith, B. G.; Hovard, R. E., and Parsons, D. S.: Renal syndrome of wide distribution induced possibly by renal anoxia. Lancet, 2:293, 1945.
18. Parsons, C. G.: Traumatic uremia. Brit. M. J., 1: 180-182, 1945.
19. Parsons, N. F.; Galub, L. J., and Hunter, R. M.: Crush syndrome in obstetrics and gynecology. J.A.M.A., 131:500-504, 1946.
20. Wells, A. H., and Call, James J.: Hydropic degeneration of kidney tubules. Clinical Pathological Conference. Am. J. Clin. Path., 33:263, 1953.
21. Wyatt, J. P., and Godberg, H.: The septic abortion kidney. Am. J. Obst. & Gynec., 59:337-344, 1950.
22. Young, J.: Renal failure after uteroplacental damage. Brit. M. J., 2:715-718, 1942.

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FINDINGS OF FIRST YEAR'S STUDY OF DOCTORS IN SERVICE

Results of the first year's survey of physicians leaving active military service are carried in a report recently issued by the AMA's Council on National Emergency Medical Service. Information obtained from this continuing questionnaire study is being used as the basis for a series of conferences with Department of Defense and Armed Forces representatives in an effort to improve the utilization of medical personnel and the formulation of a more effective voluntary officer procurement system.

During the first year of the study—July 15, 1952, to August 1, 1953—a total of 3,948 completed questionnaires revealed that the average time spent in service was 24.7 months; average tour of duty in U. S., 7.6 months; average tour of foreign duty, 17.1 months. Twenty-nine per cent felt there was overstaffing, 20 per cent understaffing and 51 per cent adequate. Of those assigned to domestic duty, 53.4 per cent were engaged in treating military personnel, 28.3 per cent in treating military dependents and 18.3 per cent in "other"; while of those assigned to overseas duty, 51.8 per cent treated military personnel, 23.8 per cent dependents of military personnel and 24.4 per cent "other." Answers to the question regarding the *type of medical care* provided for *other than* military personnel indicate that in the Army and Navy the most frequent type was outpatient care, while in the Air Force it was obstetrics and gynecology.

Regarding the question, how national and local medical associations can better serve their members in service, the following activities were suggested—more information via a newsletter, etc.; personal visits by civilian doctors to evaluate grievances; invite military doctors to civilian medical meetings; assist in locating position after discharge; assist in preventing evasion of military service; distribution of questionnaires to physicians in service; provide specialists for clinical conferences.—*AMA News Notes.*

PREVENTION OF MAJOR COMPLICATIONS DURING ANESTHESIA

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PREVENTION of complications is largely a matter of philosophy on the part of the anesthetist. That an ounce of prevention is worth a pound of cure is a growing philosophy in modern anesthesia. Prophylactic anesthesia requires that the anesthetist understand not only the problems of anesthesia but also the problems of surgery, as well as the individual physiology and pathology of each patient. Armed with such knowledge and the experience gained from the conduction of many previous anesthesias, the anesthetist can anticipate complications and can institute measures to prevent their occurrence. The anesthetist's ability to anticipate complications in their incipiency is most important in the matter of insuring safety to the patient and uninterrupted successful completion of the operation. Such ability is more important than any ability to revive the patient heroically after the jaws of death have begun to close.

Three requisites for prevention of complications are awareness of changes in the patient, early diagnosis of changes, and early treatment of minor complications. These will be discussed separately, first as to the means available today for practicing them, and later as to the means we may hope to have available after ten to fifteen years of progress.

Awareness

Awareness of changes in the patient is the most important requirement and also the most difficult to practice constantly and adequately. Man is aware of his environment through his five senses: sight, smell, taste, hearing and touch. John Snow used these same senses to study his patients, and during the one hundred years between his time and ours, improvements in the efficiency of these senses have been slow. At present, prevention of complications must depend mainly on the constant and uninterrupted use of these unaided senses. Certainly such prevention will always require alertness as well as competence on the part of the anesthetist. To be constantly in contact with the condition of the patient from moment to moment, the anesthetist must necessarily be in close proximity to him at all times. An arm's length is possibly the maximal distance that should separate the two and the practice of some early anesthetists of leaving the operating room without providing for intelligent constant attendance of the patient is not generally tolerated today. The anesthetist must necessarily be in close proximity in order to employ his five senses in unceasingly evaluating the condition of his patient.

Hypoxia is the greatest threat to the safety of most patients and is one of the most serious of complications. The symptoms of acute severe hypoxia are easily recognized and attract the attention of the surgical team as well as the anesthetist. On the other hand, the symptoms of chronic hypoxia of a slight but dangerous degree may be insidious; hence, they can easily be overlooked by the busy anesthetist who may be responsible for duties other than the constant observation of the patient. Too frequently the history, after cardiac arrest, is: "The patient was just fine and everything was going along nicely when all of a sudden the heart stopped." At necropsy no cause of death can be found and a diagnosis of "vagovagal" reflex may be made. This diagnosis made by exclusion may or may not be correct. Operations on the human heart itself have given some of us an opportunity to observe the stamina of this organ and to develop a high regard for its ability to function under difficulty. It seems logical that in some cases of unexplained cardiac arrest the history of the sequence of events leading up to the arrest may not have been just as they seemed even to the honest anesthetist. Before arrest, some hearts have likely been subjected to unfavorable circumstances but have been able to maintain some function under stress. The arrest, instead of being an initial sudden occurrence, was the climax to a slow decrease in the ability of the heart to compensate for the stress. If the anesthetist had become aware that the heart was under stress, preventive measures could have been taken to

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From the Section of Anesthesiology and Intravenous Therapy, Mayo Clinic, Rochester, Minnesota.

remove the unfavorable circumstances and the ultimate arrest could have been avoided.

All the numerous ways in which hypoxia can occur cannot be mentioned here but they should always be in the back of the mind of the alert anesthetist. Regardless of the type of surgery and the manner of draping, some part of the patient's skin or nails should be exposed in good light, so that the color can be observed and cyanosis prevented. As will be mentioned later, the color of the skin is a grossly inaccurate indication of the degree of oxygenation of the blood. However, it is certainly better to be able to observe the color of the skin at frequent intervals than not to be able to see any part of the patient. I like to have my hand on the patient's face, neck, chest or abdomen where I can feel the respiratory motion, even when I can clearly see the motion of the breathing bag on the anesthesia machine. In this way I am doubly aware of the respiration through two senses—sight and touch. If the respiratory passages become partly obstructed, I may be warned by a third sense—hearing.

If a finger is kept in the vicinity of an artery of the patient, a subconscious contact can be maintained with the circulatory system and its efficiency. Even though the anesthetist is not able to concentrate constantly on the rate and rhythm of the pulse, any sudden change in these characteristics will have an effect similar to the sudden stopping of a ticking clock in a quiet room. The recording of the blood pressure and pulse rate at frequent intervals is indispensable but at best it is an intermittent method, and to prevent complications a constant method is desirable. Even the extremely conscientious anesthetist cannot record all these readings much more frequently than once every five minutes, and when other duties must be attended to, the interval becomes much longer.

Being a co-operative and industrious individual, the anesthetist frequently becomes responsible for too many operating room chores such as assisting with draping, adjusting surgical lights, adjusting the operating table, wiping the surgeon's brow and so forth. Even the administration or supervision of the supportive therapy may be his responsibility. These added duties must of necessity distract his attention from the condition of the patient, much to the patient's disadvantage. Even the maintenance of his own anesthesia equipment in a properly functioning condition is sometimes a dangerous distraction from attention to the patient.

It is, therefore, evident that to become instantly aware that a complication is impending the anesthetist must be constantly aware of what is happening to and in his patient. There is much room for improvement by the development of more attentive habits by anesthetists, but on the other hand, it is humanly impossible for any individual to effect complete concentration on any factor for hours on end without interruption. As will be discussed later, the answer to this problem is the development of instruments which can function unceasingly. It seems possible that awareness might be made mechanical.

Diagnosis

Let us imagine for a moment that we have reached that stage of development of the specialty of anesthesia where constant awareness has been made possible and the anesthetist is immediately conscious of every change as it occurs in his patient. Then he must diagnose the change and evaluate it. This diagnosis will likely never be mechanical, and it is the function which makes anesthesia necessarily the practice of medicine in contrast to a series of technical maneuvers. The ability to diagnose is based on an understanding of the basic sciences. In addition, judgment is required, and judgment is based primarily on training and experience.

Suppose the blood pressure of the patient decreases. The anesthetist must examine many factors which might have exerted an influence to bring about the change. Then from the facts at hand he must make a decision. Basically, he must decide whether the fall in pressure is the result of a decrease in cardiac output or a decrease in peripheral resistance. Then he must go further and further into detail by constantly asking himself the question, "Why?" Rarely he may be able to make a definitive diagnosis and know minutely what is happening in his patient. All too frequently, however, the ability to make more than a provisional diagnosis is hampered by a lack of facts about the individual patient. The anesthetist may know volumes of physiology which is based mainly on animal experiments or he may know from anesthesia textbooks the symptoms and signs of many classes of complications, but rarely is he able to determine accurately the physical or chemical changes in each patient as they occur. Most of the time he only has a gross idea about even the concentration of the gases which the patient is breathing from the bag on the anesthesia machine.

Diagnosis is necessary primarily for the purpose of indicating treatment and is not usually thought of as a preventive measure. The early and accurate diagnosis of minor complications and their effective treatment is the only way of preventing major complications. The old saying, "Great oaks from little acorns grow," seems to apply here—apparently few major complications develop suddenly from a normal state. Instead, most are preceded by a series of progressively severe minor complications which either were not recognized or were misdiagnosed or mistreated. When a cardiac arrhythmia develops during anesthesia, the anesthetist must first realize that the arrhythmia exists, then he must diagnose it, evaluate its significance, and, if necessary, institute treatment to eliminate it and prevent more serious cardiac complications. But under ordinary circumstances, how will he be able to make a definitive diagnosis? He may think the arrhythmia is due to premature contractions, but how can he know whether the origin of the premature contractions are auricular, nodal or ventricular? When an experienced cardiologist would hesitate to venture diagnosis from palpation alone, how can an anesthetist definitely differentiate a tachycardia of supraventricular origin from a tachycardia of ventricular origin? Yet, the accurate diagnosis is important because the former would likely not demand immediate treatment, but the latter should be treated immediately, perhaps with an intravenous dose of a procaine-like drug or quinidine. How can one know the heart rate of a patient with both an auricular fibrillation and a variable pulse deficit? The answer to all these questions is evident—by the use of an electrocardiograph—yet how many patients have an electrograph attached before a routine operation?

Early diagnosis is the crux of the whole problem of cardiac arrest on the operating table. Preparation of emergency equipment and an outline of procedure for action by the surgical team is important but is of no use in salvaging a valuable citizen unless the anesthetist recognizes cardiac arrest at almost the moment it occurs. The average delay of three to five minutes before cerebral damage occurs is usually needed to open the chest and start cardiac massage. If the anesthetist is not aware of the complication or is not able to make a definite diagnosis until after two or three minutes have elapsed, the chance of saving an undamaged patient will be slim. Those who have been faced with the problem know that such a diagnosis is difficult, when dire consequences may result from a snap and unverified diagnosis.

What evidence is necessary for the early diagnosis of cardiac arrest: lack of ability to get a blood-pressure reading or to feel a radial pulse? Several times every month each of us is jolted by the sudden inability to get a blood-pressure reading or feel a pulse, only to find that some mechanical defect has occurred in our apparatus or that the arm has been put in such a position that the circulation has been impeded. The investigation of such factors in the differential diagnosis of cardiac arrest takes time. The raising of an alarm the very instant that we suspect cardiac arrest only to admit a mistake within a few moments several times a month will cause the surgical team to lose faith in us. Then when a real cardiac arrest occurs, we will lose time convincing them that it is not just another false alarm. If the thorax or abdomen is open, the surgeon may be asked to verify our suspicions by palpating the heart or great vessels, but if the operation is elsewhere in the body, it is more difficult to make the diagnosis definitely enough and quickly enough to justify thoracotomy. Future progress in overcoming the bad results of cardiac arrest is not likely to come from the development of better methods of treatment but from the development of better methods for making an early and certain diagnosis of the complication.

Treatment

Early accurate diagnosis by an alert and competent anesthetist makes early treatment possible. It is early and adequate treatment that prevents the bad results of major complications. Adequate treatment frequently depends on the preparation of equipment, drugs and plans in anticipation of the complication before it happens. It is almost correct to say that the good anesthetist must of necessity be a pessimist, always expecting the worst even when he is doing his utmost to prevent it. From previous experiences the anesthetist knows which complications are more likely to occur in a particular patient with a particular disease, undergoing a particular operation, by a particular surgeon, under a particular type of anesthesia, and in a particular position. So in being forewarned, the thinking anesthetist should keep himself forearmed and prepared to treat such expected complications adequately.

It is sometimes necessary to treat unusual complications at unexpected times. The fact that no preparation has been made for the prevention or treatment of such complications partly explains why they seem to be fatal more often or to be followed by serious consequences. From the reports in the literature, cardiac arrest would seem to occur more frequently in good-risk patients than in poor-risk patients. If so, this fact might be explainable on the basis that poor-risk patients are observed closely every moment, that every diagnostic aid is used to follow the condition of such patients, and that preparation is made to treat the major complications which are expected but which are prevented in their incipiency. On the other hand, in the ordinary good-risk patient no complications are expected, so that no greater than ordinary preparation is made to prevent or treat unexpected complications. Much attention to monotonous detail is necessary to keep everything set up and in good working order. In addition to being a pessimist, the careful anesthetist must also possess some of the characteristics of the overly meticulous spinster housekeeper as far as his equipment is concerned. Even the presence or absence of an efficient aspirating catheter may make the difference between life and death for the anesthetized patient whose respiratory passages have become obstructed with liquid or semisolid material. As a general statement, it might be said that the prevention of major complications during anesthesia depends on the willingness of the anesthetist to spend his time and effort fussing with little details which at the moment seem inconsequential.

In summary, it may be said that major complications are best prevented by treating them before they happen.

Instrumentation for the Future

It has been shown that continuous monitoring of the patient is necessary in order for major complications to be prevented. The anesthetist with his unaided senses is able to provide only intermittent monitoring. The shorter the intervals between observations the safer it is for the patient, but it is humanly impossible for the anesthetist to provide continuous observation of the patient without instruments to aid his senses. A few of the instruments, mostly electronic, which have shown promise of being of help will be mentioned.

Cardiotachometer.—From leads similar to those used for electrocardiography the pulse rate may be shown on a dial or recorded on paper or film. Whereas the anesthetist with his finger on a pulse can only estimate the average pulse rate, the cardiotachometer can indicate the rate from one beat to another and is therefore more sensitive and more accurate. An alarm device can be used with the cardiotachometer, so that if the pulse rate falls below a predetermined figure a bell will ring, a light will flash or some other alarm will go off to warn the anesthetist that all is not well with his patient.

Direct Blood-pressure Recorder.—The determination of blood pressure by the auscultatory, oscillatory or palpation method is inaccurate and subject to variation in individual interpretation. The direct recording of the blood pressure through a needle or small flexible tube in a peripheral artery is objective, more accurate and constant. In addition, there is promise that from the recorded pulse contour a reasonably accurate estimation of the stroke volume of the heart can be calculated,[3] and, therefore, the cardiac output can be followed from minute to minute.

Electrocardiograph.—The value of this instrument is recognized by all. At present it offers the most practical means of diagnosing cardiac arrhythmias and allowing the anesthetist to be cognizant of changes in the heart before these changes reach the status of major complications. Like so many of the electronic instruments, it introduces an explosion hazard into the operating room. However, instruments are available whereby the electrocardiographic pattern can be shown on a cathode-ray screen which is enclosed in an explosion-proof housing, safely situated at the side of the anesthetist. By pressing a switch the anesthetist can cause any significant patterns to be recorded on the direct-writing electrocardiograph which has been set up in a nonhazardous area.

Electro-encephalograph. — The determination of the depth of anesthesia by the signs and symptoms as outlined by Guedel is not accurate. The signs vary from individual to individual, and the interpretation of a group of signs is not always the same to different anesthetists. There is good evidence to indicate that the electrical activity in the brain decreases as the depth of anesthesia increases. The integration of the electrical activity of the brain as recorded with an electro-encephalograph is therefore a valid means of objectively measuring depth of anesthesia, which can be shown on the dial of an anesthesia depth meter or recorded. This principle has been used to activate a servomechanism whereby the anesthetic agent is automatically administered if the electrical activity of the brain increases and the depth of anesthesia decreases below a preset degree, whereas the rate of administration of the anesthetic agent is automatically decreased if the anesthesia becomes too deep and the electrical activity of the brain decreases.[1]

Thermometer.—During recent years much attention has been focused on the changes in body temperature which occur during anesthesia and on the effects of these temperature changes. Hyperthermia during anesthesia and operation is generally considered to be detrimental to the welfare of the patient. Especially in children the body temperature may rise to dangerous levels under conditions which are apparently similar to those well tolerated by most adults and some children. A marked rapid rise in body temperature demands immediate treatment, but how is the anesthetist to know that it is rising unless it is being recorded? Electronic and mechanical thermometers are available for continuously recording temperature in various parts of the body during anesthesia. Induced hypothermia is being employed more and more, especially during operations on the heart and great vessels for cyanotic heart disease. This hypothermia must be controlled, and to be controlled some convenient type of thermometer must be used.

Oximeter.—No condition is so likely to result in major complications as a severe decrease in the efficiency of the system for transporting oxygen to the tissues. It is well known that as the oxygen content of blood decreases, the color tends to change from red to blue, but the gradation of the change is too small to be accurately estimated by the human eye. Some of the newer models of oximeters make it possible for the delicate changes in the color of arterial blood to be constantly monitored by a photoelectric cell and for the changes to be shown on a dial or recorded on paper or film as absolute percentages of saturation of the arterial blood with oxygen. The Pauling type of oxygen meter makes possible rapid and accurate determination of the concentration of oxygen in anesthetic mixtures of gases and has been of great aid in studying the effects of known mixtures.

Carbon-dioxide Meter.—Carbon dioxide is of almost as much importance as oxygen, and the concentration in the blood is kept within narrower limits even than the concentration of oxygen. Accurate control is provided by nature for a very important reason and it behooves us as anesthetists to strive to see that nature is not hindered in providing safeguards. Anesthesia may mask the symptoms of change in carbon-dioxide concentration which occur readily in the unanesthetized individual. For that reason instruments for determining the concentration of this gas are important. Extremely accurate intermittent determinations are possible by use of the mass spectrometer, but continuous determination of the concentration in the respiratory gases is possible by several other methods.[2]

pH Meter.—One reason why carbon dioxide is so important is that it plays a big role in keeping the hydrogen-ion concentration within the narrow range compatible with life in the mammal. The increasing use of the combination of respiratory depressing agents and artificial respiration during anesthesia has increased the demand for instruments to show the changes in pH of the blood during the time when the respiratory center or other parts of the respiratory mechanism are paralyzed. Many unexplained major complications during anesthesia may well be the indirect effect of an unsuspected radical alteration in the pH of the blood. So far as I know, no instrument is available at present for continuously recording this factor, but in the future such instruments will be developed.

Up until now, only the advantages of instrumentation have been stated. The disadvantages are readily evident. Most instruments are still in the experimental stage of development and have been used largely for research studies, while commercially available models which are sturdy and simple enough to be used routinely by the clinical anesthetist have not been perfected. In their present form, many instruments are bulky, and if all that have been mentioned were employed at once they would fill the average-sized operating room. Most are electronic and because of the explosion hazard cannot be used when inflammable anesthetic agents are being administered. The operation of most of these instruments is complicated and the services of personnel trained in electronics are required. Even if the anesthetist has the ability to set up, run and service the instruments, he will not be able to administer the anesthesia and care for the instruments at the same time.

All these objections are legitimate but need not be appalling. Much work is yet to be done in perfecting the instruments for clinical use, but at least we have an ideal, a hope and a goal to work toward. Just because most of us cannot build an automobile or cannot service any defect in operation of our automobile does not keep us from deriving benefit from the use of automobiles. It would be a sad state of affairs if everyone who rode in an airplane had to be able to fly it unaided.

The aircraft industry makes a good comparison to the specialty of anesthesia. Before and during World War I, the pilot had a crude machine and few mechanical aids. He flew "by the seat of his pants" in about the same way that most of us give anesthesia today. However, the complexity of new developments did not deter the members of the aircraft industry, so that today huge ships with panel after panel of dials and safety devices travel faster than sound, sometimes without a human pilot. The anesthetist also must eagerly accept mechanical aids as they become available in order to facilitate the safety of his patients and put anesthesia on a really scientific basis. The problem of personnel will have to be solved in the same manner as used by the surgeons, namely, by the development of a team. An anesthesia team will be more expensive than the individual anesthetist, but the American public has always been willing to finance anything which it can be convinced is for its own good. When the surgeon himself could not attend to all details of an operation, the surgical team was accepted without question. It is up to us as anesthetists to provide safety and then to sell it to our patients. Safety means freedom from complications.

Summary

1. The factors in the prevention of major complications during anesthesia are awareness of change in the patient, early diagnosis and early treatment of minor complications.

2. Continuous monitoring of the patient is possible only by the use of instruments which are now being perfected.

References

1. Mayo, C. W., Bickford, R. G., and Faulconer, Albert, Jr.: Electroencephalographically controlled anesthesia in abdominal surgery. J.A.M.A., 144: 1081-1083 (Nov. 25) 1950.
2. Stow, R. W.: An infrared analyzer for carbon dioxide and its application to certain problems in respiratory physiology. Thesis, Graduate School, University of Minnesota, 1952.
3. Warner, H. R., Swan, H. J. C., Connolly, D. C., Tompkins, R. G., and Wood, E. H.: Quantitation of beat-to-beat changes in stroke volume from the aortic pulse contour in man. J. Appl. Physiol., 5:495-507 (Mar.) 1953.

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The contrast in tuberculosis mortality rates of the Japanese in Hawaii as compared with the rates in Japan is of interest. In Hawaii, the rate for the five-year period, 1918 through 1922, was 132 per 100,000 as compared with 12 in 1952. In Japan, on the contrary, the rate for 1918 through 1922 was 233 and, in 1951, the last available figure was 111. Similar differences exist in other races such as the Filipinos, Chinese, and Koreans, all having much lower rates in Hawaii than those reported in their respective countries of origin. In all probability, higher living standards and better nutrition are the most important factors to account for these differences.—HASTINGS H. WALKER, M.D., *American Review of Tuberculosis,* December, 1953.

PAROTID TUMORS

A Report of Six Cases

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FEW TUMORS have received such exhaustive studies and are still so poorly understood as tumors of the parotid gland. The surgeon approaches parotid tumors with timidity because of facial nerve paralysis resulting from their removal. The pathologist is hesitant about prognosticating which tumors are apt to recur after removal. As a result many patients are advised to forego surgery for a period of "observation" because of the fear of resulting facial nerve paralysis; occasionally x-ray therapy is offered as a therapeutic measure. These factors contribute to the rather discouraging results which are associated with treatment of parotid tumors.

Parotid gland tumors are rather rare. At Los Angeles County Hospital in 37,000 consecutive autopsies,[17] the incidence of parotid tumors was found to be only .023 per cent. Except in large tumor clinics, few surgeons have had much experience with these tumors. In 1943 a survey was made in the Philadelphia area[14] where it was found that in twenty-four hospitals, during a quarter of a century, there were 233 patients with mixed tumors of the parotid gland operated on by seventy-three surgeons. The average was 3.2 patients each. Only six surgeons had ten cases or more.

The origin of parotid tumors is not well understood.[3,6] Virchow in 1863 was the first to describe these tumors.[12] Since many of them contained cartilage, he believed them to be of mesoblastic origin and termed them "diffuse endochondromas." Cohnheim in 1877 speculated that in early embryonic development, more cells were produced than necessary in the salivary glands, and tumors often occurred because of their great capacity for growth. Wartmann in 1879 believed these tumors to be endothelial in origin. Krompocher in 1908, observing the basal layers of stratified epithelium, called these tumors basalomas. Favata[5] in 1948 was able to culture, in vitro, cells from mixed tumors and keep them alive for 140 days. He believed that there is evidence that mixed tumors may be derived from cells lining the ducts of salivary glands. Ligation of the salivary ducts in dogs is said to produce mixed tumors of the parotid gland. The interference with the flow of secretion in the production of these tumors may explain why recurrence happens so frequently when only a portion of the gland is removed in man. Of great importance is the recent work of Gross[7] who has produced salivary gland carcinomas in mice by injecting a filterable agent. In eighty-four mice of a certain strain, he was able to produce bilateral salivary tumors in fifteen mice. They developed on an average of 3.3 months after injection.

The conflicting ideas relative to the origin of parotid gland tumors have made a clear classification difficult.[20] A simple classification[15] which has been suggested is as follows:

1. *Cysts*—These are caused by obstruction, atresia, or congenital defects in the duct system. They are rare and benign.
2. *Adenomas*—Adenomas are well encapsulated tumors and grow slowly. They are composed chiefly of glandular tissue and are benign.
3. *Adenocarcinoma*—This group of neoplasms are usually stated to comprise about 10 per cent of all parotid tumors, although some reports suggest that 40 per cent would be a more accurate figure. They are poorly demarcated tumors that rapidly infiltrate surrounding structures. They include the onocytoma and cylindroma and are extremely malignant.[18] The recurrence rate is thought to approach 100 per cent, and usually takes place within a five-year period, 85 per cent recurring within three years, although some of these malignant tumors have been known to recur as long as forty years after removal. They invade vital structure and may metastasize to lymph nodes, bone, liver, spleen, lung, pancreas, thyroid, ovaries, adrenals, dura and brain. Total removal of the parotid gland is imperative, and many believe a radical dissection of the node-bearing area is indicated. Deep x-ray therapy has proven to be of little value in these tumors, as most parotid tumors are radioresistant.
4. *Mixed Tumors*—This group is the most controversial and comprises about 75 per cent of all parotid tumors. They are composed of three component structures: (a) complexes of epithelial cells in cords or duct-like structures; (b) connective tissue of the mucoid type; (c) substances identified as cartilage or pseudo-cartilage. They usually have a definite capsule and are rarely infiltrative. The histological structures are extremely variable[19] and are, therefore, difficult to classify. Mixed tumors are notorious for recurring after they have been removed. The recurrence rate is stated to be between 25 per cent and 40 per cent; the average recurrence time is seven years. Because they are prone to recur, there is persistent disagreement

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among pathologists as to whether mixed tumors of the parotid gland are benign or malignant. It does not seem possible to foretell the behavior of a mixed tumor from microscopic sections alone. The clinical course must be correlated and even then an accurate prognosis is often impossible. McFarland,[14] who has made an extensive study of mixed tumors of the parotid, attempted to find out with what accuracy pathologists could predict from microscopic sections the probability of recurrence. He submitted to eight eminent pathologists sections from fifty mixed tumors that had been followed for many years. There was no unanimity of agreement on any of the tumors. Fifty-two per cent of the tumors were diagnosed correctly and 48 per cent inaccurately diagnosed as to recurrence. The highest percentage of accuracy for any of the eight eminent pathologists was 63 per cent. From this study alone it can be seen why surgeons and pathologists are bewildered when confronted with mixed tumors of the parotid gland. Using Ewing's[4] criteria of malignancy, these tumors have the following characteristics:

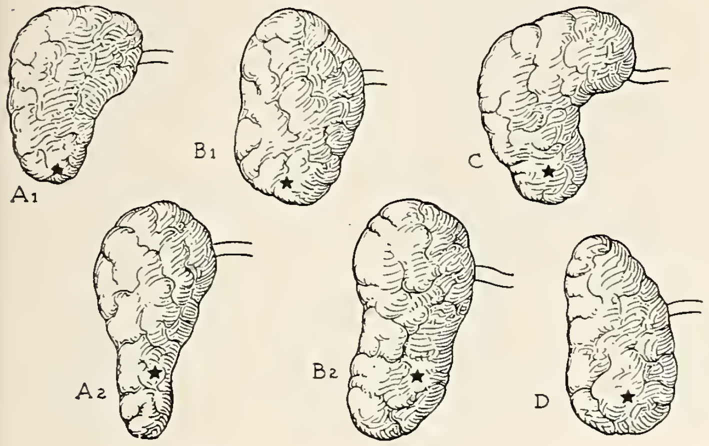

Fig. 1. Various shapes of the parotid gland (after McCormack, L. J.; Cauldwell, R. W., and Anson, B. J. Courtesy Surg., Gynec. & Obst.).

(a) *Infiltrative growth.*—Because these tumors have a definite capsule they rarely infiltrate. Should the capsule be broken, either from surgery or trauma, the tumor may infiltrate into orbit or brain.

(b) *Recurrence after removal.*—As stated previously, recurrence is far too frequent[10] and this may take place as long as twenty or thirty years after removal. For this reason, the word "cure" should be used with extreme caution.

(c) *Metastasis.*—Metastases are rare. McFarland found this to occur in but 2 per cent of his cases.

(d) *Local interference with function.*—Mixed tumors may cause deafness if the external auditory canal is obstructed. Movements of the jaw may be restricted and pressure on the facial nerve may cause facial paralysis on the affected side. If Stensen's duct is obstructed, cystic retention of secretion may result.

(e) *General toxic action of absorbed tumor products.*—There is no absorption of tumor products unless the tumor ulcerates and bacterial invasion results in sapremia or septicemia.

(f) *Local tissue destruction.*—Mixed tumors are covered by the skin of the cheek, which allows for extensive stretching and for this reason ulceration seldom occurs. The fifth and seventh cranial nerves at times become involved, causing pain and paralysis.

With these data at hand it becomes evident that in all cases of mixed tumors of the parotid gland, the prognosis should be guarded and the patient advised as to all possible eventualities.

5. *Entodermal Cysts.*—This group consists of the adenolymphomas or papillary cystadenoma lymphomatosomas. They are rare, benign and arise from lymphoid conglomeration that surrounds the first bronchial cleft and resemble tonsillar tissue. They should be removed.

6. *Tumors of Nonglandular Origin.*—These parotid tumors are extremely rare and may be composed of blood vessels, nerve[23] lymphoid or fibrous tissue. Hemangiomas of the parotid gland, with few exceptions, have been found only in infants.

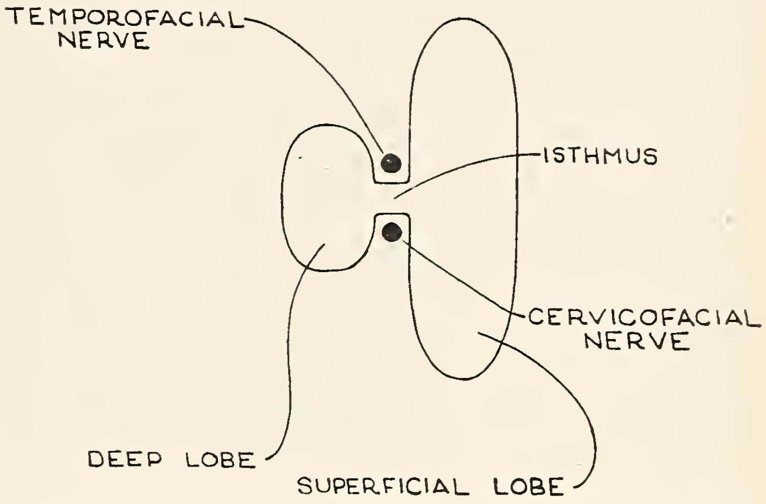

Fig. 2. Arrangement of the lobes of the parotid gland showing the isthmus at which site the facial nerve divided into the larger temperofacial branch and the smaller cervicofacial branch.

Anatomy: The parotid gland develops during the eighth week of fetal life. The glandular structure varies in shape (Fig. 1) and is held together with loose areolar tissue. There is no definite capsule, but the gland is covered by fascia which is continuous with the fascia of the neck as it passes upward from the sternomastoid to the zygomatic arch. It is bounded in front by the ramus of the mandible and posteriorly by the external auditory meatus, tympanic plate and the base of the styloid process. The gland overlies the masseter muscle. Stensen's duct emerges from the anterior aspect of the gland and courses over the masseter muscle piercing the buccinator muscle to enter the oral cavity. In the past most anatomy books have stated that the facial nerve passes *through* the parotid gland substance. This information has led most surgeons to deem it impossible to remove tumors of the parotid gland without injuring the nerve and most were hesitant to try. Fortunately the researches of McWhorter,[16] and later McCormick, Cauldwell and Anson[11] have enlightened us to the true relationship of the parotid gland and the facial nerve. They found the parotid gland is composed of a larger superficial lobe with many variable prolongations and a smaller deep lobe which are joined together by an isthmus. They further found that the facial nerve does not traverse through the gland, but rather *between* the two lobes and that the nerve invariably divides to embrace the isthmus (Fig. 2). These anatomical facts have facilitated and encouraged surgeons to attempt removal of tumors without fear of resulting facial paralysis (Fig. 3). The facial nerve emerges from the stylomastoid foramen and does not give off branches until it encounters the isthmus where it divides into two principal divisions, the temperofacial and the cervicofacial nerves. The former is two or three times the size of the latter and supplies most of the upper part of the face. These divisions usually have five terminal branches: temporal, zygomatic, buccal, mandibular and cervical. These branches anastomose freely. McCormick, Cauldwell and Anson (Fig. 4) from their dissection of one hundred individual facial nerves, concluded that the anastomosis of the terminal branches could be catalogued as belonging to one of eight types. With these facts at hand the surgeon has been reassured as to the possibility of successfully removing these tumors without resulting disfigurement to the patient.

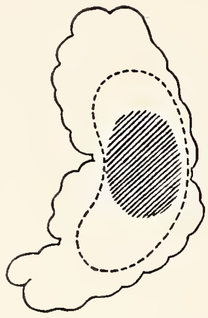

Fig. 3. Relationship of the larger superficial lobe, isthmus (Cross-batching), and smaller deep lobe (broken line) (after McCormack, L. J.; Cauldwell, R. W. and Anson, B. J. Courtesy Surg., Gynec. & Obst.).

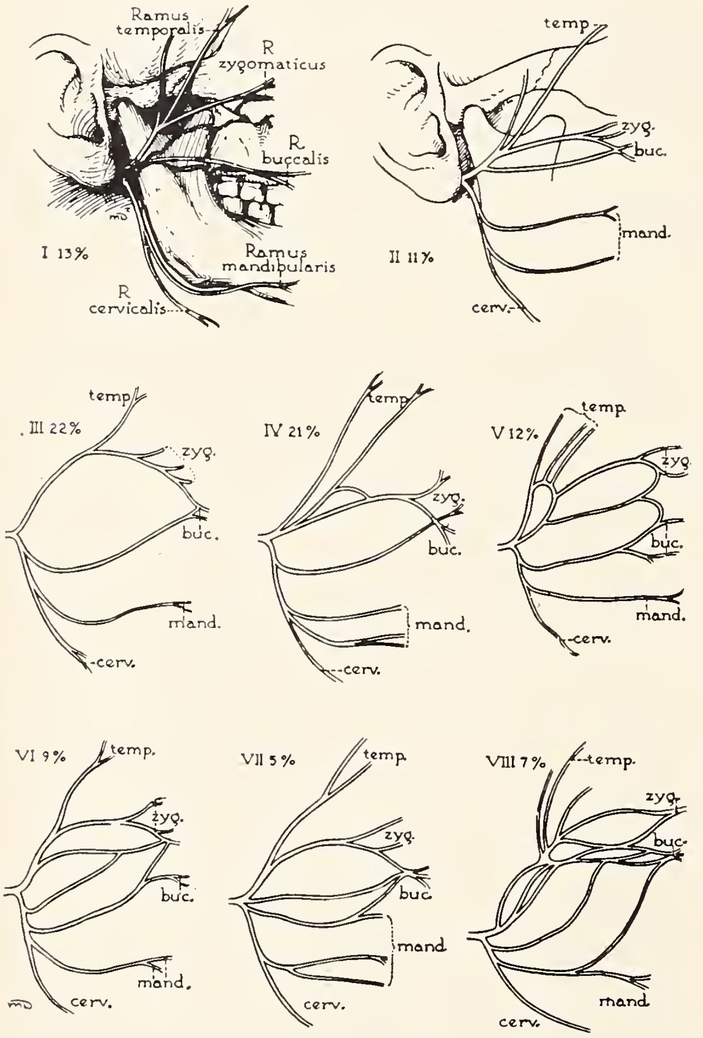

Fig. 4. Variations in the branches of the facial nerve can be catalogued into eight different arrangements.

Most patients seek treatment for parotid tumors because of embarrassment from the unsightly swelling, limitation of motion of the mandible, pain or sudden rapid growth of the tumor.[22]

Treatment: Treatment should be directed toward the complete removal of all tumor tissue with the hope of no recurrence of the tumor.[1,21] Various procedures[17] have been carried out consisting of simple enucleation of the tumor, lobectomy, or radical removal of the entire parotid gland along with the node-bearing areas. When one is dealing with adenocarcinoma, probably the procedure of choice should be the complete removal of the parotid gland.[9] Whether or not excision of the node-bearing area is indicated depends on the size and involvement of the tumor. In the case of the mixed tumors, lobectomy would appear to suffice. X-ray therapy is reported to be ineffective in the treatment of most types of parotid tumors. Technically intratracheal anesthesia, prevention of blood loss by temporary or permanent ligation of the external carotid artery, and meticulous care in separating the two lobes of the parotid gland and preserving the facial nerve will facilitate the successful removal of parotid tumors.

Case Reports

Case 1. W. P., male, aged sixty, (Fig. 5) presented himself complaining of a tumor on his left cheek which he stated had been present for two years. He stated it had been getting larger during the past two or three months. The tumor was the size of a hen's egg and was located below the zygoma and anterior to the angle of the jaw. A diagnosis was made of a parotid

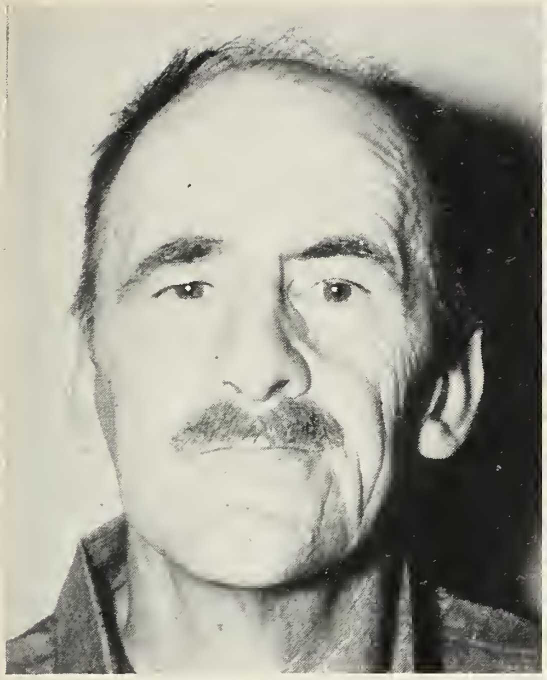

Fig. 5. W. P.—Mixed tumor of the parotid. Tumor removed by simple excision without injury to the facial nerve, except for slight inability to elevate the forehead on the affected side.

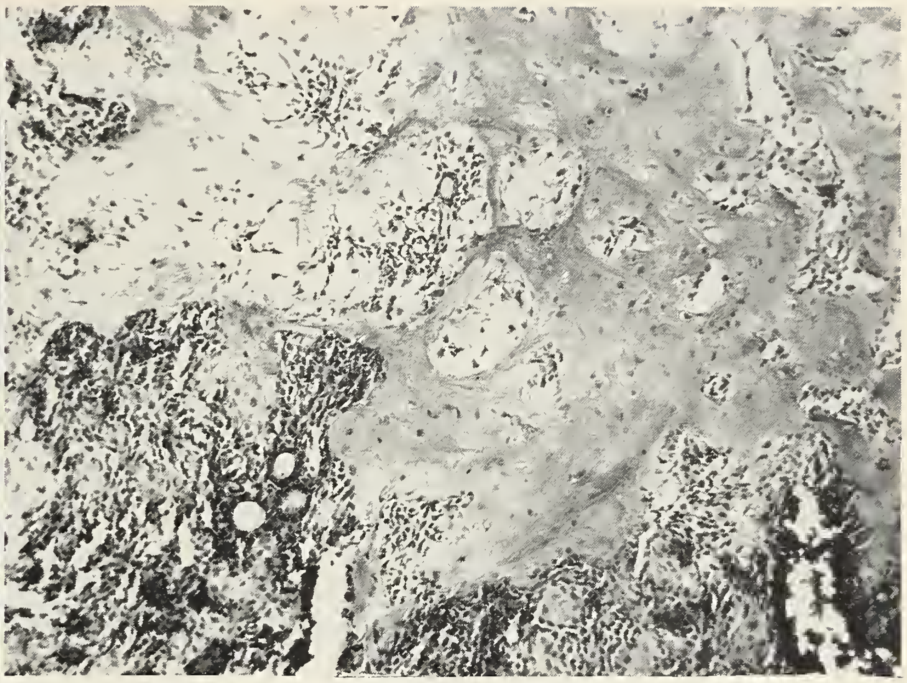

Fig. 6. Microscopically, tumor was composed of cartilage, myxomatous tissue, and masses of epithelial tissue. Some definite glands are also formed. Mixed tumor of the parotid.

tumor. On November 1, 1946, under local anesthesia the tumor was excised and it was thought at the time of surgery that a portion of the facial nerve was "in the tumor" and was sacrificed. He was discharged from the hospital on the fifth postoperative day and made an uneventful recovery. He has no facial paralysis except his inability to elevate his forehead on the affected side. There has been no recurrence.

Pathological diagnosis: Mixed tumor of the parotid gland. The tumor measures 3½ x 2½ cm., is firm, grayish-yellow in color. It is friable and appears to be poorly encapsulated. Microscopically the tumor is composed of cartilage, myxomatous tissue, and masses of epithelial tissue. Some definite glands are also formed (Fig. 6).

This patient should have had a general anesthetic and lobectomy.

Case 2. M. C., female, aged twenty-nine, complained of a swelling just below the angle of her jaw which would vary in size. She stated she had noticed the swelling for the past year. The tumor was compressible and slightly tender. A diagnosis was made of a parotid tumor and on June 28, 1948, she was operated on under local anesthesia. A cyst was encountered which contained a cloudy yellowish fluid. The cyst was excised. She made a rapid recovery and was discharged on the second postoperative day. There was no evidence of facial paralysis and there has been no recurrence (Fig. 7).

Pathological report: Cyst from the parotid gland shows no evidence of neoplasm. There appears to be a fibrous cyst wall present.

Fig. 7. M. C.—Moderately large cyst of the parotid gland excised without evidence of facial paralysis.

Case 3. L. F., female, aged seventeen, presented herself complaining of a painless swelling just below the angle of the jaw on the right side. She stated the tumor has been present for two years. She had been advised six months after onset to have the tumor removed but refused. The tumor increased in size and when first

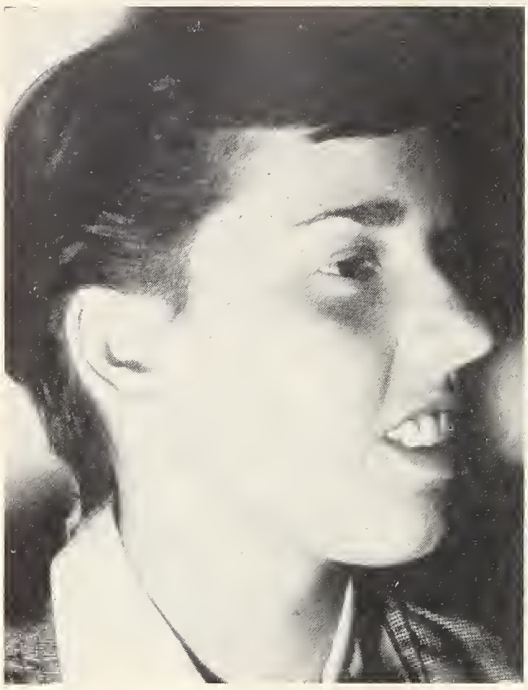

Fig. 8. L. F.—Mixed tumor of the parotid gland removed without evidence of resulting facial paralysis.

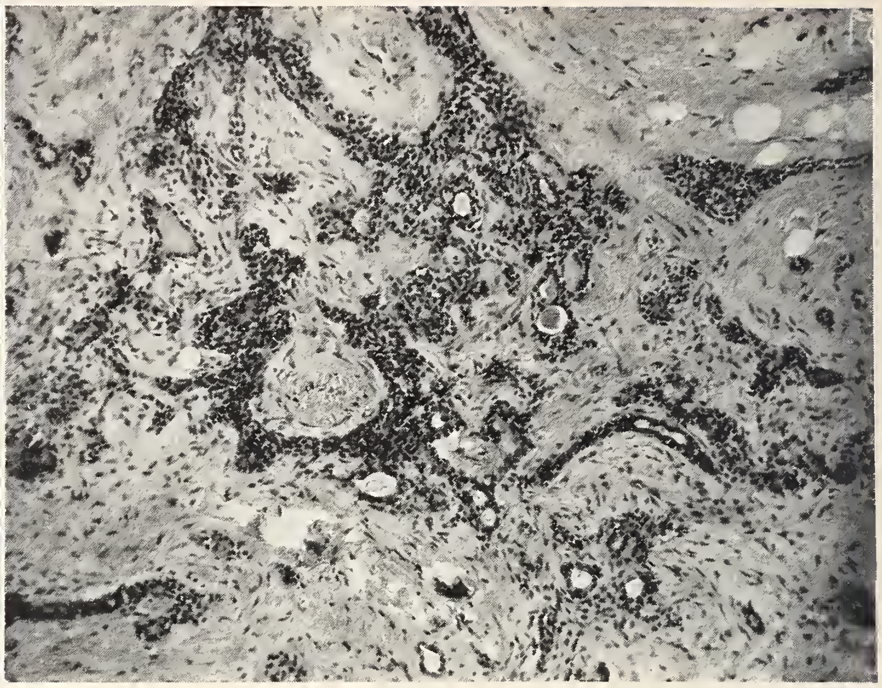

Fig. 9. Microscopically, tumor was composed of dense hyaline connective tissue stroma embedded in which are plaques of fairly well differentiated cartilage together with rather irregular cords and columns of small, deep staining epithelial cells. Mixed tumor.

seen at our clinic the tumor was the size of a lemon. On September 16, 1952, surgery was carried out under general anesthetic. There was a definite encapsulated tumor which extended from the parotid gland and was intimately adherent to the submaxillary gland. The submaxillary gland was removed along with the tumor (Fig. 8).

Pathological report: Specimen measures 4 cm. in diameter. It is firm in consistency and white in color. It has the gross appearance of malignancy. Microscopic examination shows the tumor to be composed of a dense hyaline connective tissue stroma embedded in which are plaques of fairly well differentiated cartilage together with rather irregular cords and columns of small deep staining epithelial cells. A few gland structures are formed. Diagnosis: Mixed tumor of the parotid gland (Fig. 9).

This tumor was confusing inasmuch as it seemed as much associated with the submaxillary gland as the parotid gland. Because of the type of the tumor she is being closely followed for possible recurrence.

Case 4. E. H., female, aged fifty-three, noticed a swelling in front of her right ear in August, 1952. The swelling increased in size and in November, 1952, her family doctor removed an "infected gland." The incision healed slowly and the swelling recurred. She presented herself at our clinic in June, 1953. A check was made of the pathological diagnosis in 1952 and it was found that the "infected gland" had been reported as a cylindroma and its significance not appreciated. On June 2, 1953, using intratracheal anesthesia, the external carotid artery was ligated and complete removal of both lobes of the parotid gland was carried out. Very little difficulty was encountered in preserving the facial nerve. The convalescence has been uneventful except for a transitory facial paralysis that is rapidly disappearing (Fig. 10).

Pathological report: Tumor mass measures 6 x 4 x 3 cm. It is grayish in color and very firm in consistency. There is definite infiltration with no evidence of encapsulation. Microscopic sections of the tumor show it to be composed for the most part of solid sheaths, cords and columns of epithelial cells which are markedly undifferentiated and definitely infiltrating. In a few areas glands are formed. Definite mitotic figures are present. The picture is that of a carcinoma rather than the usual mixed tumor. Diagnosis: Cylindroma of the parotid gland (Fig. 11).

This tumor was removed successfully with preservation of the facial nerve and a close follow-up will be carried out.

Case 5. S. N., aged forty-three, complained of a painless swelling over the left parotid region of two years duration. He first noticed it while shaving and had observed a slight increase in size. A diagnosis was made of a parotid tumor and excision was carried out under local anesthesia on January 11, 1941. A well encapsulated dark bluish tumor 4 x 2 x 2 cm. was removed. At the very end of the dissection the capsule was inadvertently entered and there was a slight spillage of the granular contents of the tumor. The wound was

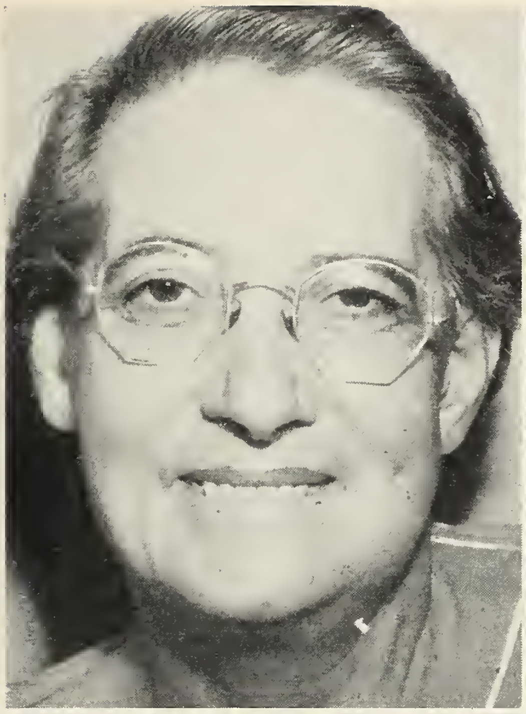

Fig. 10. *(above)* E. H.—Cylindroma of the parotid gland. Excision of both lobes of the parotid gland with preservation of the facial nerve. Four months postoperatively there remains a slight facial paralysis which has been rapidly improving.

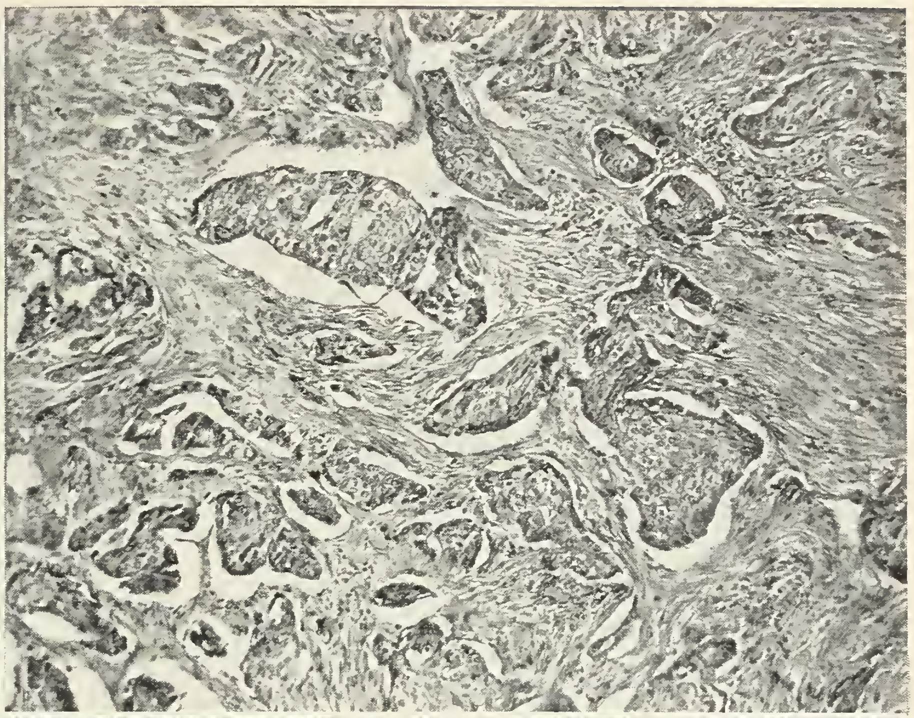

Fig. 11. *(above)* Microscopic appearance of the cylindroma showing solid sheaths, cords and definitely infiltrating. These tumors are malignant tumors.

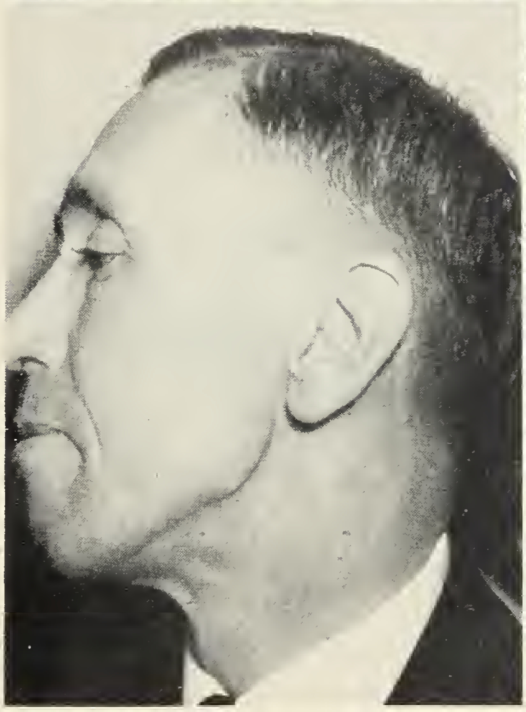

Fig. 12. *(below)* S. N.—Adenocarcinoma, grade three, removed in 1941, by local excision followed by x-ray therapy without evidence to date of recurrence. There has been no evidence of facial paralysis.

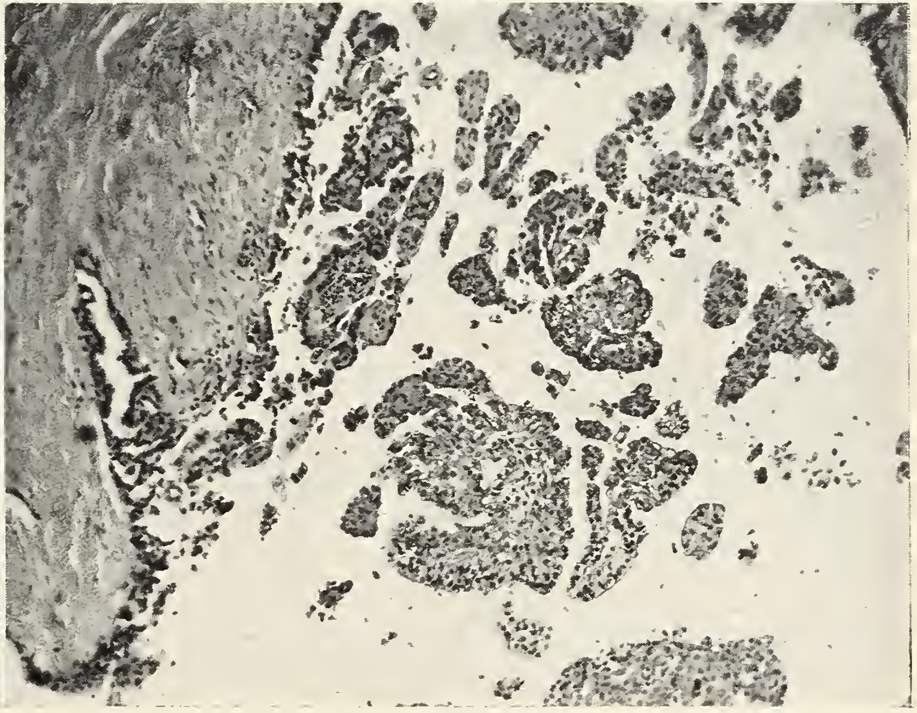

Fig. 13. *(below)* Microscopic appearance of adenocarcinoma of the parotid gland. This malignant tumor was reported by Dr. A. C. Broders, Mayo Clinic, as "adenocarcinoma, grade three, more malignant than the average grade three."

thoroughly irrigated with saline. The patient left the hospital after a few hours and healing was by primary union without any evidence of facial nerve impairment (Fig. 12). The tumor was reported by Dr. A. C. Broders of the Mayo Clinic as being "adenocarcinoma, grade three, more malignant than the average grade three." X-ray therapy was given following the healing of the incision (Fig. 13).

In the light of our present knowledge this patient should have had a complete extirpation of his parotid gland with possibly a dissection of the

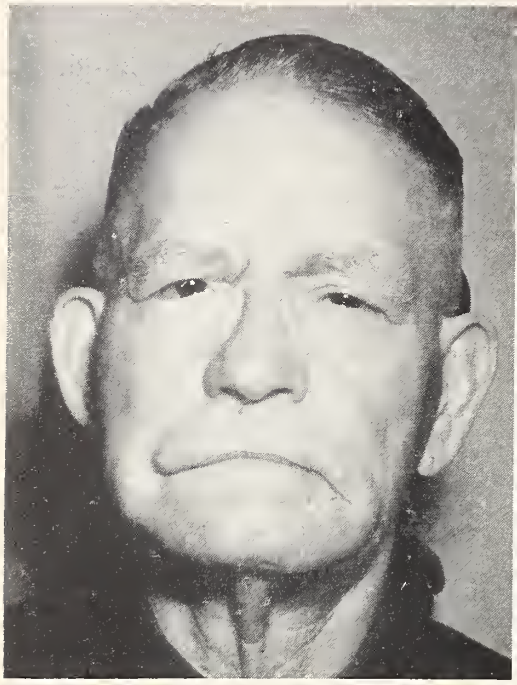

Fig. 14. H. D.—This seventy-one-year-old patient had complete removal of his parotid gland on the right side. Almost the entire gland was involved by the vascular tumor. The facial nerve was sacrificed. The facial paralysis was corrected by utilizing fascia lata from the thigh to correct the facial deformity. It is believed that this is the oldest patient ever successfully operated upon for removal of a hemangioma of the parotid gland.

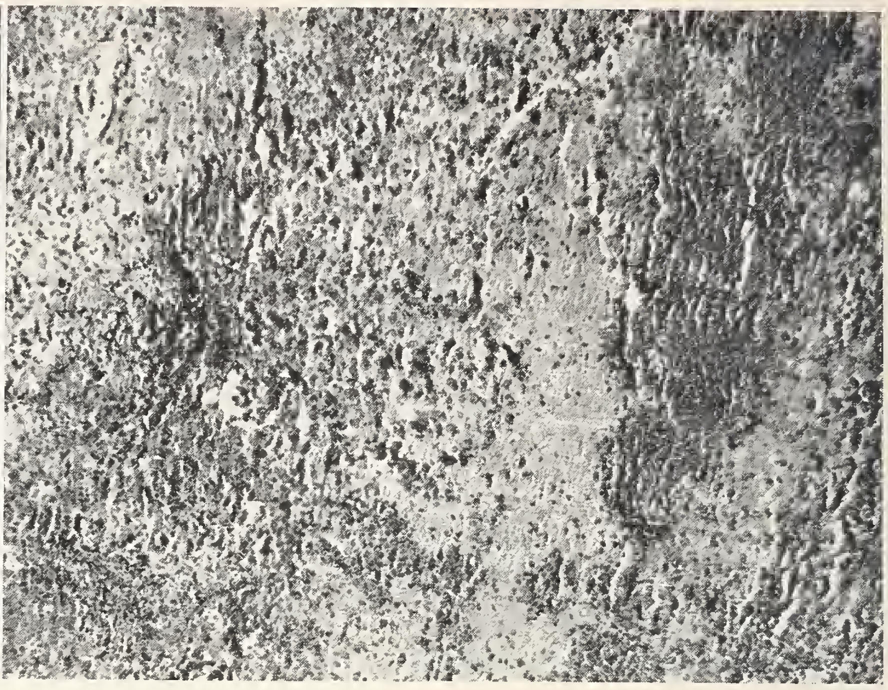

Fig. 15. Microscopic appearance of cavernous hemangioma of parotid gland demonstrating the many blood spaces.

node-bearing area on the affected side. He has been observed for nearly thirteen years without evidence of recurrence.

Case 6. H. D., aged seventy-one, was first seen in February, 1947, complaining of a large swelling on the right side of his face. The swelling had been present since boyhood but had increased markedly in size during the past year. The increase in size of the swelling had been accompanied by severe pain of the lancinating type. He had been unable to sleep for several weeks. He had been seen elsewhere and a diagnosis of a hemangioma had been made and two or three injections of a sclerosing fluid had failed to alleviate his symptoms. The tumor extended from the temporal region, covered the mastoid process, and extended below the angle of the jaw. Pressure caused an increase in the pain and a diminution in the size of the tumor. A bruit could not be heard over the tumor. Because of the vascularity and size of the tumor the patient was advised that removal would probably result in sacrificing the facial nerve with resulting paralysis. This he accepted and with sufficient blood available, surgery was carried out on February 3, 1947. Under intratracheal anesthesia, the external carotid artery was ligated and the entire parotid gland was removed. Bleeding was well controlled by this method and it was not difficult to remove the entire vascular mass down to the masseter muscle. Stensen's duct was ligated. His recovery was uneventful and he left the hospital on the eighth postoperative day. He had a typical facial paralysis, but was entirely free of pain.

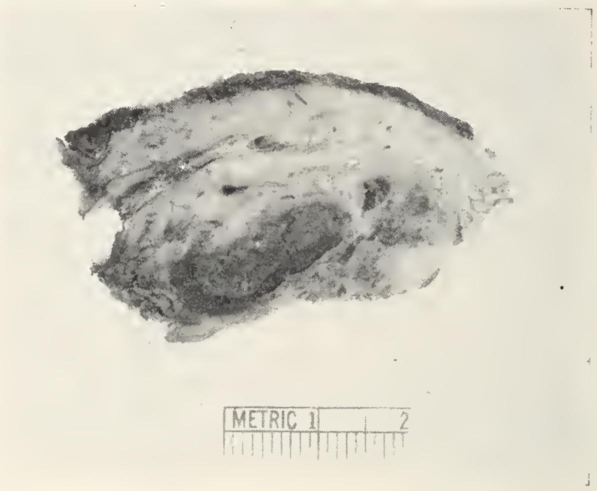

Fig. 16. Cavernous hemangioma of the parotid gland. A small portion of the tumor is shown which shows the extreme vascularity of the tumor.

Pathological diagnosis: Specimen consists of irregular mass of tissue 6 x 5 cm. (Fig. 16). In the center of the mass is an ovoid tumor 3 x 1½ cm. The tumor is hemorrhagic in appearance. The surrounding tissue has the gross appearance of salivary gland.

Microscopically: The tumor nodule is composed of blood spaces most of which are thromboses. The connective tissue contains many foreign body giant cells. These may be the result of sclerosing agent therapy. The whole tumor is surrounded by fairly dense connective tissue capsule. In certain regions the tumor shows cavernous blood spaces. The histological picture is that of a cavernous hemangioma of the parotid gland (Fig. 15).

Diagnosis: Cavernous hemangioma of the parotid gland with partial thrombosis.

On September 15, 1947, strips of fascia lata were inserted under the tarsal plate, upper lip and lower jaw and the loops sutured to the temporal fascia after the method described by J. Barrett Brown. Later a canthoplasty was done and a satisfactory cosmetic result was obtained (Fig. 14).

Hemangiomas of the parotid gland are extremely rare.[8] Those composed of larger vessels are described as capillary hemangiomas.[13] These tumors are benign but often attain considerable size and may invade adjacent structures. They most frequently occur in infants, and often are associated with a superficial vascular nevi. Files of the Registry of Oral Pathology[2] contain only six such tumors in the past twenty years. After a careful search of the literature it is thought that this is the oldest patient ever successfully operated for a hemangioma of the parotid gland.

Summary

1. Parotid gland tumors are rare. The origin of many parotid tumors is not well understood.

2. Mixed tumors of the parotid tend to recur. Even eminent pathologists are inaccurate in being able to predict which ones will recur.

3. Knowledge of the anatomy of the parotid gland will facilitate removal of parotid tumors without injury to the facial nerve.

4. Benign tumors may be treated by simple enucleation. Mixed tumors should be removed by lobectomy and malignant tumors by complete extirpation of the entire gland.

5. Six cases of parotid tumors are presented. One case, a hemangioma of the parotid gland was successfully treated by complete removal of the gland with sacrifice of the facial nerve. Facial paralysis was corrected by the use of fascia lata supporting slings. This case is thought to be the oldest patient successfully treated for a hemangioma of the parotid gland.

References

1. Bailey, H.: The treatment of tumours of the parotid gland with special reference to total parotidectomy. Brit. J. Surg., 28:337-346 (Jan.) 1941.
2. Bernier, J. L., and Tiecke, R. W.: Hemangioma of the parotid gland. J. Oral Surg., 8:171-173 (Apr.) 1950.
3. Brown, J. B., McDowell, F., and Fryer, M. P.: Direct operative removal of benign mixed tumors of anlage origin in the parotid region—with summary of parotid tumors in general. Surg., Gynec. & Obst., 90:257-268 (March) 1950.
4. Ewing, J.: Neoplastic Diseases. 4th Ed., p. 48. Philadelphia and London: W. B. Saunders, 1940.
5. Favata, B. V.: Characteristics of mixed tumors of the parotid gland growing in vitro. Surg., Gynec. & Obst., 86:659-662 (June) 1948.
6. Godwin, J. T., and Colvin, S. H., Jr.: Adenoma of of the parotid gland. Arch. Path., 46:187-189 (Aug.) 1948.
7. Gross, L.: Filterable agent causing mouse salivary gland carcinoma. J.A.M.A., 153:151 (Sept. 12) 1953.
8. Karobin, J. E.: Cavernous hemangioma of the parotid gland. Surgery, 30:367-370 (Aug.) 1951.
9. Kirklin, J. W.; McDonald, J. R.; Harrington, S. W., and New, G. B.: Parotid tumors: Histopathology, clinical behavior and end results. Surg., Gynec. & Obst., 92:721-733 (June) 1951.
10. Klapp, C. T., and Winship, T.: Treatment of mixed tumors of the parotid gland by subtotal parotidectomy. Arch. Surg., 61:477-486 (Sept.) 1950.
11. McCormack, L. J.; Cauldwell, E. W., and Anson, B. J.: The surgical anatomy of the facial nerve with special reference to the parotid gland. Surg., Gynec. & Obst., 80:620-630, 1945.
12. McCune, W. S.: Total parotidectomy. Arch. Surg., 62:715-723 (May) 1951.
13. McFarland, J.: Congenital capillary angioma of the parotid gland. Arch. Path., 9:820-827 (Apr.) 1930.
14. McFarland, J.: The mysterious mixed tumors of the salivary glands. Surg., Gynec. & Obst., 76:23-34 (Jan.) 1943.
15. McNealy, R. W., and McCallister, J. W.: Parotid gland tumors and their surgical management. J. Mich. M. Soc., 50:398-401, 426 (Apr.) 1951.
16. McWhorter, G. L.: Anat. Rec., 12:149-154, 1917.
17. Nelson, C. E., and Deeb, P. H.: Recent trends in the management of parotid tumors. A report of 100 cases. Ann. West. Med. & Surg., 4:683-685 (Nov.) 1950.
18. Quattlebaum, F. W., Dockerty, M. B., and Mayo, C. W.: Adenocarcinoma, cylindroma type, of the parotid gland; a clinical and pathological study of twenty-one cases. Surg., Gynec. & Obst., 82:342-347, 1946.
19. Rawson, A. J., and Horn, R. C., Jr.: Sebaceous glands and sebaceous gland-containing tumors of parotid salivary gland with consideration of histogenesis of papillary cystadenoma lymphomatosum. Surgery, 27:93-101 (Jan.) 1950.
20. Shucksmith, H. S.; Boyle, T. M., and Walls, W. K. J.: Surgery of parotid tumours. Brit. M. J., 2:830-831 (Oct. 6) 1951.
21. State, D.: Superficial lobectomy and total parotidectomy with preservation of the facial nerve in the treatment of parotid tumors. Surg., Gynec. & Obst., 89:237-241 (Aug.) 1949.
22. Utendorfer, R. W.: Parotid tumors. Journal-Lancet, 67:108-116 (March) 1947.
23. Wade, J. S. H.: Neurinoma of facial nerve simulating parotid tumor. Brit. J. Surg., 39:86 (July) 1951.

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RECENT TRENDS IN THE SURGICAL MANAGEMENT OF INFANTILE HYDROCEPHALUS

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INFANTILE hydrocephalus (water on the brain) has intrigued and provoked physicians for many years. Most doctors in their daily practice have had some contact with this condition for it is not uncommon. Murphy found this condition to occur congenitally, with or without spina bifida, in approximately 200 children per 100,000 births. Others have indicated an even higher incidence. The literature on the diagnosis and treatment of hydrocephalus is vast, but, in general, only two main types of hydrocephalus are recognized. These two types are the obstructive and the communicating.

The first of these, obstructive hydrocephalus, in which the ventricles of the brain do not communicate with the cerebrospinal subarachnoid pathways, is most commonly associated with neoplasms of the brain, and its relief is usually obtained by direct attack on the tumor. In those cases in which the disease is not due to neoplasm, congenital atresia of the cerebral aqueduct, congenital bands, hemorrhage, infection and so forth have been described as causative agents. Relief by surgical means will be discussed later in this paper.

Communicating hydrocephalus, in which the cavities of the brain and the pathways of the subarachnoid cerebrospinal fluid are in free continuity, is usually of congenital origin, but may follow inflammation of the meninges. Since the advent of the various chemotherapeutic agents, it has become evident that among children secondary hydrocephalus due to obliteration of the basilar cisterns may be one of the unexpected complications which develops after successful treatment of an acute phase of meningitis with antibiotics.

Classically the treatment of hydrocephalus in children has fallen into two categories. First, attempts have been made to reduce the total quantity of cerebrospinal fluid produced by coagulation or avulsion of the choroid plexuses of the cerebral ventricles,[1,8,11] dehydration, repeated spinal or ventricular punctures, ligation of the carotid arteries[3] in the neck, sympathectomy and so forth.[10]

The second general method for the relief of infantile hydrocephalus has been the attempt to divert the cerebrospinal fluid from its customary subarachnoid pathways to other depots. In this regard it is of interest to realize that it is relatively difficult to devise a "new" operation for the treatment of hydrocephalus, for over the years practically every body cavity or space, potential or real, has been utilized in the attempt to arrest the inexorable progression of this condition.

Spontaneous remission of severe hydrocephalus does occur and cases have been reported. In cases of severely progressive disease, however, it is hazardous to wait or hope for spontaneous remission since the disease can rapidly develop to a point where surgical intervention will not help.

The clinical appearance of the hydrocephalic child need not be described here for it is familiar to all physicians. In the first few weeks of life hydrocephalus in the early stages of development may not be easily detectable. More than one discerning doctor has found the successive routine measurements of the head on postnatal visits to be revealing.

The attempt to reduce the production of cerebrospinal fluid has, except in a few hands, been unrewarding. Diversionary operations have been common for, to all intents and purposes, the problem of the surgical treatment of infantile hydrocephalus seems a simple one. Yet over the

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Read at the meeting of the Southern Minnesota Medical Association, Mankato, Minnesota, September 14, 1953.

The Mayo Foundation, of which Major Mendelsohn is a Fellow in Neurosurgery, is a part of the Graduate School of the University of Minnesota.

The opinions expressed in this publication are not necessarily those of the Department of the Air Force or Department of Defense.

Dr. Dodge is from the Section of Neurologic Surgery.

years, except for sporadic successes, most therapeutic attempts have yielded discouraging results.

The most commonly employed procedures for the relief of hydrocephalus resulting from an obstruction of the ventricular pathways (obstructive hydrocephalus), except direct attack on a neoplasm, have been third ventriculostomy, that is, the establishment of a communication between the third ventricle of the brain and the basal subarachnoid cisterns,[12] and detour operations. In the detour operations the cerebrospinal fluid has been routed from the lateral ventricles in the brain to the cisterna magna in the posterior cranial fossa[14] (Fig. 1), to the mastoid antrum[7] (Fig. 2), to the pleural[4] or peritoneal cavities[9] (Fig. 3), or to the ureter after removal of the ipsilateral kidney.

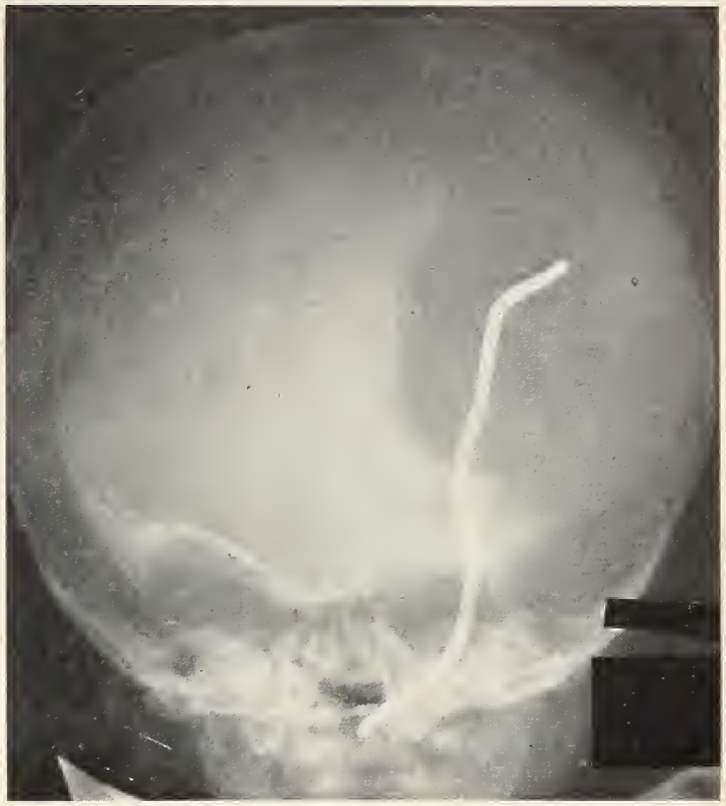

Fig. 1. Ventriculocisternal shunt.

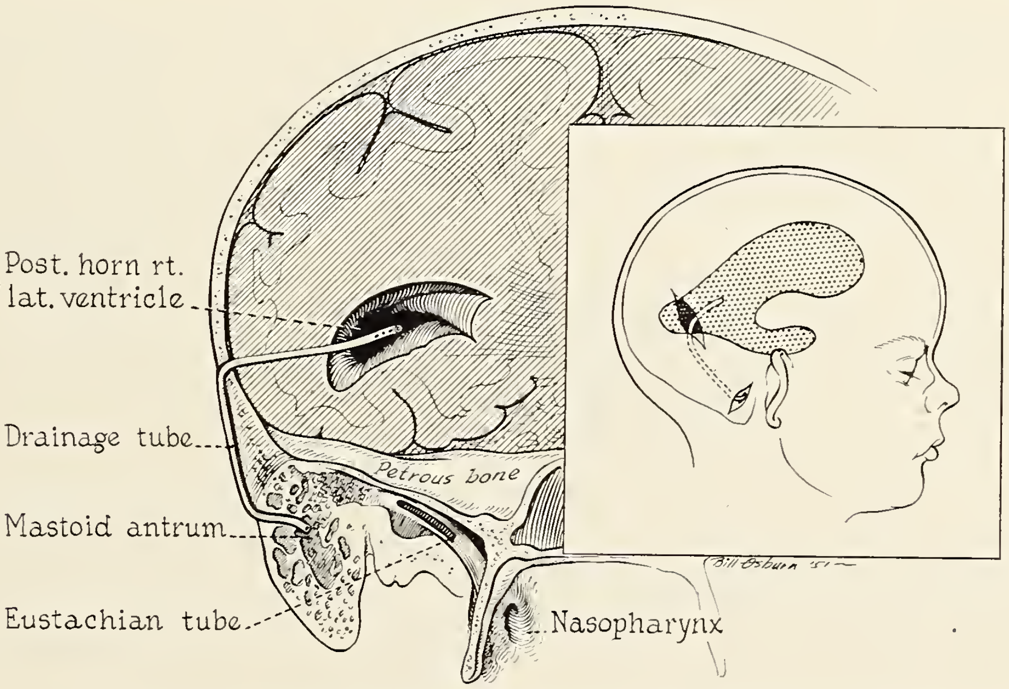

Fig. 2. Ventriculomastoid shunt, modified from Nosik. (From Svien, H. J., Dodge, H. W., Jr., and Lake, C. F.: Ventriculomastoid Shunt in the Management of Obstruction to the Aqueduct of Sylvius in the Adult: Report of Case. Proc. Staff Meet., Mayo Clin., 27:215-218 [May 21] 1952.)

For communicating types of hydrocephalus in which diagnostic ventriculography, dye tests and encephalography demonstrated free communication between the ventricular system and the cerebrospinal subarachnoid pathways, the same ventricular short circuits to extra-nervous depots have been employed, or other methods of diversion were devised from the spinal subarachnoid space to the subcutaneous tissues of the back (Seton)[13] (Fig. 4), vertebral body,[16] retrohepatic space, omental bursa (Fig. 5*a*), peritoneal cavity,[2,5,15] and ureters (Fig. 5*b*). This is done because although the ventricles and subarachnoid spaces are in free communication, the cerebrospinal fluid does not seem to be adequately absorbed or to gain egress from the nervous system.

Although the large majority of these procedures has been tried in the past with only meager reward, recently a combination of factors has resulted in renewed interest in the treatment for hydrocephalus and an awakening of effort has occurred.

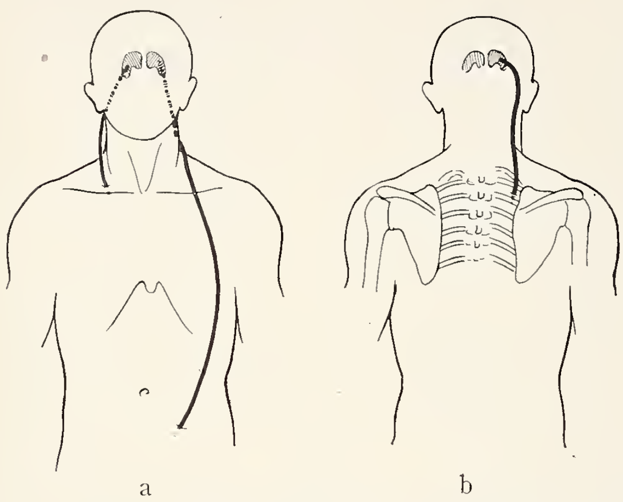

Fig. 3. (*a*) Ventriculopleural shunt (through the supraclavicular space), and ventriculoperitoneal shunt; (*b*) ventriculopleural shunt (through the second intercostal space posteriorly).

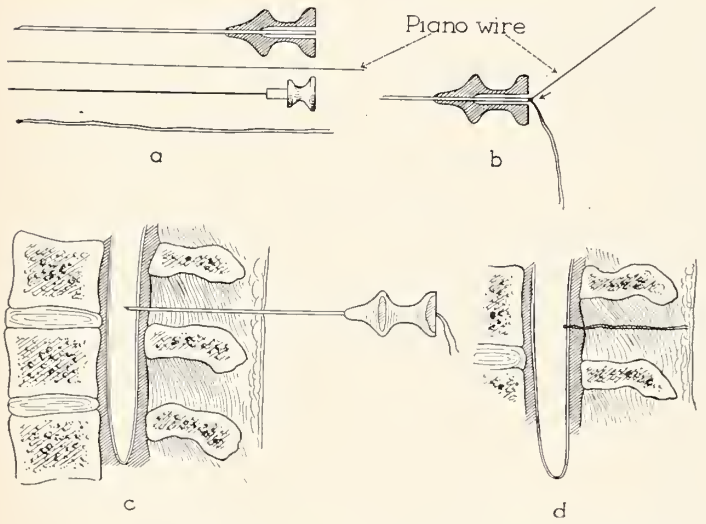

Fig. 4. Seton procedure: (a) Seton needle with stylet and knotted silk string; (b) method of inserting silk; (c) needle and knotted silk in place in the subarachnoid space; (d) knotted silk in place in the subarachnoid space and subcutaneous tissues after withdrawal of needle.

Since the last great war improved surgical techniques, new materials (such as plastics), a wider therapeutic spectrum for antibiotics, more adequate anesthesia for children, regulation of blood and electrolytes, and more enlightened nursing and supportive care have encouraged renewed surgical effort.

The increasingly wide cognizance that progressive enlargement of the head in the first year of life can be due to brain tumor or subdural hematoma, has tended to remove many patients having these conditions from the large indeterminate group of patients having progressive hydrocephalus. Many infants having subdural hematomas, and some infants having certain types of brain tumors or congenital cysts may be spectacularly salvaged by early recognition and surgical treatment.

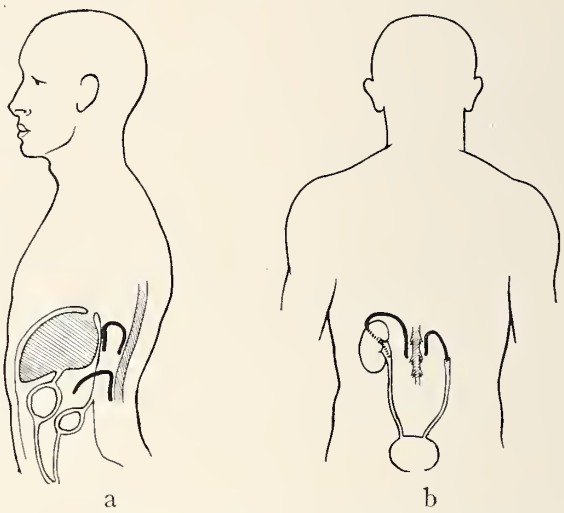

Fig. 5. (*a*) Subarachnoid retrohepatic shunt *(above)* and shunt from subarachnoid space to the lesser omental bursa (*below*). (*b*) Subarachnoid reno-ureteral shunt (experimental) *(left)* and subarachnoid ureteral shunt *(right)*.

An occasional case such as the one reported herein may serve to spur surgeon and clinician alike to greater diagnostic accuracy when confronted with progressive enlargement of the head of unknown etiology. This case is only one example of why these children need to be carefully studied as to the cause of their rapidly enlarging heads.

Report of Case

A baby boy, two months of age, was admitted to the Mayo Clinic because of rapid enlargement of the head since birth. His mother had not suffered any illness during pregnancy. The infant was delivered without instruments after two hours of labor. At birth the circumference of the head was 35 cm. On routine checkup at one month the circumference of the head was 40.5 cm., and at two months it was 45.5 cm. Development had been normal in all other respects up to the time of the child's admission.

Examination showed that the fontanel was bulging and that the cranial sutures were separated. Roentgenograms of the skull confirmed these findings. Bilateral puncture of the fontanel did not reveal subdural hematoma. Results of dye studies were indeterminate. Ventriculography revealed marked thinning of brain substance, but air could not be seen in the third or fourth ventricle. The infant was taken home and returned in one month, during which time the head had enlarged 4 cm.

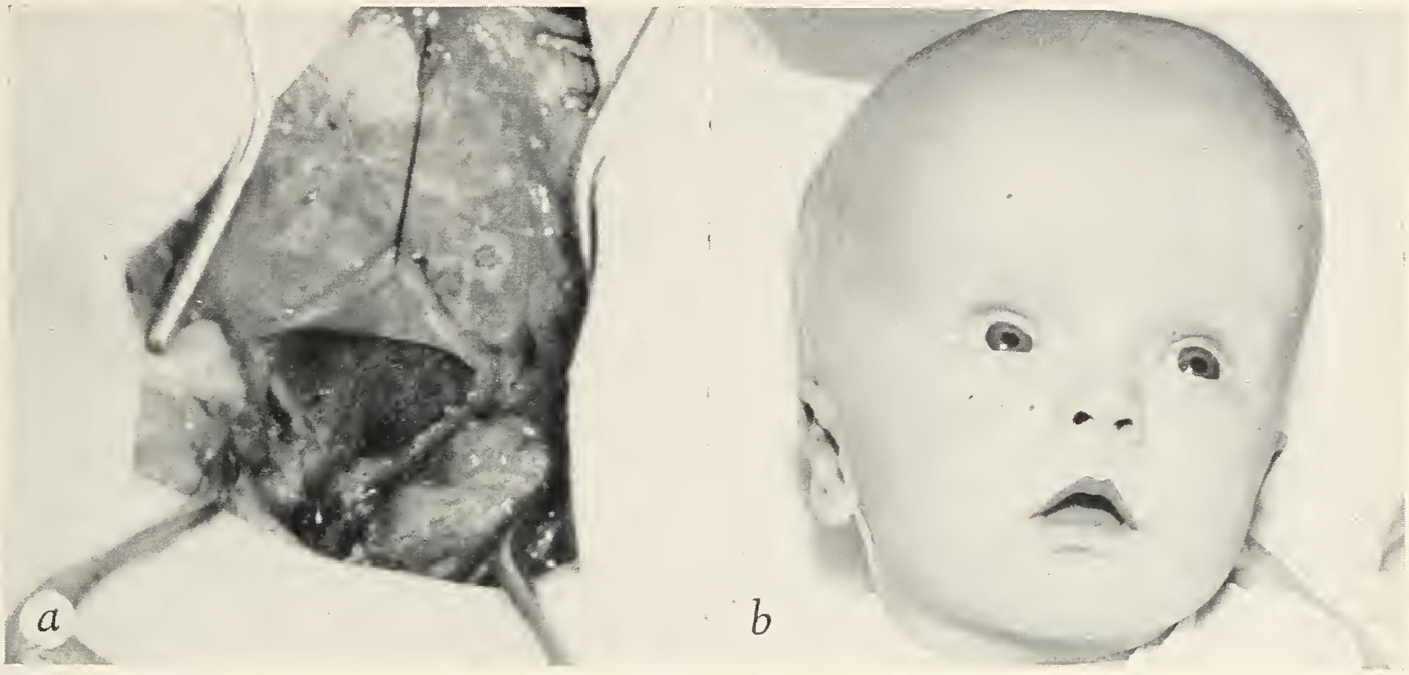

Fig. 6. *(a)* Suboccipital craniectomy showing a congenial cyst blocking the fourth ventricle. *(b)* View of patient after operation.

Suboccipital craniectomy was performed at this time and a cyst was encountered over the fourth ventricle (Fig. 6*a*). This proved to be the dilated roof of the fourth ventricle, the foramina of Luschka and Magendie being absent. The fourth ventricle was then unroofed, allowing free communication between the ventricular and subarachnoid spaces. During the postoperative hospital stay, the fontanel remained soft (Fig. 6*b*).

The last report from the infant's home physician, nine months after operation, indicated that the patient's head had grown at a normal rate for his size and age, and that his general health was good.

Realization that a number of hydrocephalic infants have other anomalies of the nervous system which most probably will not be cured or even ameliorated by relief of intracranial pressure, has tended to lessen the harsh judgment of operative procedures designed to relieve hydrocephalus. When the final analysis of results is considered and made, surgeons should be more willing to modify the "all or none" dictum and should be satisfied with relief of progressive pressure, regardless of whether the success is great or small.

Moreover, the feeling has become more prevalent regarding these pressure-relieving operations, that they are not considered as "cures" *per se,* but that they allow relief from progressive pressure until some sort of spontaneous cerebral compensation can occur. Such a philosophy promotes rather than discourages attempts at therapy.

Unfortunately it is not difficult to predict the clinical result in the case of an unattended infant having severely progressive hydrocephalus; most usually it is disastrous. However, after relief from pressure has been obtained, despite the severity of pre-existing damage, it is often exceedingly difficult to foresee the eventual result. One may be very agreeably surprised when a child with moderate damage to the brain is spared the inexorable march of hydrocephalus and proves in later life to be a relatively useful member of the community.

So, while there are seemingly few "new" operations for the relief of the patient having hydrocephalus, recent trends relative to technique, materiel, diagnosis and thinking on the subject should result in concentrated diagnostic and therapeutic effort, if possible, for each infant afflicted with progressive enlargement of the head rather than abandonment of the patient to the pitiful group of incurable progressive hydrocephalics.

References

1. Dandy, W. E.: Extirpation of choroid plexus of the lateral ventricles in communicating hydrocephalus. Ann. Surg., 68:569-579 (Dec.) 1918.
2. Ferguson, A. H.: Intraperitoneal diversion of the cerebrospinal fluid in cases of hydrocephalus. New York M. J., 1:902 (June 25) 1898.
3. Fraser, J. and Dott, N. M.: Hydrocephalus. Brit. J. Surg., 10:165-191, 1922-1923.
4. Ingraham, F. D., Sears, R. A., Woods, R. P. and Bailey, O. T.: Further studies on the treatment of experimental hydrocephalus: Attempts to drain the cerebrospinal fluid into the pleural cavity and the thoracic duct. J. Neurosurg., 6:207-215 (May) 1949.

(Continued on Page 447)

CONDITIONS MISTAKEN FOR LEUKEMIA IN CHILDREN

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THE familiarity of the lay public with medical terms is nowhere better illustrated than in the case of leukemia, as this is a malignant disease which is particularly prevalent in childhood as well as in the later decades of life (Fig. 1). Many of its victims have received much newspaper publicity which always arouses concern and sympathy among the reading public. Since leukemia is universally fatal, it is important to be certain of the diagnosis before causing parents to suffer the severe anxiety that accompanies the pronouncement of such a disease in their child. A further reason for accurate diagnosis is that the treatment of leukemia, unsatisfactory as it is in saving life, is most effective in alleviating symptoms when begun early in the course of the illness.

There is increasing evidence that the incidence of leukemia is on the upgrade in all age groups. This seems to be true even when allowance is made for the increase that is attributable to better methods of detection and to awareness of the possibility of the disease on the part of both physicians and the public.

It should be pointed out in this connection that with the continued and drastic reduction in childhood mortality from infections there has been a radical change in the relative importance of deaths from cancer as a whole among children. Data published in 1949 for children insured in one large life insurance company[2] show that cancer, including leukemia and Hodgkin's disease, is now second only to accidents as the ranking cause of death in the age group one to fourteen years inclusive and leads all other diseases in the age group five to nine years. These data also indicate that leukemia is the most common type of fatal cancer in children—that it accounts for nearly half of the deaths from cancer in the age group one to four years inclusive, for a little more than two-fifths in the age group five to nine and for more than a third in the age group ten to fourteen. In addition, these data disclose that there has been a significant increase in deaths from leukemia at all ages from one to fourteen years inclusive but especially in the age group five to nine years when the period 1943 to 1947 inclusive is compared with the period 1930 to 1934 inclusive.

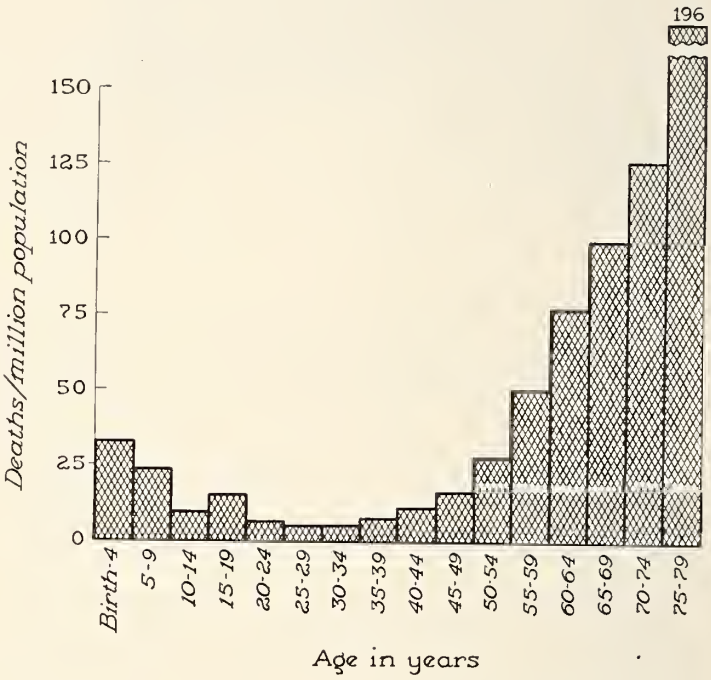

Fig. 1. Age incidence at death from lymphatic leukemia in the white male population of the United States for the year 1949. (Reproduced with permission from: Gilliam, A. G.: Age, sex, and race selection at death from leukemia and the lymphomas. Blood., 8:693-702 (Aug.) 1953.

Sachs and Seeman[3] in a statistical study showed that in the period from 1931 to 1940 there was an over-all increase of 60.9 per cent in mortality from leukemia. In the age group under one year the increase was 75.7 per cent; in the age group one to four years, 55.2 per cent; and in the age group five to fourteen years, 46.1 per cent.

Gilliam,[1] in the most recently available study, has shown that the incidence of lymphatic leukemia in children less than five years of age is greater than at any age up to fifty-five years.

Acute leukemia should be suspected in a child who has been ill for some time with fever, anemia

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Read at the Clinical Session of the American Medical Association, St. Louis, Missouri, December 1 to 4, 1953.

From the Section of Pediatrics, Mayo Clinic and Mayo Foundation.

The Mayo Foundation is a part of the Graduate School of the University of Minnesota.

and enlargement of the liver, spleen and lymph nodes and who may have purpura or bleeding manifestations and leukocytosis with an increase in lymphocytes, many of which may be immature in development. In such cases, however, only careful examination of all the formed elements of the blood as to their quantity and quality may eventually afford the answer, and at times it will be necessary to examine the site of formation of the blood, namely the bone marrow, to arrive at a diagnosis that is not apparent from a study of the peripheral blood. Furthermore, one must remember that the blood of small children reacts with greater lability than that of adults and that the normal values for the various formed elements of the blood differ from those of older patients (Figs. 2, 3 and 4). The variability in the number and type of leukocytes in young children must be taken into account when a child is suspected of having leukemia.

Because of the importance of an accurate diagnosis when the question of leukemia in a child has been raised, it seemed worth while to determine which conditions are most likely to be mistaken for leukemia. For information on this point, I have viewed the records of forty-four children who were seen at the Mayo Clinic in the years 1949 to 1952 inclusive and who were suspected of having this disease but who, in every instance, were proved to have some other condition that was responsible for the findings suggestive of leukemia. All but four of this group of children were less than five years of age and most were two years of age or less.

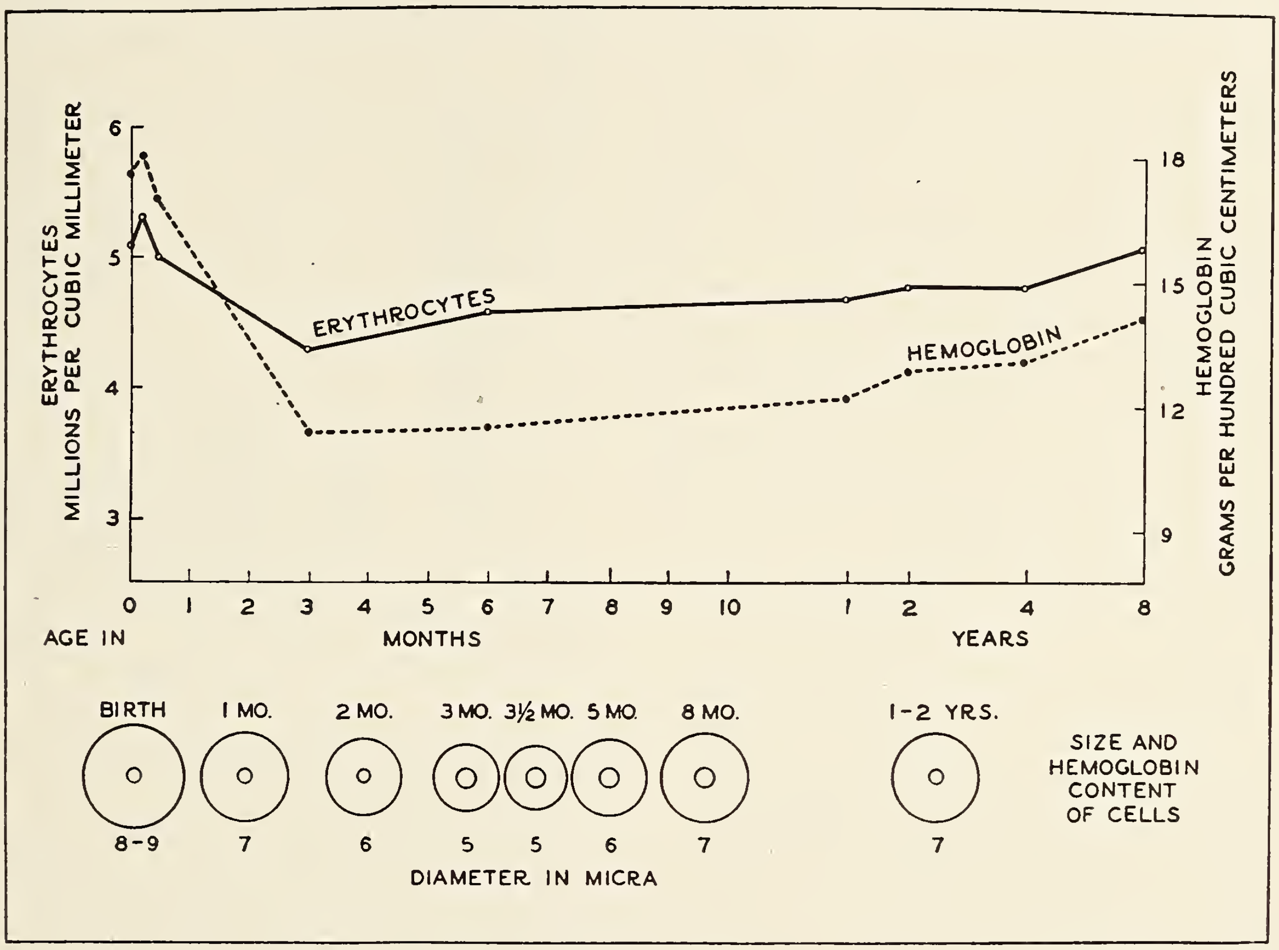

Fig. 2. Normal values for erythrocytes and hemoglobin at various ages in childhood. (Reproduced with permission from: Blackfan, K. D. and Diamond, L. K.: Atlas of the Blood in Chidren. Page 9. New York: The Commonwealth Fund, 1944.)

By grouping the conditions which were present in the forty-four patients suspected of having acute leukemia, it was found that the great majority had an acute infection to which their blood reacted with an unusual increase in total leukocytes or lymphocytes or both (Fig. 5). This is the so-called leukemoid reaction which is familiar to the hematologist and the internist and which occurs in response to certain infections, intoxications, malignant disease with metastasis to bone, and severe hemorrhage. It is well known that pertussis may cause a high leukocyte count with a predominance of lymphocytes, and this infection was found to be the basis of the disturbance in four children, all less than a year of age, one child having 130,000 leukocytes per cubic millimeter of blood of which 80 per cent were lymphocytes of immature types. Others in this group had infections of the respiratory, intestinal and urinary tracts or evidence of sepsis. Five children were proved to be suffering from infectious mononucleosis which is assumed to be of viral origin and may easily be confused with leukemia because of the enlarged liver, spleen and lymph nodes and because of the lymphocytic increase in the blood. Careful study of the peripheral blood smear for atypical lymphocytes of Downey and the presence of a high heterophil titer established the true diagnosis in these cases.

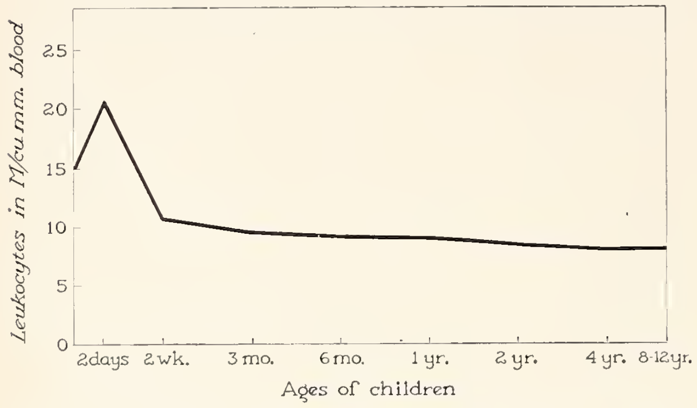

Fig. 3. Normal values for total leukocytes at various ages in childhood. (Reproduced with permission from: Blackfan, K. D. and Diamond, L. K.: Atlas of the Blood in Children, page 8, New York, The Commonwealth Fund, 1944.)

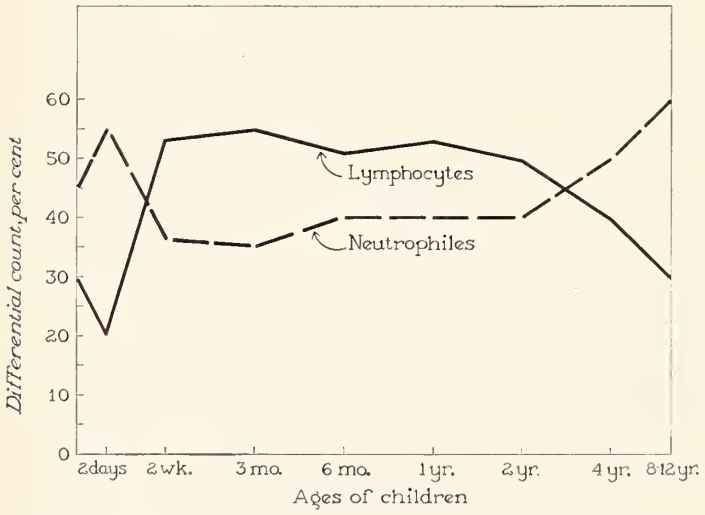

Fig. 4. Normal differential counts for neutrophils and lymphocytes at various ages in childhood. (Reproduced with permission from: Blackfan, K. D. and Diamond, L. K.: Atlas of the Blood in Children, page 8, New York, The Commonwealth Fund, 1944.)

Two children had an interesting condition, described by Smith[4] and called by him "acute benign lymphocytosis," which was characterized by elevated leukocyte and lymphocyte counts without immaturity of the latter type of cell. Both of these patients recovered.

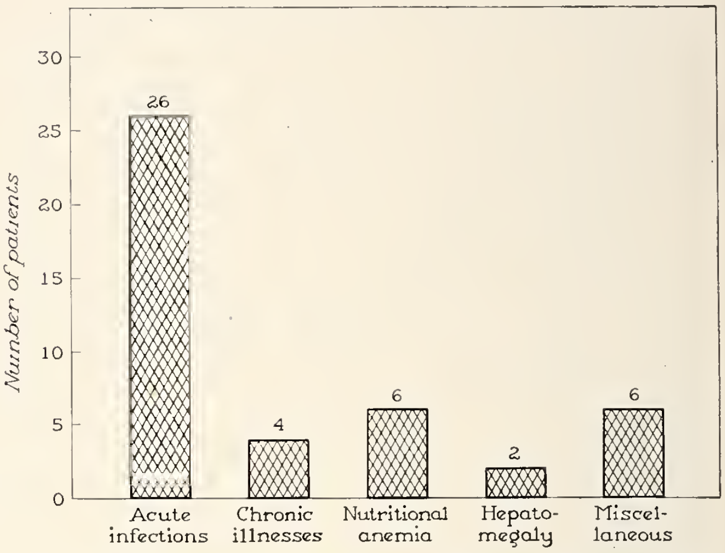

Fig. 5. Conditions which simulated leukemia in 44 children seen at the Mayo Clinic in the period 1949 through 1952.

Four children had chronic illnesses which caused reactions in their blood suggestive of leukemia. One of these children had rheumatoid arthritis, one had infantile cortical hyperostosis and the other two had chronic disorders of the skin, namely, eczema and urticaria pigmentosa respectively, which were secondarily infected.

Six children less than eighteen months of age had anemia classified as nutritional since their diets lacked the ingredients deemed essential in building blood successfully; all responded to improvements in their diets. All habitually drank milk to the exclusion of meat, eggs, cereals, vegetables and fruits. Two children with enlarged livers were found to have a hepatoma and a granuloma of the liver, respectively, but they did not have leukemia.

In the group of children with miscellaneous conditions were a sixteen-month-old child with acquired hemolytic anemia, a two-year-old child with bleeding from the bowel, a two and one-half-year-old girl with a granulocytopenia and relative lymphocytosis resulting from the toxic reaction to an antibiotic drug, a five-year-old boy whose mother feared leukemia because he bled from the site of a penicillin injection, and an eleven-year-old girl with a brain tumor. None of these proved to have leukemia.

In addition to the diseases encountered in this series of forty-four cases, there are other conditions which may be mistaken for leukemia in children. These include (1) the collagen diseases such as rheumatic fever, and infections such as typhoid fever, osteomyelitis, congenital syphilis, viral hepatitis and other viral diseases, and fungous infections; (2) malignant neoplasms, such as neuroblastoma, with metastasis to bone; and (3) intoxications such as poisoning from heavy metals.

It is not always possible to make a definite diagnosis in all children who are suspected of having leukemia. This is illustrated in the case of an eighteen-month-old boy who was referred for examination because leukemia was suspected but who was not in this series of forty-four patients. Repeated examinations of this child's blood and bone marrow failed to disclose the true nature of his disorder, yet he died of leukemia three years later. It must be emphasized that only continued observation and repeated examination of the blood will disclose the nature of the disease in most instances.

Conclusions

When confronted with a very young child suspected of having leukemia because of anemia, enlargement of the liver, spleen and lymph nodes, purpuric manifestations, and abnormal increase of the leukocyte and lymphocyte counts with or without immaturity of the latter cells, one must be exceedingly cautious in making a diagnosis of leukemia because of the extreme lability of the blood of young children in response to certain acute or chronic illnesses, intoxications, malignant diseases, and severe hemorrhage. Careful and continued study of the peripheral blood and bone marrow should enable one to exclude the diagnosis of leukemia when the child actually has some other disease.

References

1. Gilliam, Alexander G.: Age, sex, and race selection at death from leukemia and the lymphomas. Blood, 8:693-702 (Aug.) 1953.
2. Metropolitan Life Insurance Company: Cancer among children. Statist. Bull. Metropol. Life Insur. Co., 30:5-8 (Jan.) 1949.
3. Sachs, M. S. and Seeman, Isadore: A statistical study of mortality from leukemia. Blood., 2:1-14 (Jan.) 1947.
4. Smith, C. H.: Infectious lymphocytosis. Am. J. Dis. Child., 62:231-261 (Aug.) 1941.

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5. Matson, D. A.: A new operation for the treatment of communicating hydrocephalus: Report of a case secondary to generalized meningitis. J. Neurosurg., 6:238-247 (May) 1949.
6. Murphy, D. P.: The etiology of congenital malformations in the light of biologic statistics. Am. J. Obst. & Gynec., 34:890-897 (Nov.) 1937.
7. Nosik, W. A.: Ventriculomastoidostomy: Technique and observations. J. Neurosurg., 7:236-239 (May) 1950.
8. Putnam, T. J.: Surgical treatment of infantile hydrocephalus. California Med., 78:29-32 (Jan.) 1953.
9. Ransohoff, Joseph, II and Hiatt, R. B.: Ventriculo-peritoneal anastomosis in the treatment of hydrocephalus: Utilization of the suprahepatic space: A Preliminary Report. Tr. Am. Neurol. A., 77:147-151, 1952.
10. Royle, N. D.: Drainage of cerebrospinal fluid in treatment of hydrocephalus, syringomyelia and syringobulbia. J.A.M.A., 110:1264-1266 (Apr. 16) 1938.
11. Scarff, J. E.: Nonobstructive hydrocephalus; treatment by endoscopic cauterization of the choroid plexus: long-term results. J. Neurosurg., 9:164-176 (Mar.) 1952.
12. Stookey, Byron and Scarff, John: Occlusion of the aqueduct of Sylvius by neoplastic and non-neoplastic processes with a rational surgical treatment for the relief of the resultant obstructive hydrocephalus. Bull. Neurol. Inst. New York, 5:348-377 (Aug.) 1936.
13. Svien, H. J., Love, J. G., Dodge, H. W., Jr. and Keith, H. M.: Evaluation of "Seton" procedure in the treatment of infantile communicating hydrocephalus. J. Pediat., 40:298-302 (Mar.) 1952.
14. Torkildsen, Arne: Ventriculocisternostomy: A Palliative Operation in Different Types of Non-communicating Hydrocephalus. Oslo: Johan Grundt Tanum Forlag, 1947.
15. Walker, A. E.: A critique of surgery for hydrocephalus. (Abstr.) Arch. Neurol. & Psychiat., 67: 822-823 (June) 1952.
16. Ziemnowicz, Stanislaw: A new trial of operative treatment of hydrocephalus communicans progressivus. Zentralbl. f. Neurochir., 10:11-17, 1950.

DEATHS FROM PEPTIC ULCER IN MINNESOTA DURING 1952

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DURING the month of September, 1952, a seventy-two-year-old farmer went ninety miles up the North Shore of Lake Superior for a "fast." During the next nine days he ate nothing and drank only water and alcohol. On September 20, he was brought in to a hospital suffering from a perforated duodenal ulcer which had manifested itself twenty-four hours previously. He was moribund on admission to the hospital and died before an adequate examination could be performed.

Perhaps at the other extreme is the story of a seventy-seven-year-old man with cancer of the lung. While he was in the hospital for diagnosis during August, 1952, it was decided that the tumor was inoperable, but before he could be discharged, a serious hemorrhage from a gastric ulcer occurred. He was subjected to an emergency gastric resection during which a large hepatic metastasis was found. He died on the tenth postoperative day from general debility arising from the operation and pulmonary carcinoma.

Between these extremes lie the remaining ulcer deaths in Minnesota for the year 1952. Most of them are far less exciting in their circumstances but it was with a view toward analyzing all of these deaths in order to gain any useful information that they may supply that this study was done.

Source of Information Concerning the Ulcer Deaths

A list of patients probably dying of peptic ulcer in 1952 was obtained from the State of Minnesota Department of Vital Statistics.[2] This basic source of information was supplemented by questionnaires sent out to the attending physicians. One half of the patients were from the Twin City area and the other half were from the remainder of the state. In addition, the records of Minneapolis General Hospital and Ancker Hospital were combed for information which some attending physicians in the Twin City area could not furnish. This was done because of the possibility that many of these patients may have been seen at some time in these institutions on an emergency basis during a bleeding episode. Many local physicians were also reached for information by telephone.

TABLE I. CLASSIFICATION OF DEATHS ACCORDING TO TYPE OF COMPLICATION

Hemorrhage	59	(46%)
Perforation	36	(28%)
Perforation with hemorrhage	7	(5.5%)
Obstruction	17	(13%)
After elective surgery	10	(8%)
Total	129	(100%)

From the Department of Vital Statistics an original list of 143 patients who may have died as a result of peptic ulceration was drawn up. On further investigation eleven of these were found to have died of other causes and three patients were in the doubtful category. Consequently in the final analysis, 129 people died from complications of peptic ulcer in this state in 1952. Two of these had advanced carcinoma, one of the lung and the other of the larynx. If allowance is made for these two, then only 127 patients would have survived the year had they not had ulcers.

We have supplemented the information obtained from the Department of Vital Statistics in 119 of these 129 patients. The remaining ten are lost to followup. Seventy of these 129 patients had other significant although not necessarily fatal diseases excluding the two with cancer. These were for the most part arteriosclerosis in one form or another, diabetes, or emphysema. While a few had serious heart disease and were in failure at the time of their complication, they are listed as ulcer deaths.

Causes of Death

Table I shows a breakdown of the deaths into various categories. It is not surprising to see that hemorrhage heads the list. The prominence of

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Supported by the Austen S. Cargill and the Augustus L. Searle Fund for Surgical Research.

From the Department of Surgery, Medical School, University of Minnesota.

TABLE II. CLASSIFICATION OF DEATHS ACCORDING TO AGE, SEX, AND LOCATION

Age	Average age 65 years (youngest 32 years, oldest 89 years)
Sex	102 males and 27 females
Location	78 duodenal 32 gastric 3 combined 16 unknown
	129 total

TABLE III. CIRCUMSTANCES SURROUNDING DEATH IN 95 OUT OF THE 102 CASES OF HEMORRHAGE AND PERFORATION

Complication	Well Attended by a Physician During Final Episode	Moribund When First Seen	Dead When Seen	Percent Died Without Care
Hemorrhage	48	3	4	13%
Perforation	32	2	0	5.7%
Perforation with Hemorrhage	6	0	0	0%
	86	5	4	9.5%

peptic ulcer as an important cause of death is due mainly to this complication.[4] However, that perforation should follow so closely with 28 per cent of the total, appears unusual in that it is generally felt that the mortality rate for perforation has decreased markedly since the advent of antibiotics. Our mortality rate in this clinic for perforation is 6.4 per cent in cases handled since 1942, with no deaths during the last five years. Perhaps this very reliance on the antimicrobial agents accounts in part for the many deaths from perforation in that today there is a greater tendency to handle this catastrophe without surgery. The non-operative treatment of perforated ulcers utilizing constant gastric suction was abandoned in this clinic in 1935[5] except for early small perforations without pneumoperitoneum or the "forme fruste" type in which a perforation closes over shortly after the onset of pain. The patient with this latter type is often clinically well when first seen but has a small pneumoperitoneum demonstrable on x-ray. The non-operative treatment of large perforations with much free air in the peritoneal cavity and great peritoneal contamination from food carries a very high mortality rate. With safer gastric resection now common, one would also expect to see the deaths from obstruction decline.

TABLE IV. PEPTIC ULCER FATALITIES OCCURRING AFTER STRESS SITUATIONS IN THE 102 CASES OF FATAL HEMORRHAGE, PERFORATION, OR BOTH

	Hemorrhage	Perforation	Perforation with Hemorrhage
Asthma, spontaneous pneumothorax, or Cor pulmonale in failure	3	2	0
Congestive heart failure	1	3	1
Poliomyelitis	1	0	0
Recent fracture	1	1	0
Meningitis	1	0	0
Cerebral Vasc. Accident	1	0	0
Peritonitis	1	0	0
Total Incidence	9 (16%)	6 (17%)	1 (14.3%)

Age, Sex, and Location of the Ulcers

Table II bears out the well known facts that peptic ulcer deaths are more common in elderly individuals and that it is a more frequent disease in males. All types of complications follow this general pattern concerning age, sex, and location except that the average age in the obstructed group was sixty-eight years and that all ulcers in this class were duodenal. The average age of those dying after elective surgery was fifty-four years.

Conditions Surrounding the Management at the Time of Death

One frequently hears of people experiencing fatal upper intestinal hemorrhages or perforations in remote areas far from medical attention. What risk is involved for such catastrophes in people with peptic ulcer symptoms? Out of 102 cases of death due to hemorrhage, perforation, or a combination of the two, information concerning the exact nature of demise was obtained in ninety-five instances. (Table III) Five patients were moribund on admission to a hospital or when they were first placed in a physician's care, and four died before medical aid reached them, a percentage of 9.5 per cent. Typical histories here include patients who never consulted a physician at any time, bleeding while driving an automobile, and the tragic case of the patient who bled to death while receiving a transfusion in an ambulance on the way to a hospital for emergency surgery. At least six of these nine casualties had a history of epigastric distress for some years before the final incident and two had had previous hemorrhages.

TABLE V. PREVIOUS COMPLICATIONS 111 OUT OF 129 CASES

Hemorrhage	23
Perforation	10
Esophagitis with obstruction	1

Stress and Peptic Ulcer

That stress situations may activate a dormant peptic ulcer or instigate conditions leading to acute ulceration is well known. Sixteen fatal hemorrhages or perforations occurred after the development of stress situations. (Table IV). Asthma and allied respiratory crises appear to be particularly serious offenders. Cortisone may have been instrumental in two of these, and in two of the nonrespiratory induced situations, renal insufficiency and the bleeding tendency which often accompanies it may have played some part. Four of the sixteen had a definite ulcer history, seven had no past history, and this factor is unknown in the other five.

Past History of Peptic Ulcer

As one can see in Table V, over ⅔ of the 111 cases in which full information is available, died as a result of their first complication. Of the thirty-four patients with a history of hemorrhage or perforation, the majority had only one episode of one or the other in the past. None of the ulcer victims had a modern gastrectomy before the final episode; however, eight out of 120 patients did have some previous gastric surgery as follows: six had earlier perforations closed, one had a gastrojejunostomy, and one had a 50 per cent gastric resection. No one in the entire series had a vagotomy.

The majority of patients had some warning that a fatal accident might happen in that ninety-two of the 114 cases in which full information is at hand had a history of ulcer distress. There does not seem to be any correlation between the duration of ulcer symptoms and the severity of the ulcer diathesis. Many patients had histories of ten to twenty years' duration without any significant difficulties only to have a fatal complication occur in 1952, while at least twenty-two patients had no previous symptoms.

The next part of this paper will deal with each group of complications individually, as follows: (1) deaths due to hemorrhage, (2) perforation, (3) perforation with hemorrhage, (4) obstruction, and (5) deaths after elective surgery for peptic ulcer.

TABLE VI. HISTORY OF ULCER IN 114 OUT OF 129 CASES

No previous history	22
History of less than 1 year	21
History of greater than 1 year	71

TABLE VII. TREATMENT OF THE FINAL EPISODE IN PATIENTS DYING OF HEMORRHAGE

Treated conservatively—Had serious associated disease	14
Died without medical care	7
Treated conservatively—No associated disease A	19
Treated conservatively B	2
Surgery emergency	14
Unknown	3
	59 total

Hemorrhage

In the fifty-nine cases of hemorrhage supplemental information was obtained in fifty-six instances. The average age at the time of death, sex, and location of the ulcer are typical of the entire series of 129 patients. (Table II).

As regards a history of peptic ulcer, nine out of fifty-two patients had no previous history before their final episode and thirteen had a history of less than one year. Eighteen out of fifty patients had the previous complication of hemorrhage, usually only one episode, and two had previous perforations. It is interesting to note that fourteen of these twenty who had serious difficulty with the ulcer diathesis in the past were in good health otherwise and consequently could be considered candidates for earlier elective operations which may have been lifesaving.

Table VII illustrates the treatment of the final episode in fifty-six out of the fifty-nine patients with hemorrhage. Fourteen patients who had serious associated disease were treated conservatively. These include very advanced age with generalized arteriosclerosis, serious heart disease, respiratory insufficiency, and peritonitis. The seven who died without medical care were in remote locations when the hemorrhage started or else exsanguinated so rapidly when they were admitted to a hospital that nothing could be done. Nineteen patients with no serious constitutional disease that would have contraindicated surgery were treated with transfusions alone. Another

TABLE VIII. TREATMENT OF THE PERFORATION IN 31 OUT OF 36 PATIENTS

No Surgical Closure	
Those with serious disease which precluded surgery	6
No medical treatment during final illness	3
No associated serious disease which would preclude surgery	12
	21
Surgical Closure	10

two patients who were otherwise healthy did not have surgery but are placed in a separate category because one was undiagnosed and the other did not receive the usual treatment for a bleeding ulcer. Altogether there were ten patients who received no transfusions, too few transfusions, or were misdiagnosed, exclusive of those who died without medical care. This is not at all surprising since the problem of transfusion therapy for bleeding ulcer is often guided by intuition, blood volume studies being not yet generally accessible to assist greatly in this problem.

Fourteen patients died as a result of emergency surgery. Thirteen had a gastric resection and one had a gastrojejunostomy. Heart failure accounted for four of these and technical defects and postoperative complications for the other ten.

Perforation

Again the age, sex, and location of the ulcer in this group is characteristic for all of the deaths no matter what the complication. Information concerning the past history was obtained in thirty-one out of the thirty-six cases of perforation. Eleven of these had no previous history and four had a history of less than one year. Only five people out of the twenty-nine in which the complete history is known had a previous complication. One of these had a hemorrhage in the past, three had perforations, and one had esophagitis with stricture.

While twenty-four out of thirty-six patients in this group had other associated disease, only one was really doomed as he had cancer of the larynx with metastases. Table VIII classifies the treatment of the perforation in thirty-one out of thirty-six cases. Twenty patients died without surgical intervention (this does not mean that they were necessarily handled by the gastric suction technique), but six of these had serious associated disease which precluded surgery such as heart failure, or great debility. Three died rapidly after perforation before anything could be done or were in remote areas away from medical aid. However, twelve of the twenty-one conservatively handled patients probably could have tolerated surgery. Some of the other cases were not diagnosed until post mortem examination. Some such failures in diagnosis are probably unavoidable. Ten patients had surgical closures of the perforations but died in the immediate postoperative period.

Hemorrhage with Perforation

Seven people died with both hemorrhage and perforation. Only one of these had a previous complication, that of hemorrhage.

Information concerning the treatment of the final episode is known in six out of seven cases. The seventh patient had had hemorrhages in the past but circumstances surrounding her final outcome other than that she died as a result of perforation with hemorrhage in 1952 are unknown. Two of the six were subjected to gastric resection as an emergency measure and both of these continued to bleed during and after the operation and died soon in the postoperative period. Of the four who were managed conservatively, one was in an extremely debilitated condition, one was a rather irrascible individual who had his own ideas about ulcer therapy, and there was no ante mortem diagnosis in the third case. The fourth patient was an eighty-two-year-old lady with a gastric ulcer of four years duration but had no other significant disease.

Obstruction

Seventeen patients died from obstruction as a complication of peptic ulcer. All ulcers were duodenal. The average age was sixty-eight years.

Nine out of the seventeen cases had associated disease—mainly arteriosclerotic degenerative in nature. Only three out of fourteen had previous complications; these were perforations. The history of the ulcer diathesis is longer in this group as one would expect. Fifteen out of the seventeen cases had a history going back over one year, and six of these of over ten years duration.

As regards the treatment of the obstruction, eight had gastric resections, and one had a gastrojejunostomy. Four of these died from factors probably attributable to errors in surgical tech-

TABLE IX. TREATMENT OF THE FINAL EPISODE IN THOSE DYING OF OBSTRUCTION

Surgery	9
Conservative	
No medical care until extremis	2
Medical management	5
Unknown	1
	17 total

TABLE X. TREATMENT OF THE FINAL EPISODE OF ALL DEATHS 119 OUT OF 129 CASES

Treated conservatively—Had serious associated disease or very old	24 (20%)
Died without care	11 (9%)
Treated conservatively (A)—No associated disease	35 (29%)
Treated conservatively (B)	4 (3.4%)
Surgery	
Emergency	35 (30%)
Elective	10 (8%)

nique resulting in leakage of the duodenal closure or pancreatic fistulas. Seven had no surgical intervention. (Table IX). Two of these had no regular doctor and were in extremis when first seen. The other five were considered too old for surgery. Their average age was seventy-seven years and two of them had advanced arteriosclerotic vascular disease. The final outcome is unknown in the seventeenth case.

Deaths from Elective Surgery

Ten patients died as a result of elective operations. The average age was fifty-four years. Five out of nine had previous hemorrhage or perforation, and all had a history of peptic ulcer going as far back as twenty-eight years in one case. Gastric resection was the operation performed for all of these cases. All deaths in this group were due to technical factors except for one fatality due to aspiration pneumonia.

Treatment of Final Episode in All Deaths

Table X summarizes the final treatment in 119 out of the 129 cases. The four patients listed under "Treated Conservatively (B)" were either diagnostic problems or were managed in ways other than the usual methods for hemorrhage or perforation. If the mortality rate from peptic ulcer is to be reduced, it seems to be obvious that the large group of patients treated conservatively who had no associated disease which would contraindicate surgery would be a major target for more aggressive management. Early surgery in these cases may have been life saving.

It is also apparent that the deaths resulting from surgical intervention could be decreased. In this series thirty-three gastric resections and two gastrojejunostomies were performed for the final episode. The cause of death in sixteen cases was either a leaking anastomosis or duodenal stump and occasionally pancreatitis. Intraperitoneal abscesses made up a large percentage of the other nineteen cases, while failure to control a bleeding ulcer during and after surgery was a significant factor in at least three cases. The tubular gastric resection for duodenal ulcers as advocated by Wangensteen[6] eliminates the difficulties frequently encountered in closing the duodenal stump in older operations.

Comment

It is well known that the complications of peptic ulcer are more serious in the older age group. The average age in this study was sixty-five years. In many cases in this report it appeared as though the elderly individual was eking out a hazardous existence when suddenly at a most inopportune time a dormant ulcer would perforate or bleed. Perhaps one should enlarge the indications for elective subtotal gastrectomy in the older age groups. According to Cole[1] the mortality rate for such procedures in patients over the age of sixty is actually less than when resection is performed for patients under sixty years of age, with an overall mortality rate of 1.1 per cent. He stresses that as long as there are no serious associated diseases this operation can be done safely in elderly individuals but that emergency operations are tolerated only about one third as well.

Of the 102 persons who died of hemorrhage or perforation in Minnesota during 1952, twenty-five had previous hemorrhages or perforations and one had esophagitis with stricture. There were no serious constitutional diseases or factors in sixteen of these twenty-five which would have made elective surgery dangerous other than age (the average age was 68.8 years at the time of death). Potter et al[3] report a mortality rate of 1.62 per cent for elective gastric resection in patients over fifty years of age. Fifty-seven per cent of all their patients had associated constitutional disease. Five out of the seven patients who died of duodenal obstruction without surgical intervention died because they were considered too old to be operated upon. Admittedly, two of these had advanced degenerative disease.

Summary

1. One hundred and twenty-nine patients died as a result of complications of peptic ulcer in the state of Minnesota during the year 1952.
2. The incidence of fatal complications of peptic ulcer in the older age group is again shown.
3. Seventy-seven patients out of 111 died as a result of their first episode of bleeding, perforation, or obstruction.
4. No case is recorded in this series of 129 patients of any deaths which had an elective modern type gastric resection before the final episode of bleeding, perforation, or obstruction.
5. Stress appeared to be a factor in at least 16 or 16 per cent of the 102 cases of hemorrhage and perforation.
6. In an analysis of 119 of the 129 deaths, forty-five were treated surgically and seventy-four medically. Thirty-five of the latter group had no associated disease which would have contraindicated emergency surgery.
7. Technical factors appear to account for most of the surgical deaths.
8. About one out of ten people with a history of peptic ulcer distress who ultimately die of hemorrhage or perforation experience this fatal complication in situations remote from medical aid.
9. Duration of symptoms is not correlated with the severity of the ulcer diathesis as a cause of death.

Addendum

Gratitude is expressed to the Minnesota Department of Vital Statistics, and to the many physicians who contributed to this data, to the record room staffs of the Minneapolis General Hospital, Veterans Administration Hospital, Minneapolis, and Ancker Hospital for the help they have given to this study.

References

1. Cole, Warren H.: Operability in the young and aged. Ann. Surg., 138:145 (Aug.) 1953.
2. Department of Vital Statistics, State of Minnesota.
3. Potter, W. H., Andersen, M. N., Steward, J. D.: Gastric surgery in the aged. Geriatrics, 7:173, 1952.
4. Wangensteen, Owen H.: The peptic ulcer problem. Minn. Med., 36:924-934 (Sept.) 1953.
5. Wangensteen, Owen H.: Non-operative treatment of localized perforations of the duodenum. Minn. Med., 18:447 (July) 1935.
6. Wangensteen, Owen H.: Evolution and evaluation of an acceptable operation for peptic ulcer. Rev. Gastroenterol., 20:611 (Sept.) 1953.

MANUAL ON REHABILITATION CENTERS

The first authoritative manual of information on the nation's rehabilitation centers has been published by the National Society for Crippled Children and Adults, the Easter Seal Society, according to Lawrence J. Linck, executive director.

"Rehabilitation Centers in the United States," one of the most useful recent publications in the field of service for the crippled, grew out of the First National Conference on Rehabilitation Centers jointly sponsored by the National Society and the Office of Vocational Rehabilitation of the U. S. Department of Health, Education, and Welfare.

The booklet was compiled by Henry Redkey, consultant on rehabilitation centers for the federal department.

All of the centers listed participated in the Conference, which was held December, 1952, in Indianapolis, Ind., under the joint sponsorship of the federal department and the National Society. Chosen as representative of all U. S. centers, they are divided into six groups including teaching and research, hospital and medical school operated, community with beds, community outpatient, insurance, and vocational rehabilitation centers.

The volume contains descriptions and statistics on every phase of rehabilitation procedure. It is available at $1.50 per copy from the headquarters of the National Society, 11 South La Salle Street, Chicago 3, Illinois.

PSYCHIATRIC ASPECTS OF TUBERCULOSIS

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THE IMPORTANCE of emotional factors in tuberculosis has been mentioned by authors since the early days of medical writing. The Hindus[2] as early as 1500 B. C. recognized tuberculosis as a disease entity and stressed the importance of overexertion, sexual excesses, fasting, and grief in the development of tuberculosis. Other notable figures in medicine of the past who wrote of the close relationship between tuberculosis and emotions include Hippocrates and Galen.[2] The relatively modern literature includes many articles relating to emotional factors in tuberculosis. Trudeau[9] implied its importance when he said that the attitude of the physician "is at once reflected to the patient and influences his condition accordingly." Osler[7] is reported to have said, "The cure of tuberculosis depends more on what the patient has in his head than on what he has in his chest." In 1928 a survey[5] was made of the opinions on this subject among some of the best known specialists on tuberculosis. Thirteen of twenty answered. Of these only one felt that the patient's attitude neither aided nor interfered with the treatment; the others agreed that the patient's attitude was a major factor in the treatment of and the prognosis in tuberculosis. Strecker, Braceland, and Gordon[8] studied seventy-five patients, and they felt that in two-thirds of the cases "peace of mind" was the most important factor in recovery from the disease.

Because of the almost universal acceptance of the point that emotional factors are important in tuberculosis, there is no value in spending further time discussing this. It seems more important to see how the patient's personality and emotions affect the disease.

In the literature there are many statements such as the following: "In many of the patients with tuberculosis there is an undercurrent of bitterness and frustration."[8] Another group of authors[3] state their patients showed "anger, irritability, mild to pronounced reactions of either masochism and sadism . . . depression, . . . explosive effects." Jelliffe and Evans[1] present an analysis of a group of tuberculosis patients and state they are childish, selfish, self-centered, irritable, easily angered, caprious with food, dissatisfied, and ungrateful. Other authors stress the positive relationship between guilt[1] and tuberculosis, genius[4] and tuberculosis, sex[2] and tuberculosis, and the like. It appears that the literature contains a number of generalities concerning the peculiarities of patients with tuberculosis, and these generalities in being quoted become accepted almost as facts. In other words, a clinical observation such as "some patients appear to be unaware of the gravity of their illness" has been elaborated into "the fact" that patients with tuberculosis are in general euphoric.

Concomitants

It is unwise to place any segment of the population in a specific psychiatric category, for my observations indicate that the personalities of patients with tuberculosis vary as much as do any other segment of the population. Furthermore, I believe that what is observed in the behavior of tuberculosis patients depends on how their personalities handle the "concomitants" of the diagnosis of tuberculosis, namely: (1) the "meaning" of the disease to each patient; (2) the effect of being in the sanatorium itself; (3) the social implications of a chronic disease such as tuberculosis.

First of all, when the diagnosis is made, the patient is immediately categorized with a severe illness which connotes among other things that he is dangerous, infectious to others, and that he must be separated from people in general, and from his family in particular. In other words, the effects are stigmatizing and punitive. It is not hard to imagine what might happen to a patient who already is concerned with guilt feelings.

Second, the diagnosis of tuberculosis usually leads to hospitalization, and it is quite obvious that this concomitant will affect the patient tremendously. The patient is now in a milieu of

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fellow sufferers. He is in an environment where time is measured in months and years rather than days. He is looked after completely. He has some new worries, it is true, but he has also left many behind. He is fed, bathed, and in general is cared for like a baby. The life here precludes normal experiences which make for maturity in a competitive world. It is clear that any patient's feelings regarding dependency will be involved.

Third, a diagnosis of tuberculosis results in many social implications. Among others, the sick patient is no longer able to take care of himself financially, socially, or sexually. His hopes and aspirations are dashed completely or at least must be postponed. Putting it another way, the patient can no longer expend his mental energy away from himself; and, therefore, it is of necessity directed toward himself.

Normal Reaction

Up to this point, the concomitants of tuberculosis have been discussed, and now it would be well to turn to what happens to the patient who is diagnosed as having tuberculosis. No one accepts the diagnosis with equanimity. Since the disease is a threat to one's security, the usual reaction that is seen is that of depression. There are, of course, good reasons for this reaction. The patient's plans and ambitions must of necessity be altered, his financial status is threatened, his future in general, including such fields as marriage, work, and school, becomes questionable. Yet it is well known that depression is not always observed; what is seen instead are the patient's defenses against these depressive feelings. These defenses may run the full gamut of human experience, from the active defiance and denial of one who cannot tolerate illness to the opposite attitude of contentment found in those personalities who find emotional satisfaction in the passive role of suffering.

At times the patient reacts with defiance or ultracheerfulness. This defiance is seen in patients who are usually seen the first time with serious lung lesions. These are the patients who are overactive, tense, and restless when in bed. The overactivity is probably related to the patient's inability to accept dependency. The ultracheerfulness that is sometimes seen is most likely a psychological defense against an intense fear of death. Others become resentful. They may be formulated in this manner: "If I can't get what I want, I will get mad." Some become apathetic, or "I'm bad: I deserve it." And lastly, there are those who constitute an especially great problem in treating tuberculosis. This group of patients seem to relish the diagnosis with its long period of hospitalization and attached period of inactivity. To them the illness is a solution to their problems. An "acceptable" illness is what they have been waiting for, since up to now their lack of productivity and complaining has not been accepted by their doctors, family, friends, and associates. They have always felt, "I'm really a capable person, well fitted to make my way in life, dependent on no one; but I'm sick and must be excused." However, until being diagnosed as having tuberculosis, their complaints were not accepted. These patients are the ones that are so difficult to rehabilitate as recovery occurs, for a recovery means the patient must abandon his emotionally regressed attitudes and return to useful channels of effort and thinking.

Thus, it is quite clear that no single personality type is observed in patients who become infected with tuberculosis, but all personality types are found. The behavior evidenced by tuberculosis patients is due to the interaction between the personality and what has been referred to in this paper as the "concomitants of tuberculosis."

Pathologic Reaction

In the foregoing, the discussion has been limited to pointing out how the relatively stable personality types react to tuberculosis. How the so called pathologic reaction types respond to a diagnosis of tuberculosis will now be considered.*

The psychoneurotic group which makes up the great percentage of mental illness will be mentioned first. These patients in general are and should be treated by the patient's own physician, and this holds true in the sanatorium. In this group is found the patients who among other things preoccupy themselves with their ability to hold their spouses, with their doubts about sexual potency, and with their feelings of inferiority; and they are the ones who make excessive demands on their family and doctors. Only the more severe patients of this group are seen by the psychiatrist, and some of these incorporate their unique ideas of tuberculosis into their symptomatology. The anxiety hysterics, for example, may become so "scared" that after recovery they will not touch anything and might even wear a mask at all times.** The obsessive-compulsives in the sanatorium are difficult persons to handle because of their trait of stubbornness and their inability to tolerate normally the behavior of others. They are the ones who may complain a great deal that their roommates in the sanatorium are untidy, dull, and the like. The hysterics in the sanatorium are the immature, "theatrical" type, who usually develop severe somatic symptoms toward which they are relatively indifferent. The true hypochondriacs present an unusual problem in that at admission and during active treatment they are in general quite pleasant, co-operative, and not too complaining. This behavior changes abruptly when their doctors hint that discharge is being considered. At this point they start to get innumerable somatic symptoms and they may begin to indulge in behavior which will be dangerous to their chest pathology.

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*Some examples of all these types have been seen during the past two years at Glen Lake Sanatorium, where the author has been the psychiatric consultant.

Alcoholics, as is well known, constitute a major problem in the sanatoriums. These individuals cannot stand the ordinary tribulations of life; so, of course, when they are confronted with the frustrations of sanatorium life, they turn to alcohol more often than they did before.

The psychopaths, like the alcoholics, cause considerable trouble in all the sanatoriums. These are the people who act out their hostility and who, because of their psychopathology, are unable to form any type of doctor-patient relationship at the sanatorium. They are the patients who are always getting into scrapes and blaming others for their getting into them. They are the ones who have the most unexcused absences. And finally, they are the ones who always appear superficially to want help with their emotional problems, but, when it is available, never utilize it.

The schizophrenic reaction types usually manifest themselves with their apathetic, silly, and unexpected emotional responses, with their thinking disorders, and their hallucinations and delusions. Most of the time they are unconcerned about their tuberculosis. The severe schizophrenics must of necessity be committed to a mental hospital. The mild schizophrenics, however, are always about in a large sanatorium population and can be managed there. They may manifest themselves by just being mildly suspicious, queer, or odd.

The manic-depressives may evidence themselves by their disregard for their illness. Their overactivity is a severe complication to the successful management of their tuberculosis and probably is an important contributing factor to their hospitalization for tuberculosis. The severe depressives on the other hand evidence no will to get better, and this, if allowed to go on without psychiatric treatment, will ultimately lead to a poor convalescence.

The seniles' physiological mechanisms as well as their personalities are quite rigid, and thus they are unable to conform to the usual restrictions of the sanatorium. They require different surroundings from the rest of the patients in the sanatorium. If they are not segregated, either they or their roommates will leave against medical advice. It is best to cater to their whims and be somewhat lenient regarding regimentation.

Homosexual personalities find tuberculosis a particularly difficult problem to handle in the sanatorium, for they must of necessity be exposed intimately to those of their own sex. The psychiatric symptoms that may present include simple anxiety, panic, or "Don Juan" or nymphomaniac behavior.

Treatment

How the "normal" and "abnormal" personality types react to tuberculosis have been discussed. The behavior that presents itself is due to the anxiety produced by the interaction of the individual personality with the concomitants of the diagnosis of tuberculosis. Treatment here, as in all psychiatric treatment, is aimed at reducing anxiety. There is, however, a specific problem which is peculiar to patients with tuberculosis. In usual psychiatric treatment the psychiatrist at times attempts to raise the level of anxiety in order to reach the ultimate goal of reduction more efficaciously. This is not done in tuberculosis, for it is a fairly well accepted fact that an increase in anxiety is detrimental to pulmonary tuberculosis.

Although the fact has been stressed that to treat tuberculosis effectively, the anxiety must be

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**A patient of this type is now in Glen Lake Sanatorium.

reduced, this does not mean that this should be done without first getting a relatively clear picture of the personality of the patient. If a personality evaluation is not obtained, what behavior is to be expected from the patient will not be known. For example, it is important to know that in certain personality types it is almost impossible to get any reduction in the basic anxiety level, and, therefore, definitive psychiatric treatment is of limited value. In this category can be mentioned the psychopath and the confirmed hypochondriac. One of the major problems a psychiatrist has at a sanatorium is to emphasize to the house staff that with certain personality types there is little that can be done from the psychiatric point of view at this time. This is not unlike the house staff stating that some lung lesions are still untreatable in spite of all the new therapeutic tools available.

A few principles which are useful to both psychiatric and non-psychiatric personnel in lowering the anxiety level should be emphasized. These principles will be effective in some degree in treating both the so called "normal" and "abnormal" personality types.

1. A good patient-doctor, patient-nurse relationship should be set up. This can be done by letting the patient talk, and then listening with an attitude of serious professional interest—not one of indifference or over concern. The assurance of genuine interest and sympathy captures patients' confidence and secures co-operation. The patient should be led to know recovery is expected, or if that is impossible, that full use of his remaining capacities is to be achieved.

2. The patient should be told the truth about his status. Information should be made available to the patient in language he understands. Patients resent being kept in the dark, and this may lead to their attempting to get information elsewhere which may be inaccurate. Patients who understand the nature of their illness and the reasons why procedures are being carried out are less likely to act against their best interests.

3. The sanatorium should be kept as homelike as possible with a minimum of restrictions. It is well to be liberal and consistent about passes, visitors, and leaves.

4. Male and female patients should not be completely segregated socially.

Summary

Tuberculosis is a disease involving all classes of society, and the same neurotic trends and abnormal personality traits found in any cross section of society will be found in tuberculosis patients. The particular behavior evidenced is due to the interaction of the personality with the concomitants of the diagnosis of tuberculosis, namely:

1. The "meaning" of the disease to each patient.
2. The effect of being in the sanatorium itself.
3. The social implications of a chronic disease.

To treat patients with tuberculosis adequately, a study of the personality with its attached anxieties is a "must". The treatment of the personality is usually what makes the management of tuberculosis difficult; the actual treatment of the organic disease generally is relatively simple.

References

1. Banister, H.: Some psychological observations on adult male tuberculosis patients. Lancet, 1:784, 1930.
2. Fishberg, Maurice: The psychology of the consumptive. M. Rec., 77:654, 1910.
3. Muhl, Anita M.: Tuberculosis from the psychiatric approach—a follow up. Psychoanalytic Rev., 16:397, 1929.
4. Munroe, D. G.: The Psychopathology of Tuberculosis. New York: Oxford University Press, 1926.
5. Peterson, B.: Mental attitude as a factor in the treatment of tuberculosis. Am. Rev. Tuberc., 17:388, 1928.
6. Ralleston, J. D.: Folklore of pulmonary tuberculosis. Tubercle, 22:55, 1941.
7. Shultz, I. T.: Psychological factors in tuberculosis patients. Am. Rev. T. B., 43:557, 1941.
8. Strecker, E. A., Braceland, F. J., and Gordon, B.: Mental attitudes of tuberculosis patients. Mental Hygiene, 22:529, 1938.
9. Trudeau, E. L.: The value of optimism in medicine. Am. J. M. Sc., 140:1, 1910.

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The undetected case of tuberculosis in a mental hospital is a menace to everyone in the community. The patients in the hospital, the employes, the visitors, and the families to whom the patients return are all subject to infection from the unknown case.—ELIZABETH S. KLEITZSCH, *NTA Bulletin*, February, 1954.

Laboratory Aids to Medical Practice

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Rh-Hr BLOOD TYPES AND HEMOLYTIC TRANSFUSION REACTIONS

G. ALBIN MATSON, Ph.D.

Minneapolis War Memorial Blood Bank

HEMOLYTIC transfusion reactions have been caused by incompatible blood factors of various blood-group systems.

The A-B-O System

Although the A-B-O system was the first to be discovered, it still is important in hemolytic transfusion reactions. This is not for lack of technical skill or laboratory know-how. With the highly avid and potent antiserums now available as testing reagents, technical errors by the careful laboratory worker have been reduced almost to nil. Failure to read labels, inadvertent interchange of blood-grouping serums, inadvertent interchange of bottles of blood for two patients, patients with similar or identical names receiving blood meant for the other person and placing the wrong pilot tube on a bottle of blood are some of the causes of transfusion reactions in this system that harrass the transfusionist and laboratory worker. Each hospital should set up a procedure that will reduce to a minimum the possibility of such errors.

The Rh-Hr System

This system offers the most frequent cause of hemolytic reactions in sensitized patients. In this system the causes of hemolytic reactions are often due to technical errors. Typing serums often leave something to be desired in avidity and potency; even with the best of antiserums, the nature of the clumping is much less firm and more transient than is the case in the A-B-O system.

The introduction into Rh techniques of bovine albumin, by increasing the percentage of protein and making possible the demonstration of blocking (albumin agglutinating) antibodies, is nevertheless not without its problems, one of which is the tendency toward nonspecific clumping. Hemolysis in pilot tubes is an unsolved problem; in no serologic work is the absolute necessity of chemically clean glassware more important than in Rh typing and crossmatching.

Most commonly the hemolytic reaction occurs in an Rh negative, Hr_0(d) recipient who has been sensitized against the Rh_0(D) factor. Since the completely Rh negative patient, rr (cde/cde), also lacks the rh′(C) and rh″(E) factors, he may become sensitized against these factors in donors' blood, as well as against the Rh_0(D) factor.

Commonly the Rh negative patient transfused with Rh positive blood is exposed to more than one Rh isoantigen as when Rh_1(CD) or Rh_2(DE) or Rh_z(CDE) blood is given. He may, therefore, form antibodies against several factors simultaneously. However, of the Rh factors, Rh_0(D) appears to be the most antigenic. Because of these differences in antigenicity, the sensitized Rh negative recipient of Rh positive blood rarely develops sensitization to rh′(C) only, or to rh″(E) only, although the presence of antibodies against either of these in combination with antibodies against Rh_0(D) is a common occurrence.

Occasionally, a patient who is positive to Rh_0(D) but negative to rh′(C) or rh″(E) can, through multiple transfusions, develop sensitization to rh′(C) or rh″(E); or a person positive to any or all of the three specificities of Rh can become sensitized to the hr′(c) or hr″(e) factors and presumably, on extremely rare occasions, to the Hr_0(d); or the Rh positive or Rh negative patient may become sensitized by transfusions of blood involving blood factors other than those in the Rh-Hr system, such as A, B, M, S, Kell, Lewis, Duffy, Lutheran, Kidd and the more re-

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cently discovered U factor. No transfusion reactions have been reported from the P factor.

Crossmatching

Sometimes when a patient has received multiple transfusions and has become sensitized against more than one factor, the problem of selecting compatible blood for the transfusion may prove to be vexing. In these cases, it should be possible to demonstrate in the recipient's serum an antibody that clumps the donor's cells but not the recipient's own cells. In order to establish the nature of the isoantibody in such a patient's serum, as complete a typing procedure as possible should be carried out. In order to make a decision in such difficult transfusion problems, the hospital laboratory often finds it necessary to refer the blood to a central blood-grouping laboratory where rare typing serums are available and where a panel of blood specimens is at hand, some specimens containing and others lacking the blood factors in question. Nearly always, however, antigenic incompatibilities can be detected by careful crossmatching tests before blood is given to a patient.

Crossmatching has a dual purpose: (1) to detect intragroup incompatibilities and (2) to check further the accuracy of the A-B-O and Rh typings. Three methods of crossmatching should be judiciously employed in the laboratory of any hospital in which transfusions are done. For the techniques of these tests a good laboratory manual should be consulted. It is my opinion that the best outline of techniques for these tests as well as other blood-bank procedures is the recently published manual entitled "Technical Methods and Procedures of the American Association of Blood Banks," published by the Burgess Publishing Company, Minneapolis.

The three methods of crossmatching are:

1. *The Conventional Saline Test-Tube Crossmatch.*—This is the best check for incompatibilities in the A-B-O system. Moreover, isohemolysis is more easily observed by the saline agglutinating technique.

2. *The High-protein Crossmatch.*—This is the method of choice for detecting the presence of immune antibodies of the albumin agglutinating or blocking variety.

3. *The Antiglobulin Technique, or Indirect Coombs Crossmatch.*—This is the most sensitive method used in crossmatching for detecting isosensitization. It also detects rare antibodies, such as anti-Fy (anti-Duffy) and anti-Jk (anti-Kidd), which are not demonstrable by the high-protein or saline techniques. The antiglobulin method is too delicate and time-consuming, however, for routine work and should be performed, therefore, in (1) persons who have received transfusions in the past or who are receiving multiple transfusions over a protracted period (longer than a week); (2) in women who have been or are pregnant and (3) in patients in whom any type of unexplained reaction has been reported in the past.

It should be pointed out that dire emergencies may not provide sufficient time to do all the grouping, Rh and crossmatching tests. In such emergencies, group O, Rh negative "universal donor" or even group O, Rh positive blood may be used in order to save the life of the patient. A note should be entered on the patient's chart to justify the procedure.

Many blood banks do anti-A and anti-B titrations on group O, Rh negative blood donors and segregate for use as universal donors those who have low titers (less than 1:200). This practice permits ready availability of universal-donor blood for emergencies and exchange transfusions in infants.

Few of us realize that tuberculosis is both a cause and an effect of indigency. It is simple to grasp the fact that poverty lowers resistance so that the disease spreads rapidly when families live on an inadequate or unbalanced diet, are crowded into unsanitary homes, can obtain little education. But we do not always stop to think how directly tuberculosis leads to poverty in families where it did not previously exist. A recent study of a sizable group of patients pointed out that less than 2 per cent of the patients' families were relief recipients at the time of diagnosis; upon being admitted to hospitals a few months later, 16 per cent of the families of these same patients were on relief.—Mary Dempsey, Statistical Unit, NTA, Feb. 3, 1954.

President's Letter

MEET, MEET, MEET—

Sometimes it seems endless. Sometimes we feel that we shall never get through attending meetings. Sometimes we wonder if it is worth it, the time we spend at medical meetings.

But, I would like to point out that it *is* worth while, that we do gain, grow and improve as a result of spending time with our colleagues at meetings of our own groups, and of others too.

Within the past few years, especially, medical meetings have become much more plentiful. More special groups have been organizing, and established organizations have increased the number of meetings during each year. Local groups have increased in size and importance, and have expanded, making it almost obligatory to hold more meetings.

Thus it becomes a matter of selection for the physician. He must choose between attendance at several interesting and valuable sessions. He must also decide how much time away from the office and home he can spare. No wonder he occasionally finds himself in a dilemma of indecision.

A primary example of this situation is seen in the events of this and the past month. The beginning of June marked the 101st annual session of our own state association, featuring many meetings of committees, the Council, the House of Delegates, special groups—not to mention the authentic and interesting discussions in the scientific sessions. Also, many county societies held meetings prior to the Duluth convention.

And, immediately on the heels of the state meeting, the great annual session of the American Medical Association began in San Francisco. Many Association members found it necessary to leave our state meeting directly, and hurry west for the national meeting.

But, such is the life of the busy physician of today. He makes arrangements for the care of his patients if he finds it necessary to be away from his office. His colleagues are ready and willing to fill his shoes temporarily so that he can attend these excellent events, and thus benefit from what are actually postgraduate courses for all physicians. And, most likely, the following year he will arrange with a colleague to reverse positions so that each may enjoy the rich advantages of meeting attendance.

Because of the vast scope of medical knowledge available today, it can be seen that a large number of scientific meetings is one important method of disseminating information to the physicians of the country. And, because it is impossible for each doctor to be present at all such meetings available to him, it is to his lasting advantage to attend whenever possible.

I am sure that your attendance at the state meeting in Duluth proved to be not merely "meety," but meaty—a pun more true than clever. What you gained from it will become a part of your daily practice, and will be of lasting value.

J. Ohage.

President, Minnesota State Medical Association

Editorial

ARTHUR H. WELLS, M.D., *Editor;* HENRY G. MOEHRING, M.D., and JOHN F. BRIGGS, M.D.

PUBLIC EDUCATION—YOUR DUTY

THE MEDICAL needs of the people of America are increasingly yearly in many ways. Those that are obvious because of the extension of life expectancy need not be cited to any physician, such as geriatric care, refined surgical techniques, "wonder" drugs, and psychogenic problems associated with the fleeing tempo of living. However, with this change of living, an increase in means of communication has brought about another problem. One cannot listen to the radio, view television, read a newspaper, or peruse a periodical without being overwhelmed by articles or advertisements extolling the wonders of a laxative, a cigarette, a cold remedy, or a panacea. This lack of information—or worse, these untruths, have presented a new challenge to true and adequate medical care. Patients now often tell the physician what treatment they should receive as pointed out in a lay periodical, or swamp him with requests for a yet unproven remedy or cure which has been quoted by an energetic author. Medicine and health are news!

With these changing times the medical profession must adjust its mode of approach and re-evaluate its position. In making such an adjustment, the "Principles of Medical Ethics" of the American Medical Association states in Section 5:

> "*Educational Information is Not Advertising.*—Many people, literate and well-educated, do not possess a special knowledge of medicine. Medical books and journals are not easily accessible or readily understandable.
>
> "The medical profession considers it ethical for a physician to meet the request of a component or constituent medical society to write, act or speak for general readers or audiences. The adaptability of medical material for presentation to the public may be perceived first by publishers, motion picture producers or radio officials. These may offer to the physician opportunity to release to the public some article, exhibit or drawing. Refusal to release the material may be considered a refusal to perform a public service, yet compliance may bring the charge of self seeking or solicitation. In such circumstances the physician should be guided by the decision of official agencies established through component and constituent medical organizations."

In effect, then, it becomes the duty of every physician for several reasons to contribute his efforts as needed to the health education of the population. This serves: (1) to better the public relations of medicine (and this is always needed); (2) to educate the public (an educated public is reasonable and understanding); (3) to refute dangerous and unethical claims in protecting the public; and (4) to contribute his public service in a way that the population recognizes. The one and only question of past years was that of this being promotional or advertising. The code states that it is not, and established statistics prove that it is not.

Thus, the medical problem of education of the public takes its place in the practice of medicine, informally in the office, and formally in speaking or writing. To quote Dr. W. Bauer, head of Health Education of the American Medical Association, "Not only is the appearance of the doctor in public for public service accepted by the profession, but it is his duty to do so."

JAMES R. FOX, M.D.

IN DEFENSE OF ROBIN HOOD

TWENTY-FIVE years ago, Orphan Annie was being condemned by those with Communistic affiliations because she and Daddy Warbucks were counted as capitalistic propaganda. Several months ago an advisor to the Indianapolis Board of Education recommended that the Robin Hood stories should be banned because he took from the rich and gave to the poor, therefore he is Communistic. Note that the ban was advised, not because Robin Hood violated the common law by forcibly taking someone's property, not because he broke one of the Ten Commandments by stealing, but because he was soft-hearted enough to give what he had taken to the people who were in need. In passing, we wonder in what sort of position such reasoning leaves those active in Community Fund and similar drives.

However, lest we leave Robin Hood with his escutcheon besmirched—we can safely leave our

Community Fund folks to the judgment of their fellows—remember that 600 to 700 years ago the oppressors of the poor were the rich prelates and barons. To be popular, ballads, which were heard mostly by the poor, had to have a hero who turned the tables on these oppressors. Somehow that doesn't lead us to classify the hero as a Communist by whatever definition of Communism. R. Valentine Harris, author of *The Truth about Robin Hood,* points out that it is proven that a Robin Hood followed the Earl of Lancaster in battle in 1322, and that a Robin Hood was in the King's service in 1323-4. Further, that the Robin Hood of the ballads was a God-fearing Englishman who honored his king, regarded the deer as "fair game," and considered those who oppressed the people as his enemies. Rather than ban such a Robin Hood from our society, perhaps we should hope that his brand of leadership will prosper in the world.

H.G.M.

THE CHEMOTHERAPY OF TUBERCULOSIS

TEN YEARS have passed since the first report on streptomycin. During this decade, there have been many intensive investigations of this drug in the treatment of tuberculosis. During the same period, many other antituberculotic drugs have been studied in both laboratory experiments and clinical investigations. As a result, it is now agreed generally by experts in tuberculosis that chemotherapy is indispensable in the treatment of all forms of this disease.

Although much about the chemotherapy of tuberculosis remains unknown, certain basic generalizations can be made and clinical practice should be guided by these. First of all, none of the drugs available today are bactericidal. Secondly, the therapeutic efficacy of each drug is impaired by the occurrence of drug-resistant variants of tubercle bacilli; these resistant microorganisms may became predominant so that further treatment with the particular drug concerned may be of no value. In the third place, the emergence of drug-resistant strains of tubercle bacilli may be delayed or prevented by the administration of two antituberculotic drugs concurrently.

Therefore, the chemotherapy of tuberculosis should consist of the concomitant administration of at least two antimicrobial agents long enough for the defense mechanisms of the host to exert their maximal effects. The available data indicate that the combination of any two of three drugs, namely streptomycin, para-aminosalicylic acid and isoniazid, is satisfactory. The optimal duration of therapy is not known; undoubtedly it varies tremendously depending on the extent and character of the disease as well as certain immeasurable host factors. However, it is agreed generally that antituberculotic chemotherpay should be continued for a minimum of eight to twelve months, and in many instances the treatment should be kept up for several years.

In a consideration of the effect of drugs in tuberculosis, it is of value to divide the disease into reversible and irreversible parts. Medical treatment and collapse therapy is recommended for the reversible component and surgical excision for the irreversible residue whenever the latter is of clinical significance. The greatest problem at the moment is to determine which residues are of sufficient importance to warrant resection.

It should be emphasized that in spite of the development of effective chemotherapy other forms of treatment are needed to control the disease in most patients. Rest in bed and collapse therapy are still invaluable, as is adequate nutrition. For these, as well as for the education of the patient and the protection of society from a contagious disease, the sanatorium or the hospital remains the best place for the treatment of most tuberculous patients. However, it is becoming increasingly evident that it is not only impossible but also unnecessary to keep patients in a sanatorium throughout the long period required for chemotherapy.

Therefore, it is necessary for general practitioners and internists to acquaint themselves with the latest developments in the treatment of tuberculosis, especially the use of antimicrobial drugs, so that they can continue to do their share in the control of this disease.

David T. Carr, M.D.

VIRAL HEPATITIS

REPORTS of cases of infectious hepatitis have been increasing in Minnesota for the past ten years. The nation, as a whole, has experienced a similar increase. During the year 1953, there were 1192 cases with thirteen deaths reported to the Minnesota Department of Health. Cases have been reported from all sections of the state with a concentration of cases in Little Falls and vicinity. Infectious hepatitis is also known as infectious or catarrhal jaundice, and is caused by a filterable virus. The disease is most common among children and young adults, and is seen most frequently in autumn and winter.

Infectious hepatitis is an acute infection with variable symptoms including fever, loss of appetite, nausea with or without vomiting, headache, lassitude, fatigue, and abdominal discomfort with a feeling of fullness in the upper abdomen. In many cases after the fever has subsided, the urine may become dark due to the presence of bile, and the patient may become jaundiced. Occasionally, there may be a chronic impairment of liver function. Usually the white blood count is low and liver function tests show impairment of the liver function. Most cases have a benign course with many so mild that they are not recognized. Rarely acute fulminating cases occur terminating in acute yellow atrophy of the liver.

Clinically infectious hepatitis is indistinguishable from serum hepatitis (homologous serum jaundice). The chief points of difference are the incubation period and the mode of spread. The incubation period in infectious hepatitis is from fifteen to thirty-five days, the average being twenty-five days. In serum hepatitis the incubation period may be as long as six months; probably twelve to fourteen weeks is most common. In serum hepatitis, the mode of spread is by inoculation of infected blood or blood products and by use of needles and syringes contaminated with traces of blood from infected persons. Both infectious hepatitis and serum hepatitis are caused by viruses that are closely related. The causative agent of infectious hepatitis is designated Virus A and of serum hepatitis Virus B. Both viruses are relatively resistant to heat but are destroyed by autoclaving or by boiling for fifteen minutes. Virus A (infectious hepatitis) is present in the blood for a short while during the acute stage of the disease and in the stools for a longer period of time. The virus has not been demonstrated in nasopharyngeal secretions or the urine. Virus B (serum hepatitis) is present in the blood of infected persons and has been known to persist for as long as five years.

Virus A (infectious hepatitis) is spread by the fecal-oral route. Respiratory spread and spread by contaminated blood is apparently negligible for infectious hepatitis. Food and waterborne epidemics have been reported. Fingers, food, and flies are important in the spread of infectious hepatitis.

Control measures are dependent on good personal and community hygiene. Cases should be reported to the local health officer and the State Department of Health as required by regulation 301. The case should be isolated for the first week of illness. Bowel discharges should be properly disposed of. Outdoor toilets should be kept clean and fly proof. Hands should be washed carefully with soap and water after use of the toilet, before handling food and before eating. While respiratory spread probably is not important, it is advisable to dispose of nose and throat discharges in a sanitary manner. Early medical care is essential to guard against complications. Children should be excluded from school until clinically well. Quarantine of contacts is not indicated.

Immune serum globulin (gamma globulin) has proven effective at least as a temporary preventative for infectious hepatitis (Virus A) and will stop the occurrence of cases where there is close continued contact if the exposed individuals are injected before the last week of the incubation period. The Minnesota Department of Health distributes gamma globulin for this purpose to the family physician. The recommended dose is .02 cc. per pound of body weight with a maximum dose of 2.0 cc. per individual. There is no age limit for the protection. While the disease is uncommon after the age of forty, the severity of infectious hepatitis is more marked in the older age group.

Infectious hepatitis cases in Minnesota have increased since the five-year period 1934-1938 with seventy-two cases and six deaths to a total of 2,071 cases during the years 1949-1953 with forty-five deaths.

Preventive measures should do much to lessen the occurrence of this disease.

To prevent any possible spread by stylets, needles or syringes, they should be thoroughly sterilized by heat sterilization. It should be realized that in giving subcutaneous intramuscular or intravenous injections the syringe may be contaminated by traces of blood drawn into it.

D. S. Fleming, M.D.

The Dean's Page

A REPORT ON MEDICAL EDUCATION IN KOREA

During the latter part of the past winter, three members of the University faculty visited Korea at the request of the Foreign Operations Administration to determine what might be done to assist the National University of Seoul to rehabilitate and strengthen its programs in agriculture, engineering, and medicine. Dr. Gaylord Anderson represented the Medical School in this study and was accompanied by Deans Harold Macy and A. F. Spilhaus in agriculture and engineering, respectively. Dr. Anderson's report has been of real interest to all of us.

The Medical School at the Seoul National University is almost identical in size with that at Minnesota and provides instruction of physicians and nurses. Established about fifty years ago, it remained under Japanese domination until 1945. In accordance with this policy—which is far from unique in the history of colonialism—the Japanese occupied all the key positions. As they viewed the school's purpose to be the training of Koreans to give medical care to Koreans, no attempt was made to develop a faculty of Koreans who had had special postgraduate training for their teaching responsibilities.

As a consequence of this policy, the liberation in 1945 and expulsion of the Japanese found the School forced to pull together a faculty, most of whom had had no formal instruction beyond that which they had received in their own school as undergraduates. Few had ever studied outside of Korea.

With the advent of war barely five years later, the School was forced to take refuge in Pusan where it operated in makeshift quarters until the fall of 1953. With the war came the capture of several of the key members of the faculty, who were unable to escape to Pusan and are now thought to be dead. Others went into the ROK army and are still on duty.

For four years the remains of the staff operated a medical school as best they could. All equipment had been lost. Laboratories were not available and hospital equipment was limited to what could be loaned from the United States Army sources.

No library was at hand; Japanese texts were banned; Korean texts non-existent and English texts, even if available, were understood by few of the students. Thus reliance was placed almost exclusively upon didactic lectures which have always assumed a more important role in the Oriental medical curriculum than they have in ours.

The return to Seoul in the fall of 1953 meant only a new stage of improvisation, for the United States military forces did not vacate the Medical School and University Hospital compound until March 1, 1954. The return of the buildings meant nothing more than the term implies for those are mere buildings and nothing more. Four years of military occupation. during which the city changed hands four times and was occupied for extensive periods by the Communists invaders, have taken their toll upon the School and Hospital buildings. Though ample in size and firm in construction, they stand completely bare of anything resembling equipment for either teaching or the care of patients. All equipment has been lost, laboratory benches and plumbing ripped out, and physical damage done to the buildings.

Such is the situation faced today by this Medical School. Both school and hospital must be completely re-equipped. The latter has started to operate on a reduced scale with surplus equipment provided by the United States Army, but this provides only the bare essentials. The school must start from the bottom for it lost essentially everything. The faculty is intensely sincere and devoted to its task, but fully realizes the limitations of its background and that for four years of war it has been out of touch with medical developments except for its contacts with foreign military units. Even today they are devoid of books and medical journals. Furthermore, they have inherited a system of medical education that emphasizes the formal lecture and makes little provision for clinical study as we know it in the United States. The curriculum of the junior year provides for only an hour a day of clinical work and in the senior year an hour and a half. Hospital facilities for more time are not easily obtainable.

The rehabilitation of this school obviously requires equipment and staff education. The former is under order and will arrive slowly. The latter will take time. It means study outside of Korea for the senior staff so that they may not only gain further technical knowledge but also learn methods of clinical instruction. For the younger staff, who will be the teachers of fifteen to twenty years hence, it means several years of postgraduate fellowship; and for those who cannot leave Korea, it means an intensive local instruction in modern medical developments. Such are the needs of this school.

Harold S. Diehl, M.D.
Dean of the Medical Sciences
University of Minnesota

OVEREATING—THE ROOT CAUSE OF WARS

In the Golden Age, i.e., at a period when all was righteousness and when there was no competition for food, everyone ate just what one wanted and everyone was happy. But as time passed, some people ate a little too much, simply because it was available. The result was that their bodies became heavier. On account of the body becoming heavier, they became tired after performing their usual work. On account of weariness, they became lazy. On account of laziness, they developed a desire to accumulate food and articles for future use, i.e., hoarding. On account of the desire to accumulate, there arose possessiveness. On account of possessiveness, there arose greed.

In the second stage, on account of greed, the desire arose to accumulate more by deceiving others. On account of this desire to deceive others, there arose falsehood, and falsehood gave rise to many passions, such as lust, anger, vanity, hatred, haughtiness, injury, fear, excitement, greed and mental perturbation, et cetera.

The leaders who are thus perturbed in mind lead their followers into unrighteous conduct. On account of greed, anger and delusion, they insult weaker people and have recourse to violence with deadly weapons, ending in wars between countries, clans and races. They thus either destroy others or are destroyed by others in the struggle. In this manner, unrighteous conduct which starts with greed in slight overeating, leads to wars between nations.

(Theory of the cause of wars by Charaka, a medical authority of India, who is supposed to have flourished in the first century A.D.)

Medical Economics

Edited by the Committee on Medical Economics
of the
Minnesota State Medical Association
George Earl, M.D., Chairman

PENTAGON CITES M.D. SECURITY RISKS

With all the recent hullabaloo about security risks, and the famous committee hearings conducted in Washington, it is interesting to note that the Secretary of Defense has stated that fifty-one physicians or dentists have been refused commissions in the Army for security or other reasons.

As small as this figure may seem, it could be classed as "alarming" by those concerned with the feelings and policies of the medical profession.

According to an article in a recent issue of "U.S. News and World Report," which reports on the hearing on S.3114 before the Senate Armed Services Committee, Secretary of Defense, Charles E. Wilson, revealed these things: "Fifty-one physicians or dentists, called in the draft, have failed to answer loyalty questions satisfactorily or are otherwise unfit for commissions. The armed forces still want to use them, but as the lowest-paid privates, if the law is changed. Other security risks are known and being watched."

To Rout Out Risks

Secretary Wilson stated that "The Department of Defense is undertaking, through administrative action, to tighten up the procedures that deal with Communists and security risks to carry out the intent of Congress." He testified:

> "It was found that the loyalty certificates, although required of officers and enlistees before acceptance, were delayed for inductees until after induction. This is being changed to require certificates to be filled out before induction by all personnel the same as is required in enlistments and in appointment to commissions."

Secretary Wilson explained further in his testimony that only twenty of the fifty-one doctors are considered to be serious security risks, and that the remainder "have something about them that they do not qualify as officers." He pointed out that the doctors were all inductees, and had requested a commission. The Armed Services committee was conducting the hearing in order that certain changes in these regulations could be drawn up.

Pending these requested changes, Secretary Wilson had issued the following directive: "The Department of Defense has recommended to the Congress an amendment, S.3096, to the Doctor Draft Act. Pending action by the Congress, and until further notice, no physician or dentist who has been called under the Doctor Draft Act and who has been denied a commission shall be inducted into the service as an enlisted person unless he can qualify for a commission pursuant to the standards for security and loyalty prescribed by the military department concerned."

STRANGE BEDFELLOWS ON REINSURANCE PLAN

The line-up of pros and cons on the Administration's proposed plan for reinsuring present health insurance plans, shows a strange assortment of perennial opposites of national groups

Those favoring the plan include the national Blue Cross Commission, the American Hospital Association, and Dr. George Baehr who is president of Health Insurance Plan of New York City, and the Physicians Forum, Inc. A representative of the Farm Bureau Insurance companies said "We wish it were available."

Those against the plan, or favoring revision and improvements, include the American Medical Association, national Blue Shield, the United States Chamber of Commerce, the American Dental Association, the American Federation of Labor, the Association of Casualty and Surety Companies, the Bureau of Accident and Health

Underwriters, and the Health and Accident Underwriters Conference.

Hearings on the proposal brought out many thoughts, both pro and con, about the bill and its purposes. As reported in the AMA Washington Letter, John H. Miller, vice president and actuary of the Monarch Life Insurance Company, appeared in behalf of the last three groups named in the above paragraph, actually supported the general objectives of the program, but could not endorse the bill as it stands. He stated: "(1) The principles of reinsurance are the same as the principles of insurance; reinsurance cannot be applied to a risk that is not insurable; reinsurance does not help to sell insurance, nor does it reduce the cost of insurance. (2) Direct assistance to low-income groups on the local level is to be preferred to the disguised federal subsidies contemplated in the reinsurance plan. (3) It is important that the public not be misled into believing that reinsurance is a panacea, because reinsurance does not 'enhance the power of insurance' to reach those who are not now covered."

William S. McNary, vice president of the Michigan Hospital Service, was official spokesman for both the Blue Cross Commission and the American Hospital Association. His endorsement was limited to the purposes of the House bill on reinsurance. He recommended that the bill should be amended in three aspects:

1. Regulations promulgated by the Secretary should have the approval of the Health Service Council.
2. Confidential information for statistical purposes from a carrier should not be released without the approval of the individual carrier.
3. The language dealing with the publicity regarding the reinsurance plan should be rewritten and clarified.

He stated that 90 million Americans have some type of hospital prepayment coverage at the present time; that coverage of the balance is urgently needed; that the total goal to be attained under voluntary prepayment will not come easily.

Speaking for the U. S. Chamber of Commerce, Edwin J. Faulkner voiced opposition to the plan. Its enactment, he said, "would create in the minds of the general public a belief that it offers an adequate solution to health problems. This would be a delusion and, therefore, wrong."

The CIO witnesses discounted the plan as a dubious and puny assault on a tremendous problem and they voiced continued faith in national health insurance.

Dr. George Baehr, president and medical director of the Health Insurance Plan of Greater New York, said the bill would help to promote a "balanced health program for the nation," but only if H.R. 7700 is enacted with it. This bill would guarantee mortgage loans of private institutions for the purchase of medical facilities and equipment by organizations supplying voluntary, prepayment, group practice medical care.

Long on Aspirations

The American Federation of Labor, testifying in the person of Nelson H. Cruikshank, stated that the bill does not meet the nation's needs. He added, however, that a direct subsidy to voluntary health insurance plans would be a better way to extend coverage to people now ineligible or unable to afford insurance. He concluded:

> "We can find nothing in it that provides a positive incentive for the commercial insurance companies to meet these major social objectives. That is why we have come to the conclusion that this bill is long on its aspirations and goals but timorous and hesitating in its applications."

Blue Shield and Blue Cross Differ

The national Blue Cross Commission was represented by Richard M. Jones, its director, who said, "The eighty-three Blue Cross plans throughout the world feel unanimously that the program for reinsurance is a step in the right direction to facilitate exploratory measures in particular areas of the American population . . . it is the feeling of the plans that the reinsurance bill will afford the opportunity for even greater expansion of coverage."

On the other hand, Blue Shield Commission endorsed the basic objectives of the bill, but Frank E. Smith, its director, stated that the reinsurance proposal "may well be unnecessary with respect to Blue Shield plans," because these plans had "demonstrated their ability to stand on

(Continued on Page 470)

Minnesota Academy of Medicine

Meeting of April 8, 1953

The regular monthly meeting of the Minnesota Academy of Medicine was held at the Town and Country Club on Wednesday evening, April 8, 1953. Dinner was served at 7 o'clock and the meeting was called to order at 8:10 p.m. by the President, Dr. E. A. Regnier.

There were forty-three members and two guests present.

Minutes of the March meeting were not read.

The following men were elected as candidates for membership in the Academy:

Minneapolis—Dr. John J. Boehrer, Dr. Lyle Hay, Dr. C. A. McKinlay, Dr. Baxter A. Smith, Dr. R. S. Ylvisaker.

University of Minnesota—Dr. James R. Dawson and Dr. Frederick H. Van Bergen.

Amendments to the Articles of Incorporation and By-Laws were read, to be voted on at the May meeting.

The scientific program followed.

Dr. Robert F. McGandy, of Minneapolis, read his Inaugural Thesis as follows. Lantern slides were shown.

LIPOSARCOMA OF THE EXTREMITIES

ROBERT F. McGANDY, M.D.

Minneapolis, Minnesota

Although simple lipomas are quite common, malignant fatty tumors are thought of as rare. In 490 cases of fatty tissue tumors at Johns Hopkins Hospital, twelve were liposarcomas. In a similar series some years ago at New York Presbyterian Hospital 1,454 cases of lipoma were found to twenty-one liposarcoma. In 1943, Stout in his original paper on liposarcoma could only assemble forty-three cases. In the next eight years or up to 1951, he collected a total of 262 cases.

Ewing feels that liposarcomas occur much more frequently than is generally thought, and that reports in the literature do not reflect the true incidence of the disease. He furthermore states that the wide divergence in structure between fat tissue and benign lipoma and the various types of liposarcoma has stood in the way of recognition of these tumors. Stout agrees stating that liposarcomas are commonly misdiagnosed or not diagnosed at all.

The majority of these tumors develop late in life, although they can occur at any age. Sixty per cent are past the age of forty and the mean age is fifty-three. Although lipomas are more common in the female, liposarcomas seem to be distributed apparently equally between males and females.

The etiology of liposarcomas is unknown. There are those who feel they originate from pre-existing lipomas which may be benign, lying dormant for years and then suddenly become malignant and begin to grow. Stout agreed with this in 1943, but more recently feels he was in error when he re-studied an early case in his series. Most writers apparently feel that malignant liposarcomas have no connection with benign lipomas although they can appear together. Trauma, which seems to be mentioned in so many tumors and diseases, is suggested in liposarcomas but Ewing does not think it is a causative factor. Disturbances in thyroid and pituitary states, congenital situations and even alcohol have been suggested as etiological factors.

Liposarcomas can occur wherever we find fatty tissue. Ewing felt they were most frequent in the intermuscular, perineal, periarticular and mediastinal regions. Others have thought of liposarcomas as being located principally in the retroperitoneal, and perirenal regions or in the mesentery and omentum. Stout feels this variation is due to selective reporting and the fact that many liposarcomas elsewhere are missed. In his present series of 262, the general distribution has remained approximately the same, but the number in the lower extremity has grown disproportionately with any other region. In order of frequency the distribution in these 262 cases is: lower extremity, 118; retroperitoneal, omental, mesenteric and perirenal areas thirty-six; trunk thirty-six; upper extremities twenty; head and neck seventeen; breast twelve; mediastinum and diaphragm five; kidney three; vagina and vulva two; spermatic cord and testes two; broad ligament two; lung two; one each in the orbit and maxillary sinus and region not stated five. He furthermore states that the liposarcomatous elements in some mesenchymomas or mesodermal tumors were not included in this list. Stout also calls attention to the fact that liposarcomas look very much like fibrosarcomas and can imitate fibrosarcomas. He calls attention to areas with huge foamy lipoblasts and the presence of bizarre giant cells with pycnotic nuclei, which are not present in fibrosarcomas. He personally told this author that one of the reasons these tumors are frequently called fibrosarcomas by pathologists is that all mesenchymal tumors contain fibroblasts.

Liposarcomas vary greatly in size, depending on their rate of growth. One report cites a woman who had pain in her thigh at age twenty-three, swelling being noted at age thirty-six, and a small mass was removed at age thirty-eight; recurred at age forty-four, and was again removed at age sixty. Another case in the literature records a tumor growing to an enormous size in six months, with death occurring a few months thereafter.

Clinically, in the extremities, one should remember that imple fatty tumors and lipomas are more superficial, nd generally speaking, a deeper mass in the thigh or rm would lead one to be more suspicious of lipoarcoma. As a rule, liposarcomas are first noted as ounded, sort or firm swellings which appear spontaneously and grow steadily. When large, pressure effects are noted.

Grossly, these tumors not only vary in size but also in firmness, as a rule being more firm than adipose tissue or lipomas. They are bulky, nodular masses. Generally, they are encapsulated until recurrences develop as diffusely infiltrating metastasizing processes. Because they can be very vascular, hemorrhage frequently occurs in them, thus varying their color from shades of lemon and yellow to red and brown. Over half of them are slimy and mucoid. On section, normal fat can appear between the definite sarcomatous areas and in some cases, fibrous areas have occurred. Liposarcomas apparently grow more commonly by direct extension than through the blood and lymph channels. In patients with more than one tumor, they have been thought to be due to independent growth. Where metastases have occurred, about 60 per cent go to the lungs and pleura and about 25 per cent to the liver, with isolated cases in the bone marrow and central nervous system. In no case in the literature has there been widespread metastases. The majority of these tumors develop deeply in the inter- and intra-muscular zones of the extremities. In this connection, Ewing calls attention to the fact that the true fascial sarcoma of Virchow is derived from fibrous tissue and does not contain fat. Liposarcomas are closely attached to blood vessels and nerve sheaths. In the latter connection, it is reported that the development of lipomas is undoubtedly affected in some fashion by the nervous system, but there exists no proof that malignant fatty tumors spring from nerve sheaths themselves. This, nevertheless, is still disputed by some.

It is interesting that there are no chemical differences between normal and neoplastic fat, and the fat in a neoplasm is not apparently available for nutritional deficiencies. There are cases of enormous fatty tumors in the literature where the host suffered from malnutrition. Microscopically, these tumors vary more than in the gross descriptions. Some feel the variety of names given make for confusion. Stout, for example, feels that a diagnosis of myxofibrosarcoma or lipomyxosarcoma is the same as liposarcoma. Ewing describes two main types:

1. Fetal or embryonal myxoliposarcoma type, presenting fetal fat cells with much myxomatous tissue and proliferating capillaries.
2. Adult liposarcoma, containing polyhedral cells and spindle cells, highly granular cells with little fat and occasional giant cells.

Stout divides liposarcomas into four groups:

1. Well differentiated—myxoid
2. Poorly differentiated—myxoid
3. Round cell and adenoid type and
4. Mixed type.

In Stout's well differentiated types, metastases seldom occurred; in his other three types, metastases did occur.

Treatment of these tumors varies. Fatty tumors are radiosensitive and some feel x-ray should be used first, followed by surgical resection if necessary. Stout feels a diagnosis should and can, in most cases, be made by a needle biopsy before any surgery is attempted. Early simple removal is often carried out, only to be frequently followed by more malignant recurrences and a high mortality rate. This sequence is frequent in the literature. Surgical removal because of pressure symptoms is sometimes required, this being the situation in the case I wish to report. Finally, amputation must seriously be considered.

Case Report

Mrs. C. H. first came to my attention in 1951, when she was fifty-seven years of age. In 1950 she was hospitalized for coronary sclerosis. In 1951 I was asked to see her because of a large tumor in her left thigh. Her history was to the effect that she had had the tumor for nine years and some five years ago her doctor did not think she should have it removed because of hardening of the arteries. The tumor began to increase in size and caused her some pressure symptoms in the leg. In 1951 an x-ray of her chest revealed two small nodules in the right fourth anterior interspace, which were thought to represent either metastases or possibly an old tubercular deposit. An x-ray of the mass in the leg was thought by the roentgenologist to be either a lipomatous or myxomatous type of tumor. The leg x-rays showed there was same calcification of the larger vessels, but whether this was in the tumor or extrinsic to it could not be ascertained. The x-rays also showed displacement of the thigh muscles by the tumor but there was no evidence of bone involvement.

I removed this tumor through a posteromedial incision, the tumor extending from her knee to her hip. The tumor followed the fascial planes going between and displacing muscles, and had to be separated from the vessels and nerves in the leg. All the tumor was removed.

The specimen consisted of a soft, fatty tumor, with scattered firm areas; the overall tumor appeared encapsulated and weighed about 3750 gms. It measured 35 x 35 x 18 cms. On cut section mucoid material was seen. The color varied from yellow to brown. About eighteen sections were made from this tumor: No cellular detail could be identified in some because of necrosis; in others the picture was composed of pleomorphic and polymorphous cells, with the formation of some collagenous fibrous tissue. In some places considerable fat and some mucin were seen, thus accounting for the myxomatous or fatty appearance. There were numerous multinucleated cells; the nuclei were hyperchromatic and in many places the nucleoli were prominent. Mitoses were seen, but not too frequently. There was no evidence of this being a rhabdomyosarcoma. The tumor was interpreted by the pathologist as lipoma myxomatodes. Radical surgery was suggested by the pathologist.

Two days after the operation, the patient had another attack of pain in her chest which her internist regarded as cardiac in origin, and it was thought best not to consider amputation.

Late in 1952, she entered the hospital again with a diagnosis of hypertension, coronary thrombosis, auricular fibrillation and myocardial insufficiency. Early in her admission I saw her and could find no clinical evidence of recurrence. She died without an autopsy.

Subsequently in a personal interview with Dr. Stout, he repeated that in his experience, liposarcoma, next to fibrosarcoma, is the most common of the malignant sarcomas. At that time he felt that the lower extremity is the most common site for the development of liposarcomas. He distinguished them from fibrosarcomas by bizarre giant cells, single or multinucleated, which in liposarcoma have clumps of pycnotic material in the nucleus, which are in the process of degeneration.

Liposarcomas differ from true myxomas in the fact that the former usually has a vascular pattern similar in appearance to the vascular pattern of embryonal fat. Also, the stellate cells in a myxoma do not have vacuoles in their branched areas which contain droplets of lipoid, as is the case in liposarcomas.

In conclusion:

1. Liposarcomas are quite common in the thigh.
2. They can in many cases be diagnosed by needle biopsy.
3. Simple removal is not infrequently followed by recurrences.
4. Radical amputation should be considered.

Discussion

Dr. F. E. B. Foley, Saint Paul: Several years ago at the Ancker Hospital I encountered a tumor of this sort which gave very bizarre urologic findings. The woman had what appeared to be a very large left kidney. A shadowgraph catheter introduced into the left ureter and kidney pelvis crossed the midline above the sacrum and its tip lay in the region of the right kidney. Bilateral pyelograms showed both kidney pelves in the right renal region and somewhat overlapping each other. The appearance was that of unilateral fused kidney with crossed dystopia of the left renal mass, but was not typical of that condition.

At operation through the left flank, an enormous fibrolipomatous mass was found lying against the muscles of the renal fossa. The front of the mass, covered by peritoneum, extended over to the right kidney and had carried the left kidney before it placing the anterior surface of the left kidney against the anterior surface of the right kidney.

The fibrolipomatous mass was readily shelled out and separated from the left kidney which was returned to normal position.

Within a year there was a recurrent mass almost as large as the original. This, too, was readily removed. Again there was recurrence and the woman died within a year following the second operation.

The meeting was adjourned.

Robert E. Priest, M.D., *Secretary*

Meeting of May 13, 1953

The regular monthly meeting of the Minnesota Academy of Medicine was held at the Town and Country Club on Wednesday evening, May 13, 1953. Dinner was served at 7 o'clock and the meeting was called to order at 8:10 p.m. by the President, Dr. E. A. Regnier.

There were forty-six members and five guests present.

Minutes of the April meeting were read and approved.

The guest speaker of the evening was Dr. Richard R. Trail, Medical Director of Papworth Community (Tuberculosis Rehabilitation Center), England.

The President asked Dr. John Briggs to introduce Dr. Trail who spoke on medical conditions in England.

Dr. Martin Nordland, Minneapolis, read a paper on "Diagnostic Difficulty in Surgical Lesions of the Small Intestine." This was illustrated with lantern slides.

The meeting was adjourned.

Robert E. Priest, M.D., *Secretary*

MEDICAL ECONOMICS

(Continued from Page 467)

their own feet financially." He further said that Blue Shield plans are "underwritten and hence, in fact, reinsured by the physicians who sponsor them."

The above account of the unusual alignments of sentiments on this issue, shows conclusively that groups known to be widely divergent, get together with similar views on a subject, but for entirely separate reasons. It also shows that matters of health are not only of concern to differently-motivated groups, but that matters of health often hinge on political and business considerations. Playing politics and business games with such matters can be dangerous, especially when the lives and welfare of an entire population can be affected by it.

◆ Reports and Announcements ◆

HOWARD W. BLAKESLEE AWARD

The American Heart Association has announced that the Howard W. Blakeslee Award for outstanding scientific reporting in the field of heart and blood vessel diseases will be divided into several awards to cover specific categories of newspapers, magazines, radio and television, instead of the single award established last year.

The 1954 Awards have been deferred until the combined meeting of the Second World Congress of Cardiology and the Twenty-Seventh Scientific Sessions of the American Heart Association to be held September 12-17 in Washington, D.C. The Blakeslee Award originally was scheduled for presentation at the Association's annual meeting in the spring.

The Awards will provide a minimum of $500 for each category to be selected by the judges rather than the original single honorarium of $1,000.

The Blakeslee Award was established last year in memory of Howard W. Blakeslee, the late science editor of the Associated Press and founder of the National Association of Science Writers, who died of heart disease. It is awarded annually to individuals whose creative efforts in any medium of mass communication are judged to have contributed most to public understanding of heart and circulatory diseases.

AMERICAN ASSOCIATION OF BLOOD BANKS

The Seventh Annual Meeting of the American Association of Blood Banks will be held at the Shoreham Hotel in Washington, D.C. from September 13-15, 1954. There will be another excellent scientific program, interesting exhibits, a special course for technologists and round-table and panel discussions led by national and international authorities. For further information write to the Office of the Secretary, American Association of Blood Banks, 3500 Gaston Avenue, Dallas, Texas.

COURSE IN POSTGRADUATE GASTROENTEROLOGY

The National Gastroenterological Association announces that its Sixth Annual Course in Postgraduate Gastroenterology will be given at The Shoreham in Washington, D.C. on October 28, 29, 30, 1954. The course will again be under the direction of co-chairmanship of Dr. Owen H. Wangensteen, Professor of Surgery of the University of Minnesota Medical School, who will serve as surgical co-ordinator and Dr. I. Snapper, Director of Medical Education, Bethel Hospital, Brooklyn, N. Y., who will serve as medical co-ordinator. Drs. Wangensteen and Snapper will be assisted by a distinguished faculty selected from the medical schools and Walter Reed Army Hospital, whose presentations will cover all phases of gastrointestinal diseases and problems. The entire session on Friday, October 30, 1954, will be given at the Walter Reed Army Hospital. For further information and enrollment write to the National Gastroenterological Association, Department GSJ, 33 West 60th Street, New York 23. N. Y.

MINNESOTA SOCIETY OF NEUROLOGY AND PSYCHIATRY

The Minnesota Society of Neurology and Psychiatry held their May meeting at St. Mary's Hospital in Rochester. Surgical clinics were held in the operating rooms at 8:00 A.M. The subject for the morning program was "Neurologic Manifestations of Some Collagen Diseases." Those participating were Dr. A. A. Bailey, Dr. L. M. Eaton, Dr. E. H. Lambert, Dr. C. H. Millikan and Dr. R. M. Shick. In the afternoon Dr. N. W. Barker spoke on "Bird Photography and Time-Lapse Photography."

AMERICAN OCCUPATIONAL THERAPY ASSOCIATION

The 37th Annual Conference of the American Occupational Therapy Association will be held at the Shoreham Hotel, Washington, D. C., October 16-22, 1954. The meetings will be as follows:

October 16-17	Preliminary Meetings
October 18-19	Institute—Interpersonal Relations
October 20-22	General Conference—Theme: "Capitalize Your Assets"

COURSE IN CARDIOVASCULAR DISEASES

A course in "Newer Developments in Cardiovascular Diseases" will be given at The Mount Sinai Hospital, New York, October 11 through 15, 1954, under the auspices of the American College of Physicians. As the title implies, the recent advances will be stressed. Dr. Arthur M. Master and Dr. Charles K. Friedberg will direct the course, and prominent cardiologists and cardiac surgeons will participate.

INTER-SOCIETY CYTOLOGY COUNCIL

The second annual meeting of the Inter-Society Cytology Council will be held in Boston, Friday and Saturday, November 12 and 13, 1954. Those having material to present are invited to submit three copies of the title and an informative abstract of not more than 200 words to Dr. John B. Graham, Chairman of the Program Committee, 32 Fruit Street, Boston, Massachusetts, before July 15, 1954. Abstracts of all papers accepted will be published in the official program.

In Memoriam

THOMAS LLOYD DAVIS

Dr. Thomas Lloyd Davis II, of Wadena died as a result of an automobile accident, April 27, near Paynesville. He was thirty-two years old.

The accident occurred while Dr. Davis was returning home from a Phi Chi fraternity banquet in Minneapolis. He was alone in the car at the time.

Dr. Davis was the son and grandson of physicians. Two cousins are practicing physicians and a brother, William, is a senior medical student at the University of Colorado. He practiced with his father, Dr. Thomas L. Davis and his cousin Dr. Luther F. Davis in the Davis Clinic at Wadena.

He was born at Wadena and received his education at Macalester College and the University of Minnesota Medical School. He served his internship at Hurley Hospital, Flint, Michigan, and, after a year of practice at Wadena, took postgraduate training in eye, ear, nose and throat work in Chicago and, later, in Portland, Maine. In 1949, he spent several months in eye surgery at the Patna Mission, near Calcutta, India. He returned to practice with the Davis Clinic in 1950. He was a member of the Upper Mississippi Medical Society and of the Minnesota State Medical Association and the American Medical Association.

Surviving are his wife, the former Geraldine Fryklund; three children, Bonnie (9), an adopted daughter whose father was killed on Okinawa; Thomas (4), Richard (3), Jeremy, seven months; his mother and father, Dr. and Mrs. Thomas L. Davis; his brother, William, Denver; two sisters, Mrs. Denise Ferron, Honolulu, and Mrs. Judy Oehler, Aberdeen, and his cousin and associate, Dr. Luther F. Davis.

FRED C. GROVER

Dr. Fred C. Grover, veteran West Duluth physician, died March 19, 1954, at the age of seventy-nine in Duluth.

Dr. Grover was born at Olathea, Kansas, and established himself in practice in West Duluth in 1912. He received his medical education at Hamline University and at a medical college in Kalamazoo, Michigan. He was a former member of the St. Louis County Medical Society, the Minnesota State Medical Association and the American Medical Association.

Surviving are his wife, Artense C.; a son, Rev. Eugene F. Grover, Beardsley, Minnesota; two daughters, Mrs. Melvin E. Bloomquist and Mrs. Bonnie Carlson, both of Duluth; two brothers, William R. Grover, Detroit, and Dr. Jerry G. Grover, Chicago; two sisters, Miss Mildred E. Grover, Chicago, and Mrs. Ed Carter, St. Petersburg, Florida, and eight grandchildren.

GLENN ROBERT MATCHAN

Dr. Glenn Robert Matchan, seventy-seven, died March 27, 1954, in St. Cloud.

Dr. Matchan was a former Minneapolis city health inspector. After a number of years in general practice and eight years with the health department, he joined the staff of the University of Minnesota Medical School to teach pediatrics. He retired because of illness twenty-three years ago and had been a patient at the Veterans Hospital in St. Cloud for the past three years.

Dr. Matchan was an affiliate member of the the Hennepin County Medical Society, the Minnesota State Medical Association and the American Medical Association.

Survivors are a son, George R., Minneapolis, former state legislator; a daughter, Mrs. Frances M. Carter, Saint Paul, a sister, Mrs. Grace Schroder, Minneapolis, and three grandchildren.

ARTHUR E. STOECKMANN

Dr. Arthur E. Stoeckmann, fifty-three, died after an extended illness in the Community Hospital at St. Peter, April 30, 1954.

He had been a member of the St. Peter State Hospital staff since 1944, and had been associated with the Earl Clinic in Saint Paul from 1929 to 1940.

Dr. Stoeckmann was born at Sheboygan, Wisconsin, and was graduated in medicine from the University of Minnesota medical school. He served his internship at Minneapolis General Hospital and took nine years postgraduate training in surgery and psychiatry at Mounds Park Hospital in Saint Paul. He was a member of the Nicollet-Le Sueur County Medical Society, the Minnesota State Medical Association and the American Medical Association.

JOSEPH J. STRATTE

Dr. Joseph J. Stratte, formerly of Hallock, died suddenly in Minneapolis, April 9, while attending a continuation study course on urology at the Center for Continuation Study on the University campus. He was sixty-seven years old.

Dr. Stratte had practiced in Grand Forks, North Dakota, since 1932. He was born in Dawson, Minnesota, received his medical education at the University of Minnesota and served his internship at the University Hospital. He became assistant to the chief of surgery at the University in 1918 and opened his practice in Hallock in 1922. He remained a staff member at the Kittson Memorial Hospital at Hallock and he had just completed a term as chief of surgery at Deaconess Hospital in Grand Forks.

He was a member of the Minnesota State Medical Association until 1932, when he transferred his membership to the Grand Forks District Medical Society and the North Dakota State Medical Association. He was also a member of the North Dakota-Manitoba Urological Society.

Survivors include his wife, the former Irene Dille; four children by an earlier marriage, Dr. John Stratte, Enumclaw, Washington; Dr. Paul Stratte, Redding, California; Mrs. B. M. Griffin, Mauston, Wisconsin, and Mrs. E. L. Billing, San Francisco; a brother, Henry Stratte, Dawson; a sister, Mina Stratte, Glendale, California, and ten grandchildren.

Of General Interest

Drs. C. L. Haney and Cyril M. Smith were honored at a dinner of the Northern Pacific Veterans' Association on April 12. They have been medical examiners for the Northern Pacific for more than thirty years.

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Dr. George R. McNear, Jr., has joined the staff of the Mankato Clinic. He recently returned from England where he had served as chief of obstetrics and gynecology at a United States air force hospital for three years. A native of Boston, he earned his doctor of medicine degree at Cornell university medical college in 1947. He was an intern at New York Lying-In hospital and was assistant resident obstetrician and gynecologist at the same hospital for the next year. He was chief resident obstetrician and gynecologist at New York city hospital from 1949 until his call to active duty with the air force in 1951.

The medical staff of Winona General Hospital has named **Dr. E. E. Christensen** as president, succeeding **Dr. P. A. Mattison. Dr. L. I. Younger** has been reelected vice president.

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Dr. Harold Elliot, Hayfield, former medical missionary to Africa, spoke to the members of the Community Covenant Church of Taopi, on Sunday, April 11. In addition to his speech, he showed slides illustrating his work in Africa.

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Dr. and Mrs. E. H. Wood left April 18 for a visit to Mexico. Dr. Wood participated in the program celebrating the tenth anniversary of the foundation of the National Institute of Cardiology in Mexico City. Also in Mexico City he gave a lecture at a meeting of the National Academy of Medicine. In Acapulco he addressed the Second Congress of the Society of Internes and Residents of the Institute of Cardiology and presides at a meeting of the section of hemodynamica and physiology of the society. They returned home about May 1.

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Dr. Lewis J. Glynn, Jr., Saint Paul, was the guest speaker at the April meeting of the Bethlehem Academy Parent-Teacher Association. He explained the purpose of psychiatry, emphasizing the importance of competent psychiatric help in some cases.

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Dr. C. W. Rogers, pediatrician of the Winona General Hospital, was the principal speaker at the meeting of the American Legion Auxiliary on April 13. Dr. Rogers supplemented his talk with motion pictures.

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Dr. F. M. McCarten and Mrs. McCarten returned April 9 from Chicago, where they attended the three-day meeting of the American Association of Railway Surgeons.

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Dr. Paul A. O'Leary, senior consultant in the section of dermatology at the Mayo Clinic, has been honored by a group of former fellows in the Mayo Foundation who received training under him. In the March issue of the *Journal of Investigative Dermatology,* eleven scientific papers written by the former fellows appear. These have been dedicated to Dr. O'Leary. The foreword was written by his colleague, **Dr. Louis Brunsting,** present head of the dermatology section. "The occasion renders tribute to one whose professional acumen and educational talent have combined to dignify and enhance the stature of our specialty in his generation," Dr. Brunsting wrote. "Dr. O'Leary's career refutes the assumption that dermatology is a superficial specialty. Long association with expert medical colleagues in group practice has made him a clinician, first and foremost. His concern has always been with the patient as a whole."

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Thirteen Mayo Clinic physicians attended meetings of the Federation of American societies for Experimental Biology at Atlantic City, New Jersey, in April. Those attending were: **Drs. E. J. Baldes, J. L. Bollman, H. E. Essex, E. V. Flock, W. S. Fowler, H. F. Helmholz, Jr., F. D. Mann, H. L. Mason, V. R. Mattox, M. H. Power, G. M. Roth, K. G. Wakim and E. H. Wood.** Speaking before the various societies were Drs. Bollman, Flock, Fowler, Helmholz, Mann, Mattox, Wakim and Wood. Dr. Essex is president-elect of the American Physiological society.

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Dr. S. Shirai, Coleraine, gave a talk on personal health for the Woman's Club of Coleraine at their meeting in April.

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Dr. and Mrs. H. P. Linner, Minneapolis, have recently returned from a cruise around South America which included a visit to the most southern city of the world, Punta Arenas, on the Strait of Magellan. The Linners were away two months aboard the Swedish luxury liner, *New Kingsholm.* Highlights of the sunshine circle were Buenos Aires, Argentina, and Rio de Janeiro, Brazil.

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Dr. and Mrs. Owen Robbins, Minneapolis, recently returned from a six weeks' business and pleasure trip to the Far East. They visited Japan, Formosa, Hong Kong, Philippine Islands, Thailand and Hawaii. While in Manila, Dr. Robbins delivered a paper to the Society of Obstetricians and Gynecologists and to a medical procurement group.

Dr. H. N. Sutherland, who recently sold his interest in the Shipman hospital, was honored at a dinner in April. **Dr. and Mrs. Jack P. Grahek** were host and hostess for the affair which was attended by thirty-one employes of the hospital. Dr. Sutherland was presented with a gift from the hospital employes.

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Dr. John Carroll, formerly of Montevideo, has established a medical practice in Winsted. Winsted has been without a doctor for more than a year.

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Dr. Charles W. Mayo addressed the Mower County Education Association in April. He also was the principal speaker at the April meeting of the Kenyon Parent-Teacher association.

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Dr. William Kosiak, chief of staff of the Two Harbors Community hospital, addressed a meeting of the Saturday Lunch club of Duluth in Ajril. He discussed "The Group Health Program vs. Socialized Medicine." His talk was followed by a question-and-answer period.

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Dr. E. J. Tanquist, Alexandria, attended the sixth annual session of the International Academy of Proctology and abdominal surgery in Chicago. Dr. Tanquist is a Fellow in this organization.

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Dr. B. J. Cronwell, Austin, attended two medical conventions in Chicago. The American Heart meeting and the American College of Physicians.

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Two Mayo Clinic staff men, **Drs. Louis A. Brunsting** and **Paul A. O'Leary,** of the section of dermatology, attended the seventy-fourth annual scientific session of the American Dermatological association in White Sulphur Springs, West Virginia. Dr. Brunsting, president of the association, gave the presidential address on "Observations on Porphyria Cutaneo Tarda." Dr. O'Leary participated in discussions and attended a meeting of the honorary and corresponding memberships committee of which he is chairman.

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Dr. Robert Rosenthal, Chief of Staff, Childrens Hospital, Inc., addressed the Annual Meeting of the Childrens Hospital Association on April 26. His subject was "The Child in Ancient Egypt."

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Dr. John Raihala, Virginia, has been hospitalized in the University Hospital, Minneapolis.

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Dr. J. A. Myers, professor of medicine and public health at the University of Minnesota, addressed the annual meetings of the North Dakota and Iowa Tuberculosis and Health associations in April.

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Dr. Neil Lidenberg, Saint Paul, has invented a small plastic device that will help break the thumb sucking habit of infants and small children. It was developed by Dr. Lidenberg as a hobby, but his invention interested manufacturers. A temporary factory now is in operation in Minneapolis and plans are being completed for a plant to be located in Saint Paul.

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Dr. Richard Bardon, Duluth, addressed the St. Louis County Medical society on April 8. His topic was the history and traditions of the society.

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Dr. and Mrs. E. W. Arnold, Adrian, returned home on April 16, from a six weeks' vacation trip to Tallahassee and Miami, Florida. On their return home they stopped in Chicago where they attended the American Association of Railroad Surgeons' annual meeting. They report a very enjoyable trip, but were satisfied to be at home again.

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Dr. Joseph B. Gaida, St. Cloud, has been elected second vice-president of the Minnesota Academy of Ophthalmology and Otolaryngology for the coming year.

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Three Mayo Clinic staff men participated in special programs in New York, Wisconsin and Canada in April. **Dr. Philip Hench** addressed a meeting of the Buffalo Academy of Medicine in Buffalo, New York. His subject was "The Use of Cortisones and Corticotropins in General Medicine." **Dr. T. J. Dry** participated in a Wisconsin teaching program at Eau Claire, Stevens Point and Appleton. At each meeting he presented a paper on "Cardiac Diseases Which Might Be Amenable to Surgical Treatment." **Dr. W. H. ReMine** participated in a postgraduate refresher course at the University of Manitoba in Winnipeg, Canada. He presented two papers at the course and also participated in a panel discussion on preoperative and postoperative care.

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Dr. H. F. Helmholz, Rochester, vice president of the Minnesota Congress of Parents and Teachers, was guest speaker at the meeting of the Lincoln P.T.A. in Faribault on April 20. Dr. Helmholz had as his subject "The Drug Menace." Dr. Helmholz has had many years of P.T.A. work and has served on the State Board of Managers as Exceptional Child chairman.

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Dr. Frank J. Carthey and Georgia Roberts were united in marriage at the St. Peter's Episcopal Church in New Ulm on April 12. Following their honeymoon in the Black Hills and in Northern Minnesota, Dr. and Mrs. Carthey returned to New Ulm where Dr. Carthey is associated with the Seifert clinic.

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Dr. W. S. Neff, Virginia, spent a week in Detroit, Michigan, where he participated in a course in clinical electrocardiography of the American College of Physicians.

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Dr. and Mrs. Joseph Janes left Rochester April 19 for New York where they sailed on a two-month trip to Europe aboard *The Britannic.* They were accompanied by Mrs. Janes' sister, Miss Sadie Johnson, Atlanta, Georgia. They first visited England where Dr. Janes remained to visit friends and to observe at medical institutions there and in Scotland. Mrs. Janes and her sister toured the continent. They visited in Norway, where they visited relatives, Denmark, Italy, Switzerland, France and Scotland. Dr. and Mrs. Janes sailed for home aboard the Queen Mary and are expected back in Rochester about June 24.

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Dr. Norman Hoover, of the East Range Clinic, Virginia, gave a talk entitled "Health" at the April meeting of the Mountain Iron Parent-Teacher Association.

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Dr. John Damion Krafchuk and Markell Brooks were married April 19 at the Unity Church in Saint Paul. Dr. Krafchuk has been on the staff of the University of Minnesota hospitals.

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Dr. Claude Hitchock, director of the cancer detection center which is a part of the University of Minnesota, spoke at the second clinical conference held for the medical staff at the city hospital in Montevidio in April. Sponsors of the clinical conferences in addition to the medical staff at the Montevideo hospital were the Minnesota Department of Health, University of Minnesota, Minnesota Medical association, Minnesota Heart association and the Minnesota Cancer Society.

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Dr. Richard W. Anderson, associate professor of psychiatry and neurology at the University of Minnesota and director of psychiatric services at Minneapolis General hospital, spoke on "Family Planning for Emotional Maturity" at the annual meeting of the Ramsey County League for Planned Parenthood.

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Dr. Rolland A. Olson has joined the staff of the Martinson Clinic in Wayzata. Dr. Olson was born in Mankato, he graduated from Maplewood Academy in Hutchinson, following which he took his premedical course at Union College, Lincoln, Nebraska. Following graduation from the College of Medical Evangelists School of Medicine in 1945, he served an internship at the Portland Sanitorium and hospital, Portland, Oregon. He served nineteen months in the army, most of which were in Japan. After discharge, he interned six more months, followed by a three year fellowship in internal medicine at the Los Angeles County General Hospital, Los Angeles, California. After a seventeen-month period of private practice, he was recalled into the army. He served in the capacity of chief of the medical service at the Fort Lawton Army hospital, Seattle, Washington, until he received his discharge in March.

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Dr. Allen Hodges of the Mental Health clinic, Albert Lea, was guest speaker when the Business and Professional Women of Albert Lea met on April 14. Dr. Hodges traced the beginning of the mental health program in the state and explained his work with the local clinic.

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Dr. Rudolph Koucky, Minneapolis pathologist, spoke at a regular meeting of the Southwestern Minnesota Medical Society on April 19. Doctors from a six-county area attended.

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Dr. and Mrs. Waltman Walters and son James, left Rochester April 17, on a six weeks' trip to Europe. They sailed from New York on the SS Constitution. After docking at Naples, they toured Italy and Switzerland before going to England where Dr. Walters attended surgical meetings. He was guest of the Association of Surgeons of Great Britian and Ireland at Leeds, at which time **Drs. John Waugh** and **S. W. Harrington,** also of Rochester, presented papers. At the London meeting of the American College of Surgeons, May 17-19, Dr. Walters presided at a luncheon at which a panel discussion was held on "Intestinal Obstructions" in which Dr. Waugh also participated. Dr. Walters was on a panel discussing "Massive Gastrointestinal Hemorrhages." On May 20, Dr. Walters attended a reception of the International Society of Surgery in Paris and he attended operative clinics in different hospitals in Paris May 21 and 22. The Walters family visited briefly on the Riviera before flying from Paris to the United States on June 2.

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Dr. and Mrs. D. E. Dille and **Dr. and Mrs. C. A. Wilmot,** Litchfield, drove to Cleveland, Ohio, to attend the National Medical Convention of the American Academy of General Practitioners which met March 22 thru 25. The doctors attended scientific sessions of lectures on all the latest medical works. One of the outstanding parts of the program was a lecture by Sir Alexander Flemming.

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Dr. Francis Stutzman is now located at the Variety Club Heart Hospital, Minneapolis, following his discharge from service. Dr. Stutzman was stationed in Bremerhaven, Germany, and while there, he and his family were fortunate in being able to tour Europe.

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Dr. S. L. Elliott, Osakis, was able to resume his practice about May 1 following plastic surgery on his fingers due to old x-ray burns. The plastic surgery was done in Miller hospital in Saint Paul.

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The Minnesota Heart association has begun a survey on the relation of heart disease to employment problems. **Dr. Karl W. Anderson,** medical director of Northwestern National Life Insurance Company and chairman of the association's "Cardiac-in-Industry" committee, said the co-operation of 100 Twin Cities commercial and industrial firms has been solicited. The survey ends June 30. Dr. L. G. Reeder, research associate in industrial sociology at the University of Minnesota, is directing the survey.

Dr. Harold J. Walder, Duluth urologist, indorsed a message of hope of the American Cancer society in his report on cancer involvement in prostate gland surgery in the Duluth area. Speaker at the Duluth Rotary club meeting, Dr. Walder told members that hormone treatment developed at the Mayo Clinic, improvement in surgery techniques and treatment of and general increase in public knowledge about cancer have contributed to the decline of what was almost a certain killer at the turn of the century.

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Dr. Roy G. Holly, Associate Professor of the Department of Obstetrics and Gynecology at the University of Minnesota, was in charge of a clinical session at the St. Francis Hospital, Breckenridge, on May 6. These clinical sessions are sponsored by the Minnesota Department of Health, the University of Minnesota School of Medicine, the Minnesota Medical Association, the Minnesota Division of the American Cancer Society, and the Minnesota Heart Association.

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Dr. Merrill D. Chesler, Minneapolis, was certified by the American Board of Plastic Surgery, by examinations held at Galveston, Texas, April 17-19, 1954. Following the examinations he attended the meeting of The American Association of Plastic Surgeons held at the University of Texas Medical School at Galveston, April 21-23.

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Dr. W. R. Brooksher, Editor of the *Journal of The Arkansas Medical Society* for the past twenty years, has resigned from his position..

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Dr. M. K. Beebe, formerly of 1512 Brook Avenue, S.E., Minneapolis, has announced that he will retire from active practice.

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Dr. Cyril Smith, Duluth, was appointed by Governor C. Elmer Anderson to another term on the state athletic commission.

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Value of America's free medical system was stressed by **Dr. Maurice Meller,** Brainerd, at a meeting of the Lions club. Dr. Meller praised America's medical freedom after telling of his experiences in Poland, Czechoslovakia, Italy and Shanghai. Before he left Poland in pre-war days, he said, socialistic institutions were creeping into the country and medical practice was being regimented. Each patient could be allowed only a couple of minutes per call, which resulted in inefficient diagnosis and treatment. The same socialization was occurring in Czechoslovakia and Italy. This led him to go to Shanghai in 1939. Medicine was comparatively free from interference upon his arrival there, but after the Japanese came, socialization of medicine developed, resulting in poor treatment again. Since his arrival in America, Dr. Meller said, he has discovered what a really free medical practice can accomplish for the health of a nation.

Dr. Carl O. Kohlbry, Duluth, spoke on "Poliomyelitis" for the Duluth Business and Professional Women's club, April 13.

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Dr. Byrl J. Kennedy, Assistant Professor in the Department of Medicine, University Hospital, Minneapolis, spoke at the first of two clinical conferences on April 12, at the Hibbing General Hospital. Dr. Kennedy presented case histories of cancer patients, emphasizing the use of hormones in the treatment of breast cancer. He also reviewed recent trends in diagnosis and treatment in the field of cancer.

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Dr. B. F. Van Valkenburg, Long Prairie, observed his ninetieth birthday on April 30. An open house was held at his home from 3 to 5 p.m.

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Dr. and Mrs. E. C. Bayley, Lake City, returned in April from a combined business and vacation trip. They first went to New York where they boarded the *S.S. Patricia* for a West Indies cruise. The cruise included visits to the ports of Port Au Prince, Haiti; Kingston, Jamaica; a port in Colombia, South America; Panama and Havana, Cuba. After landing in New York they spent a week there attending the National Blue Cross and Blue Shield Convention.

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Dr. R. V. Williams was awarded a fifty-year certificate and life membership in the Rushford Commercial Club held in May. He was honored for his fifty years of service to the community. He went to Rushford in 1904. He is a member emeritus of the Minnesota Medical Association and a member and past president of the Southern Minnesota Medical Association.

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Dr. F. R. Kotchevar, Eveleth, president of the East Range Clinic, left May 1 on a month and a half trip to Europe, where he and other members of the American College of Surgeons attended meetings concerning surgery and medicine. The American physicians held a get-together at the Statler hotel in New York on May 4, the day before they sailed. They arrived in Southampton, England, on May 8, then traveled thru Scotland, England, France, Germany, Switzerland and Holland. The trip was sponsored by the Association of British and American Surgeons.

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Dr. Theodore Rasmussen, a former fellow in the Mayo Foundation and brother of Dr. W. C. Rasmussen of the Mayo Cluic, recently was appointed professor of neurology and neurosurgery at the Montreal Neurological Institute at McGill University, Montreal, Canada. Dr. Rasmussen was a fellow in neurology in the Mayo Foundation from 1936-1939, and since 1947 had been professor of neurologic surgery at the University of Chicago and head of the division of neurologic surgery at the University of Chicago clinics.

Two additional doctors have joined the staff of the state hospital at Fergus Falls. **Dr. Samuel McDaniel,** formerly from Brunswick, Georgia, and **Dr. Harry C. Browne,** formerly from the Sandstone State hospital. Dr. McDaniel previously practiced medicine in Virginia for nine years. Dr. Browne received a fellowship in internal medicine with the Mayo foundation in 1937. In 1941 he entered the army but stayed at Rochester teaching fellows going into service until 1948.

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Dr. Arnold Robbins, formerly of Flushing, New York, arrived in Heron Lake in April to join the practice of Dr. Richard W. Nicholson at the local hospital. Dr. Robbins was recently discharged from the service where he was staff surgeon; he worked both in Alaska and Korea. Dr. Robbins and Dr. Nicholson were associated in practice while in the army when both were in Alaska.

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The athletic department of the Butterfield school received a very fine gift from **Dr. A. D. Matson** of St. James. The gift was a whirlpool bath device.

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The medical staff of Mt. Sinai hospital presented a portrait of **Dr. Moses Barron,** first chief of staff, to the hospital in ceremonies May 2. The portrait was painted by Cameron Booth. Dr. Barron began his medical career in 1912 when he was named an instructor in pathology at the University of Minnesota.

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Dr. Richard Johnson, Saint Paul orthopedic surgeon, was the keynote speaker at the first annual meeting of United Cerebral Palsy of Minnesota on May 3, in Minneapolis.

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Dr. Howard Gray, Mayo Clinic surgeon, delivered three Sommer Memorial lectures at the Oregon Medical School Alumni association meeting in Portland in April.

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About thirty-five managers of state medical clinics and members of the Mayo Clinic's administrative staff attended the fifth annual Minnesota Clinic Managers' conference in Rochester in April. Speakers were **Dr. Samuel F. Haines,** G. Slade Schuster, J. W. Harwick, Harry A. Blackmun, Ernest H. Schlitgus, Robert C. Roesler, **Dr. Victor Johnson** and **Dr. Charles F. Code,** all of Rochester.

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Appointment of **Dr. Harold Joffe,** Virginia, as pathologist at Falls Memorial hospital has been announced by the directors of the hospital. Dr. Joffe also has been named to the hospital medical staff. He is pathologist at the Virginia Municipal Hospital and will be at the Falls hospital two days each month to consult with members of the staff and also will conduct pathological conferences. He will be available at other times on call for consultation.

Albert Lea sponsored a very successful orthopedic clinic in April. Children with post-polio, scoliosis, congenital absence of bones and Perthes disease attended. Clinicians were **Dr. Edward H. Juers,** orthopedic surgeon, and **Dr. George Kimmel,** pediatrician. Both are from Red Wing.

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Dr. Hamilton Montgomery, Rochester, received an award from Governor C. Elmer Anderson at the Minnesota Safety council's award presentation dinner, May 12. Dr. Montgomery has been active in safety work in Minnesota for many years.

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Dr. Daniel J. Moos, Minneapolis, attended the seventy-eighth Annual Session of the Arkansas State Medical Society at Fort Smith, Arkansas, on April 19, where he presented a paper entitled "Intestinal Obstruction." In the afternoon meeting he conducted a forum on "Fluid Balance in Relation to the Surgical Patient."

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The appointment of **Dr. Arnold J. Kremen,** Minneapolis surgeon, as chief of the surgical service in the Francis Delafield cancer hospital in New York City and professor of surgery in the Columbia university College of Physicians and Surgeons, effective July 1, has been announced.

Dr. Kremen joined the University staff as an instructor in surgery in July, 1947, became cancer coordinator for the United States Public Health Service at the University's medical school in 1948. He has been an associate professor of surgery since 1950. Since 1951, he has served as director of the University's surgical service at Mt. Sinai hospital, which is affiliated with the University's medical school.

In his new post, Dr. Kremen also will serve as attending surgeon at Presbyterian hospital in New York City. Both the Delafield and Presbyterian hospitals are units of the Columbia-Presbyterian Medical Center.

Author of many publications on subjects pertaining to surgery, Dr. Kremen is a member of the American College of Surgeons, the American Surgical Association, the American Medical Association, the Society of University Surgeons, Sigma Xi, Alpha Omega Alpha and many other organizations.

BLUE SHIELD-BLUE CROSS NEWS

Blue Shield enrollment for the first three months of 1954, according to the quarterly report, showed an increase of 19,205 participant subscribers. This gain for the first quarter of 1954, if projected over the entire year, reflects a greater increase of participant subscribers during the year 1954 than for each of the preceding two years. However, a quarter is too short a period of time, and the increase is too small to permit any positive predictions of a rate of increase greater than has been experienced during the past two years. This is consistent with a slower rate of increase of Blue Shield enrollment nationally, for the past two years as compared with previous years.

After the first full year of operation of Blue Shield in Minnesota, that is on December 31, 1948, a total of 130,639 subscribers had been enrolled. During the following year, 1949, the total number of subscribers increased to 260,501. This represents practically 100 per cent enrollment increase during the year 1949. During the year 1950, an additional 151,232 participant subscribers were enrolled, bringing the total to 411,733. During the year 1951, 112,524 participant subscribers were added to the Blue Shield rolls, bringing the total number of subscribers at the end of the first four years of operation to 524,257. Thus, the average increase in the number of participant subscribers in Minnesota for the first four years was 131,064 per year.

From this time on, continued growth, but at a slower rate, was effected when during the year 1952, the total enrollment increased to 554,961 participant subscribers. This was a net gain of 30,704 participant subscribers. For the following year, a net gain of 54,513 subscribers was added to Blue Shield enrollment to bring the total to 609,474 for the year of 1953. This represents an average annual increase in the number of participant Blue Shield subscribers in Minnesota of 42,609 for the years 1952 and 1953, whereas the average yearly increase for the four preceding years was 131,064. Quite definitely this shows a slowing of the rate of enrollment for Minnesota Blue Shield.

This enrollment experience in Minnesota is consistent with a decrease in the rate of increase of Blue Shield enrollment nationally in the past few years. In 1950, total enrollment of Blue Shield plans in the United states showed a 35.6 per cent increase. The following year, 1951, the percentage of enrollment increase dropped to 27 per cent. Again in 1952, enrollment growth nationally fell to 16.7 per cent and last year, 1953, it amounted to 14 per cent. Thus, nationally, also, the rate of Blue Shield enrollment increase has shown a tendency to level off and become static.

It is important that Blue Shield's enrollment not only be maintained but increased as rapidly as possible if it is to serve the various purposes for which it was organized. For this reason it is encouraging that the increased enrollment for the first quarter this year might portend a slightly greater rate of growth than during the preceding two years.

Since October, 1953, Blue Cross has been represented at the various medical staff meetings in hospitals throughout the state by Victor Anderson, former superintendent of Abbott Hospital and now Hospital Consultant for Blue Cross.

Speaking at these various medical staff meetings, Mr. Anderson has presented the facts of Blue Cross. He has explained fully the benefits, the exclusions of the contract, and presented the facts relating to the high utilization experienced during 1953.

A large number of doctors throughout the state had considered that a Blue Cross contract covered practically every service rendered by a hospital. Many doctors did not realize that out-patient care for diagnostic purposes was not covered by Blue Cross and that Blue Cross rates were based on the amount of hospital service rendered to subscribers.

One of the chief questions directed at Mr. Anderson at the majority of the meetings was for a complete explanation of out-patient service. Many doctors felt that the Blue Cross contract did not go into detail enough to explain the exclusions, and it was up to the doctor to explain these to his patients.

After advising the doctors that Blue Cross was intended to be of service only to people who primarily needed hospitalized bed care and that the care of patients for purely diagnostic purposes can readily be taken care of in the doctor's offices, many doctors admitted that they had unintentionally been lax, and that they were often faulty on admitting patients who did not necessarily need hospitalized bed care. The doctors agreed after knowing the facts of Blue Cross that they would be only too willing to co-operate, and asked that Blue Cross remind them of this fact from time to time.

During the past month all doctors in the state received an open letter from Blue Cross describing a survey of 12,000 clinical case records from hospitals in the state of Michigan. This survey was conducted by the Michigan Hospital Service (Blue Cross) under the supervision of the State Medical Society Advisory Committee. This survey revealed that 18 per cent of the Blue Cross cases overstayed a total of 2,516 days as compared with 7.3 per cent of self-paying cases. This demonstrated that many patients with Blue Cross were more than two and one-half times more inclined to stay in the hospital longer than was considered necessary as compared to the patients paying their own bills.

This study also revealed that 11.7 per cent of the 12,000 cases were clinical cases in which admission apparently was made primarily for diagnostic study—where no definite treatments were received—and used 2,515 Blue Cross days care as compared to 4.4 per cent of cases in which payment was made privately which indicates that patients with Blue Cross were approximately two and one-half times more inclined to go to the hospital for diagnostic studies than patients who were paying their own bills.

Blue Cross now protects over a million Minnesotans. When protecting a third of the population of the state, it can readily be seen how any slight abuse affects the amount of benefits paid. During 1953, one out of every six persons protected by Blue Cross was hospitalized for an average of 6.5 days compared with one out of every seven staying an average of 6.2 days in 1952. This increase incidence, hospital costs and longer stay of patients during 1953 amounted to $1,200,000. If the use of ancillary services could also be reduced just 10 per cent this would result in a substantial saving to Blue Cross of approximately an additional $830,000 a year, and not require increased rates to a point prohibitive for those in low income brackets.

Blue Cross is rendering a very valuable service to the public, the hospitals, and the doctors. Blue Cross wants to serve more people, and it is only through the continued efforts and co-operation of the doctors that we will be able to keep Blue Cross on a voluntary basis.

During the first three months of 1954, Blue Cross hospital expense for subscribers and their dependents totaled $4,350,600.00.

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EPIDEMIC ACUTE GLOMERULONEPHRITIS AT RED LAKE

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THERE IS a growing awareness that acute glomerulonephritis can and does occur in epidemics. Rammelkamp and Weaver[4] have summarized the larger outbreaks as well as instances where multiple cases have occurred in small population units and in family groups. The same investigators[2] are now following a group of 140 cases which occurred on a military installation in Maryland. The Epidemic Intelligence Service of the United States Public Health Service[6] has recently investigated two outbreaks of nephritis in two of the southeastern states. In 1953, Reed[5] reported an epidemic of twenty-two cases which occurred in a rural area of Nova Scotia.

Scarlet fever and streptococcal infections of the upper respiratory tract are recognized as the common precursors of acute glomerulonephritis. However, it is generally accepted that glomerulonephritis occurring after scarlet fever has not been as common during the past several decades as in former years. The association of skin lesions and glomerulonephritis has been known for over sixty years. Yet, it is only comparatively recently that this relationship has been stressed. Callaway and O'Rear,[1] in reporting on seventy-three cases of glomerulonephritis seen over a period of four years, remark that about half were associated with skin lesions of a pyodermatous character and not with any upper respiratory infection.

The epidemic reported here, occurred on the Red Lake Indian Reservation. This reservation is a relatively isolated area in northern Minnesota about 900 square miles in extent. Its population is approximately 2,800, and is distributed among the three villages of Red Lake, Redby, and Ponemah. Red Lake and Redby are five miles apart and Ponemah, across the lake, is twenty-five and thirty miles away, respectively, from the other two villages. The beginnings of the epidemic were first noted in a report submitted to the Branch of Health, Bureau of Indian Affairs, and to the National Office of Vital Statistics. This report is dated August 14, 1953, and in part, it says: "You will notice that the monthly report for July lists five cases of scarlet fever. In the previous month, two such cases were noted on the reservation. These cases were the first seen at Red Lake in five years. There has been nothing unusual about these cases except that in two instances there were no pharyngeal symptoms or signs. These two cases did, however, show marked pyodermatous lesions which had been present for a week or more before the outbreak of the scarlatinaform rash."

"These cases take on added significance because of the fact that there has been a surprising frequency of acute glomerulonephritis within the last two months. In these two months there have been approximately twenty such cases admitted to the hospital. A great percentage of these cases show marked pyodermatous lesions."

"It seems safe to assume that the child population here has been subjected to a wave of streptococcal infection. This is now becoming apparent as scarlet fever, as nephritis, and possibly, as pyoderma."

By August 28, 1953, there had been sixteen cases of scarlet fever and we had, by then, seen

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Much of this material was presented to the Staff of the Lutheran Hospital, Bemidji, Minnesota, October 21, 1953.

Dr. Kleinman is Senior Physician of the Red Lake Indian Hospital.

twenty-four cases of acute glomerulonephritis. On September 10, 1953, the totals stood at twenty-two cases of scarlet fever and thirty-eight cases of nephritis. On October 21, 1953, it was possible to present data on fifty-six cases at a staff meeting of the Lutheran Hospital, Bemidji, Minnesota. The last case, the sixty-third, was seen on November 8, 1953. The week by week incidence of the nephritis cases is shown in Figure 1.

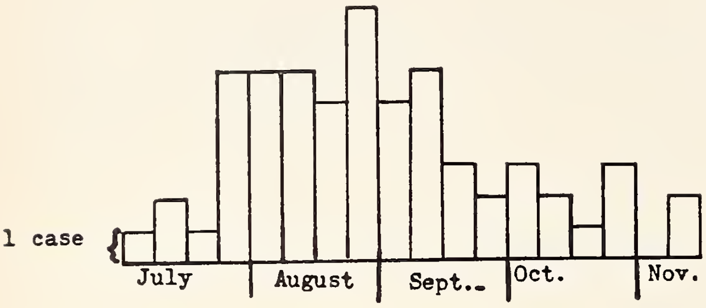

Fig. 1. Week-by-week incidence of glomerulonephritis.

Only children were affected in this outbreak. The youngest was fifteen months old and the oldest was a girl of thirteen years. The most common age groups involved were those in the late pre-school and the early school years. On the basis of this segment of the population at Red Lake, the attack rate comes out to 6.6 per cent. It was common to have two individuals in the same family ill with nephritis at the same time and in one family four siblings were involved.

From the clinical standpoint, these cases were classical:* Only two patients did not have edema in some form and these two cases were subsiding ones. In eight cases the edema was considered 4 plus and in eleven it was 3 plus. Only five cases did not show evidence of early oliguria while in the hospital and these again were in cases that were considered subsiding.

Hematuria, of course, is the keystone in the diagnosis. In our series this varied from microscopic to frank and gross hematuria. Many of the specimens presented the typical "smoky" appearance.

Hypertension was present in twenty-four (42.5 per cent). In fifteen of these twenty-four, the blood pressure readings were better than 150/100. Hypertension, when it occurred, lasted for about a week, although there was one case who remained hypertensive for over six weeks.

Heart failure occurred three times, one of the instances being in our single fatal case. We considered a case in heart failure when we noted an increase in pulse rate, pulmonary congestion, and a sudden smooth enlargement of the liver. It was surprising with what rapidity the livers in these cases enlarged. Such patients, too, were noted to be breathless, restless, and preferred to sit up rather than lie down in bed. Anemia of some degree was present in all cases. In ten instances, it was thought wise to transfuse the patients before discharge. All others had enough hemoglobin deficiency to warrant the prescription of iron preparations orally.

Pneumonia occurred in nine instances. The pulmonary infiltration was characteristically lobar in extent and often was bilateral. It is a moot point as to whether these infiltrates were pneumococcal or streptococcal. In any event, they cleared rapidly under penicillin therapy. A good proportion of the children had evidence of lesser respiratory infections such as nasopharyngitis and bronchitis.

Two of the nephritic patients had a complicating infectious hepatitis and four had complicating specific dysentery due to Shigella Sonnei I. These complications are not surprising because, since our epidemic last year, sporadic cases of hepatitis have been occurring, and from April on, the reservation has been the site of an epidemic of Shigellosis.

However, the most intriguing feature of all has been the pyoderma. Fully two-thirds of our nephritis cases had associated skin lesions. These lesions were not impetigo nor were they impetiginizations of other skin diseases. They were so definite and characteristic that one could, with ease, look at one case and say, "This is it," and look at another and say, "This is not." By actual count, thirty-seven of the nephritics had accompanying pyoderma. Of these, twenty were more or less generalized and were considered quite severe. They were, in fact, severe enough to warrant hospital admission even in the absence of evidence of glomerulonephritis.

The lesions were characteristically distributed over the lower extremities and the lower half of the trunk. The individual lesion was round, slightly raised, pale red in color, and about five to seven mm. in diameter. These lesions developed a small vesicle which was central in location with respect to the entire lesion but which did not extend over the full area of the raised portion. The crust resulting from the rupture of the vesicle was flat and relatively thin. When seen at a later stage, when secondary infection had set in, the crusts were thicker, more irregular, and covered the entire area of the initial lesion. The initial lesions were well circumscribed and in only several instances was there a widespread complicating cellulitis. Itching, while present, was not a prominent symptom.

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*The summary of the clinical features is based on the first fifty-six cases. The findings in the last seven cases would not materially alter the overall picture.

Penicillin apparently effected rapid healing in all cases. Some of the oldest lesions now show some evidence of localized de-pigmentation. In a few cases, the pyoderma recurred after the nephritic child was discharged from the hospital. Again, additional penicillin was rapidly effective.*

Two cases of pyoderma were sent to the University of Minnesota Hospitals for study.** Cultures from the skin lesions of both yielded a non-typeable Group A streptococcus, frequently in pure culture but occasionally with staphylococci. Similar strains of streptococci were isolated from the nose and throat of one of the patients, but not from the other. Cultures of the blood and urine were sterile in both. Neither child developed manifestations of acute glomerulonephritis. Microscopic sections of the skin lesions showed a cellular response which was primarily lymphocytic and eosinophilic rather than neutrophilic. This was somewhat of a surprise although a non-neutrophilic response has been described by Weinstein et al[7] in their studies of experimental streptococcal pneumonia in hamsters. These investigators noted an initial histiocytic response.

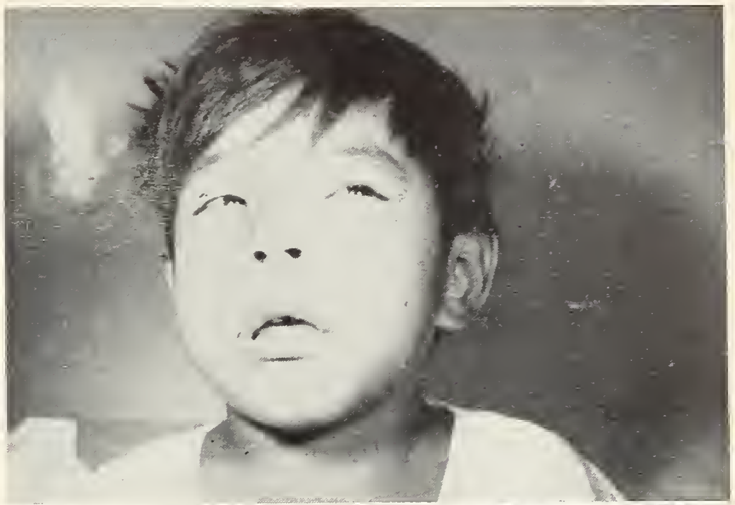

Fig. 2. *(above)* K. S., aged six, on admission, showing typical facial edema.

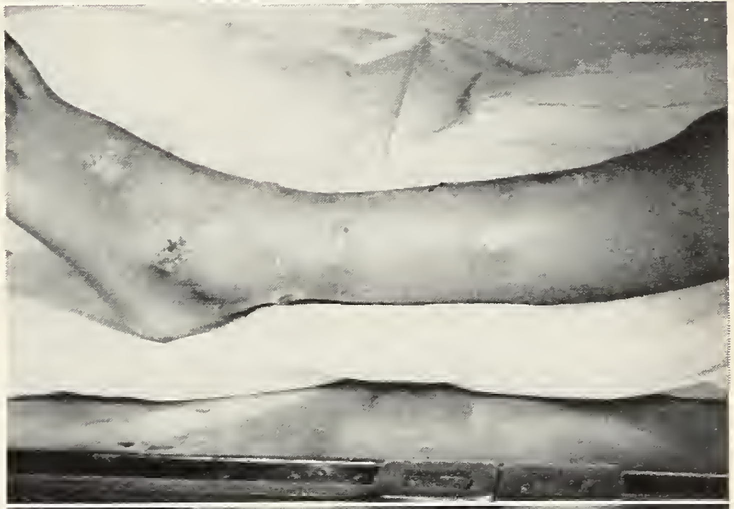

Fig. 3. *(center)* Skin lesions in varying stages—secondary infection in one lesion below the external malleolus.

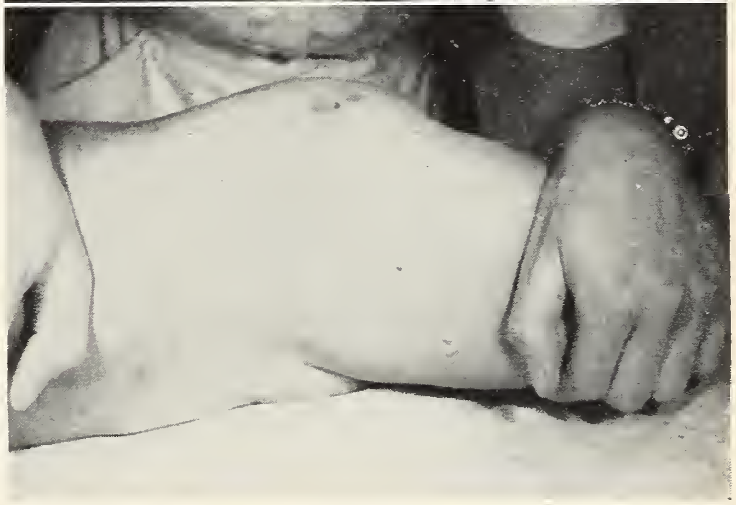

Fig. 4. *(below)* Healed lesions showing characteristic distribution over lower trunk area.

The one fatality in the series occurred in a boy of two. Death intervened on the sixth day of illness and on the fourth hospital day. This child was severely ill from the onset. Edema was marked; urinary output was low; and for the last twenty-four hours he was practically anuric. A spreading pneumonia was detected early. Heart failure was evident at least forty-eight hours before death. The autopsy findings were interesting but not unusual. There was a general anasarca. The right pleural sac contained a large amount of hemorrhagic fluid. The left pleural sac contained a lesser quantity of a like material. There was a diffuse broncho-pneumonia throughout the middle and right lower lobes. The left upper lobe was practically solid with a pneumonic

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*Dr. Samuel H. Silverman, Senior Assistant Surgeon, United States Public Health Service, assigned to duty at Red Lake was responsible for the clinical management of these cases and I am indebted to him for the data on the clinical features, as well as the treatment.

**Through the courtesy of Dr. Lewis W. Wannamaker.

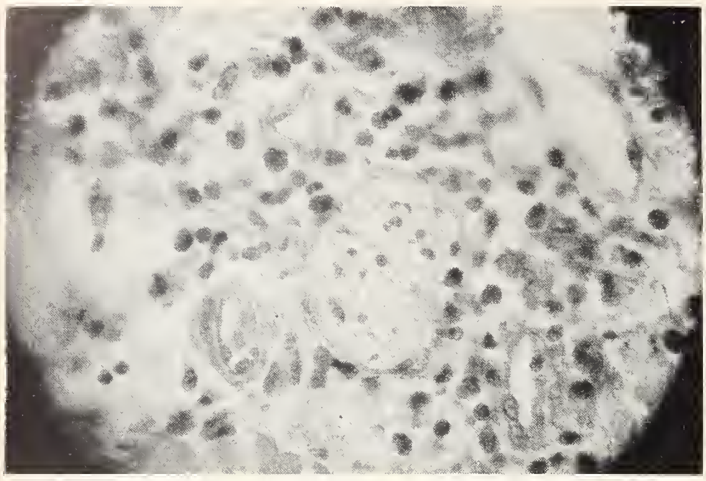

Fig. 5. Section from pyodermatous lesion.

process. The uninfiltrated portions of the lungs were edematous. The pericardial sac contained no excess of fluid and the heart appeared to be grossly normal. There was a moderate amount of ascites. The liver was large uniformly and its edge was smooth, rounded and blunt. The cut surface showed intense congestion. The kidneys weighed together 140 grams which is the norm for a six-year-old child. They were pale with a pinkish undertone. Sections, examined later, showed acute proliferative glomerulonephritis. The bladder contained only a smear of bloody sludge.

Mention should be made here, too, of a list of fifteen cases who were labeled suspects. All of these had albuminuria but with no other findings sufficient to justify a diagnosis of nephritis. It is, of course, entirely possible that some of these may have been nephritics in a subsiding phase.

The treatment accorded these patients was along conventional lines. All patients were confined to bed and given 300,000 units of a repository penicillin every other day. The twenty-four hour urinary output was measured each day and blood pressure readings taken every four hours. When the output was adequate, no other treatment was given and no dietary restrictions were imposed. Where the output was low, the diet was restricted to fluid in an amount equal to the basic fluid requirement of the child plus the amount of the urinary output. The fluid chosen was ordinary gingerale. Gingerale was considered good because of its general palatability, its adequate dextrose content, and its low potassium content. We felt, too, that the nitrogen and salt sparing action of the dextrose was a desirable feature.

Special treatment was required for heart failure, hypertension, and anemia. For heart failure, single dose digitalization was used and except in the fatal case, worked well. Magnesium sulphate in 50 per cent solution was used intramuscularly for the control of hypertension and its action was by and large reliable. The routine administration of phenobarbital did not seem to obviate the necessity for using magnesium sulphate. Anemia, when severe, was treated with repeated blood transfusions given at appropriate times and followed by oral ferrous sulphate preparations. Where the child remained oliguric over a period of several days, there was nothing we could do but "sweat it out." When magnesium sulphate was administered to such patients it was done with some temerity and the patient closely watched for evidence of depression.

A detailed epidemiological survey was started on September 11, 1953, by Dr. Cecil R. Reinstein, Epidemic Intelligence Officer of the United States Public Health Service.* The preliminary survey was completed by September 23, and consisted of the following items:

1. Obtaining clinical data on all cases of nephritis.
2. Obtaining nose and throat cultures on all such cases.
3. Obtaining contact data from all known contacts, familial, and otherwise.
4. Obtaining nose and throat cultures from all contacts.
5. Obtaining, where possible, cultures from the pyodermatous lesions.
6. Drawing blood for anti-streptolysin titre from a substantial number of nephritis cases and some contacts.

As controls, some seventy-five first and second grade children in the Red Lake Schools were similarly cultured. None of these children had had scarlet fever, nephritis, or pyoderma. A slightly larger control group was also cultured in the Cass Lake Area. This is a community fifty miles from Red Lake and its school population is mixed Indian and non-Indian.

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*Dr. Reinstein also headed the teams which came to the reservation later to help conduct the mass prophylaxis program. The bacteriological work was under the direction of Dr. Elaine L. Updyke, Head of the Streptococcus Laboratories of the United States Public Health Service at Chamblee, Georgia.

TABLE I.

	Positive for Beta Hemolytic Strep.		Group A	
	No.	Per cent	No.	Per cent
42 Cases	11	26	9	21
109 Contacts	22	20	9	83
19 Suspects	12	63	10	53
43 Red Lake controls (First Grade)	13	30	3	6

TABLE II.
POSITIVE CULTURES FOR BETA HEMOLYTIC STREPTOCOCCI BEFORE TREATMENT

Group A				Group C	Group G	Total
Not typeable	Type 12	Type 4	Other			
41	3	10	4	17	4	79

Per cent positive for beta hemolytic streptococci . . 20.73
Per cent positive for Group A, not typed 10.76

TABLE III. POSITIVE CULTURES FOR BETA HEMOLYTIC STREPTOCOCCI AFTER TREATMENT

Group A—not typeable	Other Groups
1	4

Per cent positive for beta hemolytic streptococci . . 1.31
Per cent positive for Group A, not typed 26

All of the facts contained in the epidemiological data have not yet been sorted and appraised. This is also true of the bacteriological work. Both of these phases will be the subject of future papers. However, several sets of culture results are now available. Table I shows the results of cultures taken at the time of the preliminary survey.

Beta hemolytic streptococci were recovered from the pyodermatous lesions in sixteen instances, often in pure culture.

On the basis of the bacteriological data obtained at the time of the preliminary epidemiological survey, a program of mass prophylaxis was projected and carried out. The treatment consisted of a single injection of dibenzyl ethylendiamino di Penicillin G.* The dose for individuals over fifteen years of age was 1,200,000 units while those younger than fifteen years received half that amount. The injections were started on October 5, 1953, and when they were completed five days later, 1,997 persons had been treated. This number represented 70.25 per cent of the total available population.

At the time of the treatment, nose and throat cultures were taken on every fourth individual. Care was taken to have this sample random and representative. Three weeks later, during the first week of November, 1953, cultures were again taken to appraise the efficacy of the prophylaxis program. It was possible, in November, to reculture 381 individuals who had been first cultured in October. It is the results of these 381 "before" and "after" cultures that are shown in Tables II and III.

The five positives listed in Table III were not positive at the time of the mass prophylaxis program. All those who were positive at that particular time were negative on reculture three weeks later.

The culture data shown in Tables I and II indicate a high rate of streptococcus recovery in the general population at Red Lake, as well as in the nephritis cases, the contacts, and the suspects. The data in Table III show that, within three weeks after the prophylaxis program, the organisms had all but disappeared. A most interesting feature of the results was the inability of the laboratory (except in a few instances) to type these organisms against any of the known antisera.

Rammelkamp et al[3,4] have pointed out that acute glomerulonephritis is especially likely to follow infection with Type 12 streptococci. Reed[5] considered Type 12 streptococci responsible for the cases of nephritis which he reported. While the Red Lake strains remain as yet unidentified, there is little reason to doubt their nephritogenic properties.

No new cases of acute glomerulonephritis have been seen at Red Lake since November 8, 1953. Nor have there been any instances of the typical pyoderma.

Acknowledgment

The author wishes to express his gratitude to Dr. A. J. Chesley, Secretary and Executive Officer of the Minnesota State Department of Health, and to those persons at Red Lake who helped to organize and execute the mass prophylaxis program.

Summary

1. An epidemic of acute glomerulonephritis is described as it occurred in an Indian community of approximately 3,000 persons.

(Continued on Page 489)

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*Supplied by Wyeth, Inc., Philadelphia, Pa.

SUBDURAL HEMATOMAS

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and
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Rochester, Minnesota

ALTHOUGH subdural hematomas are not rare, they frequently are difficult to distinguish from intracranial tumors. In cases of this disease, the symptoms and results of examination may simulate those in cases of such diseases as cerebral arteriosclerosis, chronic encephalitis and posttraumatic syndrome.

For some time we have been interested in analyzing a series of cases to discern, if possible, any diagnostic leads, criteria for operation and methods of improving postoperative treatment. In spite of the optimism which prevails with regard to the prognosis in cases of subdural hematoma in which operation is performed, the mortality rate is greater than one is led to believe.

Material

This report is based on a review of the clinical histories in 163 cases of acute or chronic subdural hematoma in which operation was performed at the Mayo Clinic. An extradural or intracerebral hematoma was not associated with the subdural hematoma in any of these cases.

Age and Sex of Patients

Subdural hematomas may occur at any age. Although they have been said to occur most frequently among infants and elderly persons, none of the patients in the 163 cases was less than fifteen years of age. In forty-three (26.4 per cent) of the cases, the patients were between sixty and sixty-nine years of age (Table I). One hundred and forty-seven of the patients were men and sixteen were women.

Signs and Symptoms

The clinical history revealed that a mild or severe injury of the head had occurred in 132 (81 per cent) of the 163 cases. An interval of three to five months usually had elapsed between the time of the injury and the occurrence of symptoms. In seventy-three cases (44.8 per cent) the patients had been unconscious for five minutes to three days immediately after the injury. A definite lucid interval had occurred in nineteen (11.7 per cent) of the cases.

TABLE I. AGE AND SEX OF PATIENTS

Age, years	Patients		
	Number	Males	Females
15 to 19	9	8	1
20 to 29	12	12	—
30 to 39	35	28	7
40 to 49	24	22	2
50 to 59	32	30	2
60 to 69	43	41	2
70 or more	8	6	2
Total	163	147	16

Symptoms of a subdural hematoma are caused by increased intracranial pressure, local pressure on the brain and meningeal irritation. The symptoms which caused the patients to consult a physician were as follows: headache in 137 cases (84 per cent); disturbances of cerebral function (coma, drowsiness and psychosis) in 106 cases (65 per cent); weakness of one or both sides of the body in ninety-two cases (56.4 per cent) and visual disturbances in fifty-two cases (31.9 per cent).

Chronic papilledema was present in fifty-three (32.5 per cent) of the cases. The papilledema usually was of 1 or 2 diopters, but it was of 6 diopters in one case in which the patient was thirty-two years of age. Although acute subdural hematomas frequently produce hemorrhage around the margins of the optic disks, such hemorrhage was present in only a small number of the cases in this series.

Examination disclosed a homonymous defect of the visual fields in nine (5.5 per cent) of the 163 cases. In six of the nine cases, the subdural hematoma was contralateral to the homonymous defect; in the three remaining cases, it was ipsilateral. In these three cases, it is probable that herniation of the temporal lobe occurred into the incisura tentorii and caused a shift of the brain stem and pressure on the opposite posterior cerebral artery which produced ischemia of the

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Dr. Craig and Dr. Miller are from the Section of Neurologic surgery of the Mayo Clinic and Mayo Foundation. The Mayo Foundation is a part of the Graduate School of the University of Minnesota.

optic radiation opposite to the subdural hematoma.[7]

In thirty (18.4 per cent) of the cases, impairment of function of the oculomotor nerve was evidenced by a dilated, sluggish or unresponsive pupil, by ptosis, or by dysfunction of the ocular muscles supplied by this nerve. Paralysis of the trochlear nerve was present in three cases (1.8 per cent), paralysis of the trigeminal nerve in one case (0.6 per cent), paralysis of the abducens nerve in two cases (1.2 per cent) and paralysis of the facial nerve in twenty-five cases (15.3 per cent). According to Oldberg,[12] a central type of paralysis of the facial nerve occurs commonly in cases of subdural hematoma.

Convulsions occurred in sixteen (9.8 per cent) of the cases. The convulsions were slightly more common in cases in which the patients were in the third or fifth decade of life than they were in cases in which the patients were in the other age groups. They were of the jacksonian type in ten of the sixteen cases and were generalized in the nine remaining cases. In the ten cases in which the convulsions were of the jacksonian type, they occurred on the side contralateral to the subdural hematoma. The generalized convulsions did not have a tendency to occur predominantly on either side. The occurrence of convulsions did not necessarily indicate that the patients' condition was deteriorating rapidly, as mentioned by other authors.

Electro-encephalography was used in eighty-one (49.7 per cent) of the 163 cases, and it revealed evidence of an intracranial disorder in seventy-six of the eighty-one cases. The most frequent electro-encephalographic abnormality was a delta rhythm. This occurred over the site of the lesion in forty-five cases, on the opposite side in ten cases, bilaterally in seventeen cases and over the entire brain area in four cases. Marsh, Hjartarson and Courville[8] said that subdural hematomas commonly produce a suppression of the alpha activity. They expressed the opinion that this is due to depression of the pacemaker in the parieto-occipital area. They also said that posterior lesions are more likely to affect the pacemaker than are anterior lesions. Dislocation and distortion of the brain may be the cause of bilateral or contralateral dysrhythmia.

Roentgenographic examination disclosed evidence of fracture of the skull in thirty (18.4 per cent) of the 163 cases. A shift of the pineal body was apparent in twenty-two cases (13.5 per cent). Oldberg[12] and Engeset[4] said that a shift of the pineal body is caused more commonly by a subdural hematoma than it is by any other intracranial mass.

Because of a tentative diagnosis of tumor of the brain, pneumoencephalography was performed in forty-three (26.4 per cent) of the cases. It revealed a shift of the ventricular system away from the lesion in forty-two cases. In the remaining case, the pneumoencephalogram was unsatisfactory. In ten cases, this diagnostic procedure did not reveal any air in the subarachnoid space over the cortex involved by the subdural hematoma.

Other roentgenographic findings included calcification in the membrane of the subdural hematoma in one case, thinning of the skull over the site of the hematoma in two cases and thickening of the skull over the site of the lesion in one case. Erosion of the skull by a subdural hematoma has been reported by Engeset.[4]

The hematoma was situated on the right side in seventy-three cases (44.7 per cent) and on the left side in fifty-eight cases (35.5 per cent). In thirty-one cases (19 per cent), a hematoma was found on each side. In one case the hematoma was situated in the posterior fossa. In this case, ventriculography revealed symmetrical dilatation of the ventricles, and an exploratory operation disclosed a large subdural hematoma in the posterior fossa. The patient made an uneventful recovery.

Surgical Treatment

The primary objective of the surgical treatment of subdural hematoma is evacuation of the hematoma to allow the compressed brain to expand beneath the clot.

Two hundred and two operations were performed in the entire group of 163 cases. In forty-five of the 163 cases, the initial exploratory operation was a craniotomy with turning back of a bone flap; in the 118 remaining cases, the initial operation was performed by making holes in the skull with a burr. A second exploratory operation was performed in sixteen of the forty-five cases in which the initial operation was a craniotomy. In these sixteen cases, the bone flap created by the previous craniotomy was turned back in order to remove the membrane of the hematoma and the blood that had collected.

A second exploratory operation also was performed in twenty-three of the 118 cases in which the initial operation was performed by making a hole in the skull with a burr. In fourteen of these twenty-three cases, the holes previously made with a burr were reopened. A craniotomy was performed in the nine remaining cases.

A craniotomy should be performed in all cases in which the membrane continues to fill with fluid after trephination, and in cases in which the clot is solid and cannot be removed by suction through a small opening.

Postoperative Treatment

Postoperative treatment should be started as soon as the diagnosis is established at operation. As soon as the hematoma has been evacuated, intravenous administration of isotonic solution of sodium chloride should be started to encourage an increase in the intracranial contents to fill the space previously occupied by the hematoma. The intrathecal injection of this solution also has proved effective. Blood transfusions should be used when indicated. The intravenous administration of vitamin K occasionally may help to control troublesome hemorrhagic oozing from the membrane of the hematoma.

Cerebral edema may occur and become so severe as to cause herniation of the temporal lobe into the incisura tentorii with resultant compression of the brain stem, hemorrhage and death.[7] The treatment of this complication includes limitation of the fluid intake, administration of 1 ounce (30 gm.) of magnesium sulfate once a day and elevation of the patient's head. Many therapeutic agents have been administered intravenously in an attempt to relieve this complication. These include serum albumin, whole blood, a 20 per cent aqueous solution of dextran and a 50 per cent solution of sucrose. All of these agents appear to be effective in some cases but their place in the treatment of cerebral edema remains to be established. The intravenous administration of 7½ grains (0.5 gm.) of caffeine and sodium benzoate four times a day and the oral administration of small doses of amphetamine sulfate at similar intervals seem to aid in combating the cerebral edema in some cases.

Postoperative treatment is difficult in cases in which the patients are comatose. It includes the maintenance of an adequate airway by means of a laryngeal or tracheotomy tube, the use of an indwelling urethral catheter and adequate care of the skin to prevent decubital ulcers.

Results

In ninety-eight (60.1 per cent) of the 163 cases, the convalescence was uneventful, and the patients remained in the hospital for seven to twenty days after operation. Twenty-four (14.7 per cent) of the 163 patients died after the operation. They seemed to respond satisfactorily for the first or second day, but they became comatose and died on the fourth or fifth day after the operation.

In thirty-two (19.6 per cent) of the cases, postoperative examination revealed a neurologic or mental deficit of 10 to 20 per cent. The patients in these cases were evenly distributed throughout the various age groups. It is well known, however, that the surgical risk is higher and the results of operation are less satisfactory in cases in which the patients are elderly persons.

Generalized convulsions occurred postoperatively in eight (4.9 per cent) of the 163 cases, and the anticonvulsants were required for their control. In six cases (3.7 per cent), the patients complained of such symptoms as dizziness, headache, fatigue and photophobia, which occur commonly in cases of postconcussion syndrome.

Craniotomy was performed in seventeen of the twenty-four cases in which the patients died postoperatively. In eight of the seventeen cases, the craniotomy was performed initially, and the blood clot and the membrane of the hematoma were removed. In two of the eight cases, a recurrence of the symptoms necessitated a second exploratory operation which was performed by turning back the bone flap created by the initial craniotomy. Craniotomy was performed one to five days after initial exploration with a burr in nine of the seventeen cases. In seven of the twenty-four cases, the only surgical procedure consisted of opening the skull with a burr and draining the hematoma. Table II shows the postmortem findings in eighteen of the twenty-four cases in which death occurred.

In cases in which intrapontine hemorrhage and infarcts were associated with the subdural hematoma, the patients did not respond satisfactorily after the operation. Spasms of the extensor muscles of the lower extremities developed, Babinski's reflex could be elicited bilaterally and the patients died within four days after the operation.

Comment

There has been much debate concerning the true nature of subdural hematomas. It is well known that some hematomas are the result of rupture of the communicating vessels between the cortex of the brain and the dural sinuses, as has been reported by numerous authors. On the other hand, several authors have demonstrated that a subdural hematoma can be an intradural lesion and can be caused by hemorrhage between the dural layers, the inner layer of the dura acting as the inner membrane of the lesion[6].

In many cases, severe injury of the head will not be followed by subdural hematoma as the brain will undergo swelling immediately after the injury. Because of the finding of laminated, solid blood clots in a subdural hematoma, one of us (Craig[3]) has suggested that recurrent bleeding may occur in a hematoma. Munro and Merritt[10], Gardner[5], Zollinger and Gross[15] have expressed the opinion that the increase in size of a subdural hematoma may be due to the development of fluid, within the subdural membrane, which is high in protein content and is caused by the breakdown of the erythrocytes in the hematoma, which produces a body of fluid surrounded by a semipermeable membrane which has the ability to draw fluid into the sac by osmotic pressure. This drawing of the fluid into the sac by osmotic pressure has been said to be the cause of the fluctuation of the symptoms commonly present in cases of subdural hematoma.

In three of the cases, the patients attributed their symptoms to a lumbar puncture which had been performed previously. It is assumed that the lumbar puncture disturbed the balance between the brain and the hematoma and thus produced signs of increased intracranial pressure. In two of the three cases, operation had been performed with spinal anesthesia two and three months, respectively, before the onset of the symptoms of the subdural hematoma. Lumbar puncture had been performed one day before the onset of coma in the remaining case. In this case, it must be assumed that the progression of symptoms might be attributable to herniation of the cerebellar tonsils into the foramen magnum or herniation of the hippocampal gyrus of the temporal lobe into the tentorial notch as a result of the lumbar puncture.

TABLE II. POSTMORTEM FINDINGS IN EIGHTEEN CASES OF SUBDURAL HEMATOMA

Lesion	Cases
Cerebral Edema	12
Compression and Distortion of Brain	2
Cortical and Subcortical Hemorrhage	9
Infarction of Cerebral Hemisphere	8
Degeneration of Brain	3
Pontine Hemorrhage	5
Infarction of Pons	3
Infarction of Cerebellum	1
Degeneration of Pyramidal Tracts	1
Subarachnoid Hemorrhage	1
Meningitis	2
Contusion of Brain	4
Laceration of Brain	3

Cooney and Baker[2] recently reported a case in which a subdural hematoma developed after the removal of a large tumor from the lumbar segement of the spinal cord. These authors expressed the opinion that a large reservoir of cerebrospinal fluid was maintained in the area formerly occupied by the solid tumor, and, as the fluid left the subarachnoid space and ventricles, the brain was distorted and a surface vessel was torn, thus allowing the blood to accumulate in the subdural space. Bucy[1] reported a case in which a subdural hematoma developed after pneumoencephalography.

It is interesting that mental defects occurred in a rather large number of the cases. In these cases, the clinical manifestations indicated the presence of an organic lesion rather than neurasthenia.

Angiography produces a rather characteristic roentgenographic picture in cases of subdural hematomas. Engeset[4] reported that the angiograms of fourteen of twenty-one patients with subdural hematoma revealed a characteristic avascular area between the skull and the compressed part of the brain. An extradural hematoma is difficult to distinguish from a subdural lesion unless the sagittal sinus is shown to be displaced inferiorly by the extradural hematoma.

There are two schools of thought concerning the initial surgical treatment of subdural hematoma. Many surgeons prefer to make bilateral openings in the skull with a trephine, drain the hematoma, and allow the brain to regain its normal position in relation to the skull, thus obliterating the space occupied previously by the hematoma. Others prefer to perform a craniotomy initially and remove the clot and the membrane of the hematoma. Both of these procedures have their advantages. In some cases, the surgical risk is poor because of the general physical condition of the patients. In such cases, trephination and relief of the existing pressure may be the treatment of choice. If the hematoma is so solid that it cannot be evacuated through openings made with a trephine, or if the membrane of the hemotoma refills after trephination, craniotomy is definitely indicated. In some cases, the mere presence of the membrane of the hematoma is sufficient to prevent the brain from expanding to its normal position. In these cases, the membrane should be removed by means of a craniotomy. Since subdural hematomas occur bilaterally in 20 to 30 per cent of all cases of this lesion, it is imperative that openings be made on both sides of the skull. The incidence of bilateral subdural hematomas is much higher than this in cases in which the patients are children.

It is discouraging that a second exploratory operation was necessary in thirty-nine (23.9 per cent) of the 163 cases. It is well known that craniotomy is difficult to perform in many cases of subdural hematoma owing to the extreme vascularity of the scalp, bone, dura and subdural membrane, and the resultant hemorrhage at the time of operation. The membrane of the hematoma left behind may be the site of subsequent hemorrhage, or it may, as some authors believe, secrete fluid and cause further compression of the brain. In some cases, the mere presence of the membrane may prevent the compressed brain from expanding and obliterating the space previously occupied by the subdural hematoma.

The convalescence is complicated in many cases. In twenty-four of the 163 cases, the patients died within four days after the operation. Since arteriosclerosis was present in several of these cases, the prognosis was poor. Vascular lesions of the pons commonly are associated with subdural hematoma. In any case of an expanding intracranial mass, herniation of the temporal lobe is likely to occur into the incisura tentorii and produce pressure on the brain stem and subsequent hemorrhage in the mid-brain and pons. According to Schwarz and Rosner,[13] necropsy disclosed herniation of the hippocampal gyrus through the incisura tentorii in 83 per cent of a series of cases of expanding supratentorial lesions. Moore and Stern,[9] Van Gehuchten[14] and Nelson[11] have reported cases in which vascular lesions of the brain stem and occipital lobe were associated with a tumor of the brain.

In one case in which a large subdural hematoma was removed from over the right frontoparietal area by means of a right transfrontal craniotomy, diabetes insipidus, hemiplegia and convulsions developed postoperatively, and electro-encephalography disclosed severe injury of the brain at the site previously occupied by the hematoma. The patient in this case was thirty-eight years old. It is assumed that the diabetes insipidus was caused by vascular lesions of the hypothalamus.

Summary

This report is based on a review of 163 cases of subdural hematoma, observed at the Mayo Clinic. The ages of the patients ranged from fifteen to eighty-eight years. One hundred and forty-seven of the patients were men and sixteen were women. There was a history of a mild or severe injury of the head in 132 cases. The hematoma was situated on the right side in seventy-three cases and on the left side in fifty-eight cases. In thirty-one cases, a hematoma was found on each side. In one case, the hematoma was situated in the posterior fossa.

The chief symptoms were as follows: headache in 137 cases; coma, drowsiness or psychosis in 106 cases; weakness of one or both sides of the body in ninety-two cases, and visual disturbances in fifty-two cases. Convulsions occurred in sixteen cases. Electro-encephalography was used in eighty-one cases, and it revealed evidence of an intracranial disorder in seventy-six of the eighty-one cases. Pneumoencephalography was used in forty-three cases, and it revealed a shift of the ventricular system away from the lesion in forty-two of the forty-three cases.

Two hundred and two operations were performed in the entire group of 163 cases. In forty-five cases, the initial operation was a craniotomy; in the 118 remaining cases it was performed with a burr. A second exploratory operation was necessary in thirty-nine of the 163 cases.

In twenty-four cases, the patients died within four days after the operation. The convalescence was uneventful in ninety-eight cases. In thirty-two of the 163 cases, postoperative examination revealed a neurologic or mental deficit of 10 to 20 per cent.

The primary objective of the surgical treatment of subdural hematoma is evacuation of the hematoma to allow the compressed brain to expand beneath the clot. This may be accomplished by craniotomy or trephination. If the hematoma is so solid that it cannot be evacuated through openings made with a trephine, or if the membrane of the hematoma refills after trephination, craniotomy should be performed. Since subdural hematomas occur bilaterally in 20 to 30 per cent of all cases of this lesion, an opening should be made on each side of the skull.

References

1. Bucy, P. C.: Subdural hematoma. Illinois M. J., 82:300-309 (Oct.) 1942.
2. Cooney, J. F. and Baker, G. S.: Subdural hematoma following an operation on the spinal cord: report of case. Proc. Staff Meet., Mayo Clin., 28:364-367 (July 1); correction 405 (July 29) 1953.
3. Craig, W. M.: Chronic subdural hematoma: a condition that follows everyday accidents. Surgery, 1:761-769 (May) 1937.
4. Engeset, Arne: On roentgen examination in head trauma (a preliminary report from the past five years). Acta radiol., 34:288-298 (Oct.-Nov.) 1950.
5. Gardner, W. J.: Traumatic subdural hematoma: with particular reference to the latent interval. Arch. Neurol. & Psychiat., 27:847-855 (Apr.) 1932.
6. Kernohan, J. W.: Personal communication to the authors.
7. Kernohan, J. W. and Woltman, H. W.: Incisura of the crus due to contralateral brain tumor. Arch. Neurol. & Psychiat., 21:274-287 (Feb.) 1929.
8. Marsh, Clemson, Hjartarson, G. D. and Courville, C. B.: The electroencephalographic findings in subdural hemorrhage and hematoma: review of the literature and report of thirteen cases. Bull. Los Angeles Neurol. Soc., 14:163-181 (Sept.) 1949.
9. Moore, M. T. and Stern, Karl: Vascular lesions in the brain-stem and occipital lobe occurring in association with brain tumors. Brain, 61:70-98 (Mar.) 1938.
10. Munro, D. and Merritt, H. H.: Surgical pathology of subdural hematoma based on a study of 105 cases. Arch. Neurol. & Psychiat., 35:64-78 (Jan.) 1936.
11. Nelson, Jack: Involvement of the brain stem in the presence of subdural hematoma. J.A.M.A., 119:864-870 (July 11) 1942.
12. Oldberg, Eric: Subdural hematoma., M. Clin. North America, 29:62-72 (Jan.) 1945.
13. Schwarz, G. A. and Rosner, A. A.: Displacement and herniation of the hippocampal gyrus through the incisura tentorii: a clinicopathologic study. Arch. Neurol. & Psychiat., 46:297-321 (Aug.) 1941.
14. Van Gehuchten, Paul: Le mécanisme de la mort dans certaines cas de tumeur cérébrals. Encéphale., 32:113-127 (Sept.-Oct.) 1937.
15. Zollinger, Robert and Gross, R. E.: Traumatic subdural hematoma: an explanation of the late onset of pressure symptoms. J.A.M.A., 103:245-249 (July 28) 1934.

EPIDEMIC ACUTE GLOMERULONEPHRITIS AT RED LAKE

(Continued from Page 483)

2. There were a total of sixty-three cases observed, in children only. The common age groups attacked were the late pre-school and the early school age groups.

3. Clinically, the cases were classical.

4. There was one fatality.

5. A characteristic pyoderma was observed in association with the nephritis in two-thirds of the cases.

6. A Group A non-typeable beta hemolytic streptococcus was isolated from nose and throat cultures in a substantial percentage of cases, contacts, as well as from a representative segment of the general population.

7. A similar organism was isolated from many of the skin lesions.

8. A mass prophylaxis program using a long acting penicillin preparation succeeded in practically obliterating the streptococcus within a three-week period.

9. The occurrence of nephritis was likewise abruptly terminated within the same period.

References

1. Callaway, J. Lamar, and O'Rear, Harvey B.: A.M.A. Arch. Dermat. & Syph., 88:159, 1951.
2. Rammelkamp, Charles H. Jr.: Personal communication.
3. Rammelkamp, Charles H. Jr., Weaver, Robert S., and Dingle, John H.: Significance of the epidemiological differences between acute nephritis and acute rheumatic fever. Trans. Assoc. Amer. Phys., 65:168, 1952.
4. Rammelkamp, Charles H. Jr., and Weaver, Robert S.: Acute glomerulonephritis. The significance of the variations in the incidence of the disease. J. Clin. Investigation, 32:345, 1953.
5. Reed, R. W.: An Epidemic of Acute Nephritis, Canad. M.A.J., 68:448, 1953.
6. Reinstein, Cecil R.: Personal Communication.
7. Weinstein, L., Colburn, C. G., Seltser, R., Mallory, G. K., and Waring, G. W., Jr.: Experimental streptococcal pneumonia. I. The pathologic and bacteriologic features of the untreated disease. J. Immunol., 67:173, 1951.

ETHER FLUSH TREATMENT OF RETAINED POSTOPERATIVE COMMON DUCT STONES

A Local Survey

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SINCE disparate opinions on the usefulness of solvent or Pribram type of common bile duct flushes vary from complete rejection to what might appear to be over-enthusiasm, we have conducted a survey of local surgeons who have had experience with this technique. Such an evaluation has suggested that within certain limitations, provided certain essential basic principles have been followed, T-tube flushing is a very useful tool in the treatment of retained common duct stones.

Operative management is all too often incomplete. Pribram[22] states that 16 to 25 per cent of operations for choledocholithiasis are followed by retained stones. Best[5] and Hicken[15] estimate that about 7 per cent of patients with gallstones also have liver stones. The presence of stones high in the hepatic radicles at the time of operation and their subsequent downward migration, as a result of more free flow of bile after operative removal of the common duct stones, no doubt explains in many cases the surprising appearance of stones within the common duct on the postoperative cholangiogram.

Composition of Gallstones

Soft bilirubin stones or mud may collect in the common duct in the presence of partial obstruction and infection. Phemister[21] has found that these soft stones contain no calcium carbonate. Stones originating from the gallbladder usually contain calcium carbonate deposited in rings about cholesterol, protein and bile salts.[21,28,31]

Retained common duct stones have the same composition as the gallbladder stones and therefore the operative specimen stones should be tested in a bottle of ether to obtain an index of their solubility before flushing the duct. Since cholesterol rapidly saturates a small amount of ether, this test should be made on at least one-half an ounce of ether.

Well over 90 per cent of stones will dissolve rapidly in ether.[7,27] Doubt as to the accuracy of this statement has been raised by investigators who have tested hardened, dry or formalin-fixed stones. A chemical explanation has not been advanced but the marked difference in solubility between fresh and aged stones appears to be a correct observation as we have been able to demonstrate on many occasions.

Gallstone Solvents

Although many different solvents have been tried,[7,10,13,27,30] pure ethyl ether or warm chloroform are by far the best from the standpoint of solvent action.

Ether was the original and subsequently the most widely used solvent. In 1891 Walker[29] reported the dissolution of a gallbladder stone by the injection of ether into a sinus tract.

Chloroform is a somewhat better solvent than ether, but some chance of liver damage from chloroform may exist. It would seem reasonable to be concerned over the likelihood of absorption by liver cells adjacent to the hepatic ducts of a substance known to be as strong a hepatotoxin as chloroform. Damage might be even more likely in a liver recovering from duct obstruction. Narat and Cipolla[20] first reported the use of chloroform at operation to soften an impacted stone. More recently Best[7] has substituted chloroform as a common bile duct flush at intervals during the periods of ether flushes. Our experience consists of three chloroform flushes on only one patient. In each instance considerable pain lasted about one hour whereas ether in the same patient did not cause pain. Information on another patient in this vicinity related the same type of pain in spite of the use of nupercaine. This patient also developed a temporary three-plus cephalin flocculation.

Although Best[7] did not find liver damage, further investigation into the possible hepatotoxic effects would seem indicated before extensive use of chloroform is undertaken particularly since ether, a safe solution, is quite effective.

Mode of Action of Common Duct Flushes

Even though ether is a proven stone solvent *in vitro,* a natural question arises as to its mode of action within the common bile duct. That stones are cleared from the duct is easily established by eventual clear cholangiograms in a high percentage of cases subjected to ether flushes. Best[7] recently cited 80 per cent success; and this series has an 85 per cent successful end result, in cases treated at least half of the average required time. Some authors[18,30,32] believe that expulsion is the main mode of action and no doubt stones do pass spontaneously in some cases that are being flushed; however, three cases in this series show definite x-ray evidence of progressive decrease in stone size as the flush progresses. Figure 3 demonstrates this point.

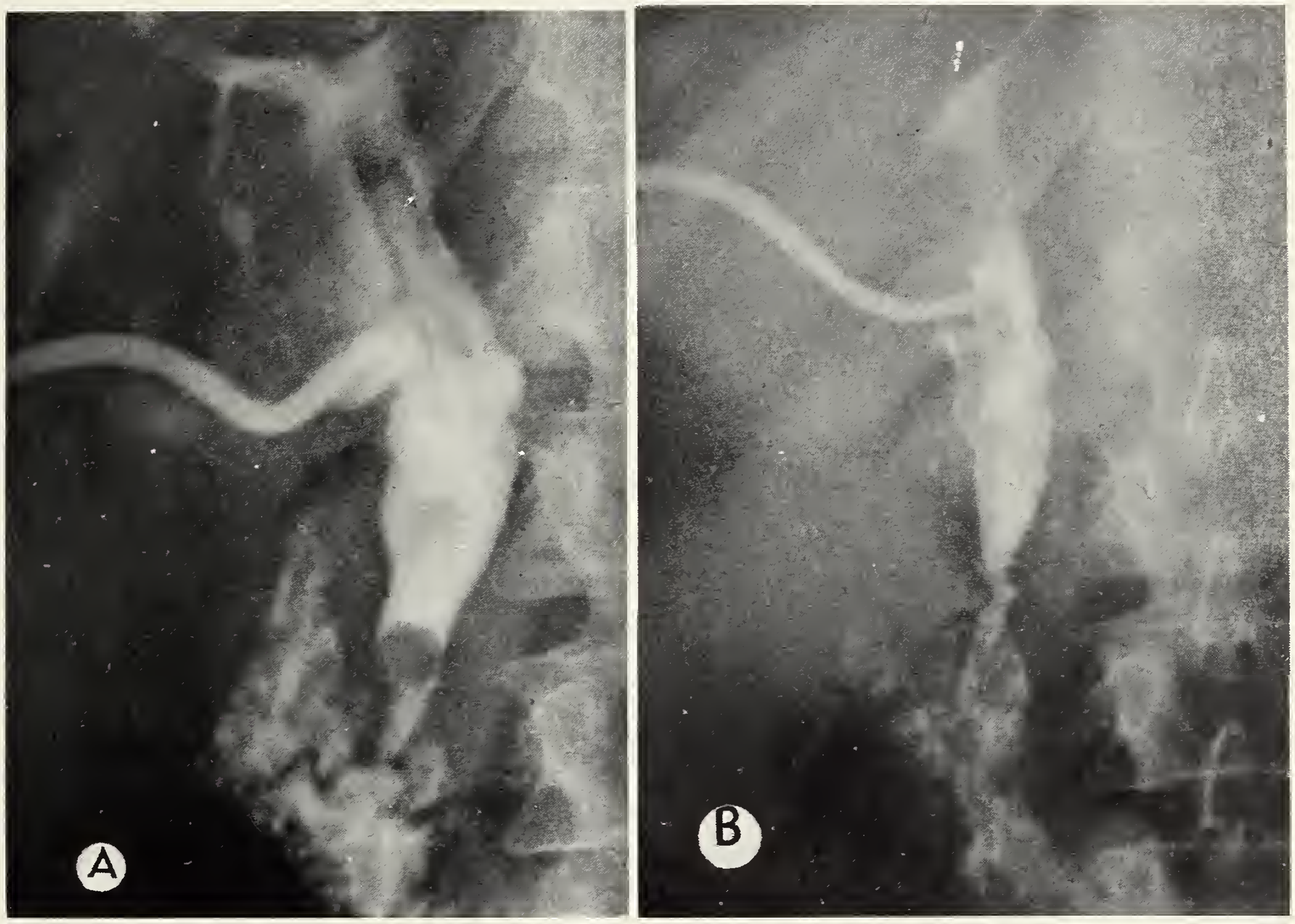

Fig. 1. These two cholangiograms were taken eighty-four days apart. They represent the initial picture of one large stone in the distal common duct and the final clear duct. This patient had already had two operations for removal of common duct stones. In each instance a clear operative cholangiogram was obtained, indicating that the stones had descended from the hepatic ducts.

The greater danger from the use of ether results from rapid elevation of intraductal pressure. Ether boils at body temperature. One cc. of ether will evaporate to 222.1 cc. at 38° C. at 760 mm. Hg pressure.[26]

Bergh[3] has shown that elevation of duct pressure will induce sphincter spasm, and Howell[16] had demonstrated an elevation of serum amylase following pancreatic duct filling during cholangiography. Aside from a study involving injection of relatively huge amounts of ether into a dog's gallbladder and permitting it to remain long periods,[25] there has been no experimental or clinical evidence of damaging effects from ether flushes done over a period of many months,[8,26,27] providing undue pressure is not allowed to develop.

Results from Common Duct Flushing

Pribram, who first reported common bile duct ether flushes in 1935,[22] has reported fifty-one cases with almost uniform success.[23,24] Others have described various modifications and varying degrees of success.[1,6,9,11,18,19,25,26,27,30]

A double catheter[24,26] or a two-way T-tube[1,6,17] are ideal for flushing the common duct. When the ordinary T-tube is used, the problem of providing a vent is best resolved by injecting the ether into a polyethylene tube of small bore which has previously been inserted down the T-tube to the cross-arm of the T-tube. By this device the ether vapor may escape freely through the T-tube by flowing around the polyethylene tube. The double catheter method removes the danger of increased intraductal pressure and therefore makes the procedure safe in the hands of the patient or any lay person.

Cost and time involved make it necessary that lay persons perform the flushes.

A scrutiny of the reported failures in the literature,[2,11,13,15] and of the cases done locally indicates that the main cause of failure is a lack of persistence. The average time required for success was forty-seven days. It then becomes obvious that if the flush technique is to be attempted at all, one must plan on doing it regularly—daily or more often—for a minimum of two months. A failure reported in less time is not a fair evaluation of the method; no more than, for example, one would be justified in treating a toxic goiter medically for a week or two and abandoning treatment as a failure at the end of so short a period.

We do not believe that one should abandon the flushes under a minimum of two months because of a lack of evidence of decreasing size since a stone which is gradually softened by the ether may collapse, fragment and pass rather suddenly. Most of the follow-up cholangiograms at two-week intervals do fail to show a decrease in stone size, but suddenly at the final two-week cholangiogram the duct is clear. Some surgeons have reported removing the T-tube in spite of small stones and without subsequent trouble. If one were to rely only upon spontaneous passage of the stone, it would seem better to clamp the T-tube until the stone passed since the risk of partial duct obstruction, build-up of bilirubin salts, and the danger of cholangitis and infection can manifest trouble even a few years later.

Intraductal Anesthesia

A topical anesthetic solution is an important adjunct to the ether flush technique. Any foreign agent is likely to induce sphincter spasm. In a few cases with elevated sphincter resistance tests the intraductal injection of Nupercaine has been followed by a significant drop in resistance. It is assumed that the stimuli of irritation and of duct distention are blocked by topical anesthesia. This effects a negative or minimal distress to the patient and the advantage of an open sphincter to allow debris or stone fragments to flow into the duodenum.

Nupercaine 1:500 was first proposed by Harris and Marcus[14] in 1946 as the anesthetic agent for sphincter relaxation as an aid to stone passage without ether flushes. Glenn and Hill[12] later employed 1.5 per cent Metycaine for the same purpose. Finally, the benefits of topical anesthesia were combined with the ether flush.[7,27]

Additional benefit may result from the simultaneous use of the Best[4] type physiologic flush composed of antispasmodic drugs and cholegogues.

Recommended Technique

Check the solubility of the patient's fresh gallbladder stones in one ounce or more of ether.

1. Aspirate bile from the duct and T-tube.
2. Inject 10-20 cc. of 1:500 Nupercaine. (Quantity is dependent upon the size of the duct and the degree of ampullary obstruction.)
3. Clamp T-tube for twenty minutes.
4. Aspirate T-tube.
5. Insert intravenous type polyethylene tubing down T-tube at least as far as cross arm of T within common duct. Number 19 gauge I.V. needle is then inserted into the outer end of PE tube.
6. Inject slowly 5-10 cc. of pure ethyl ether. Do not compress T-tube or impede free back flow of ether vapor.
7. Allow open T tube drainage for twenty to thirty minutes after flush.
8. If tolerated, clamp T tube until next flush.
9. Flush two to four times daily. The patient or other lay person may safely perform the procedure after instruction.
10. Cholangiogram at two week intervals.

The key points in safe and effective use of ether are:

a. Avoidance of pressure.
b. Topical anesthesia.
c. Persistence.

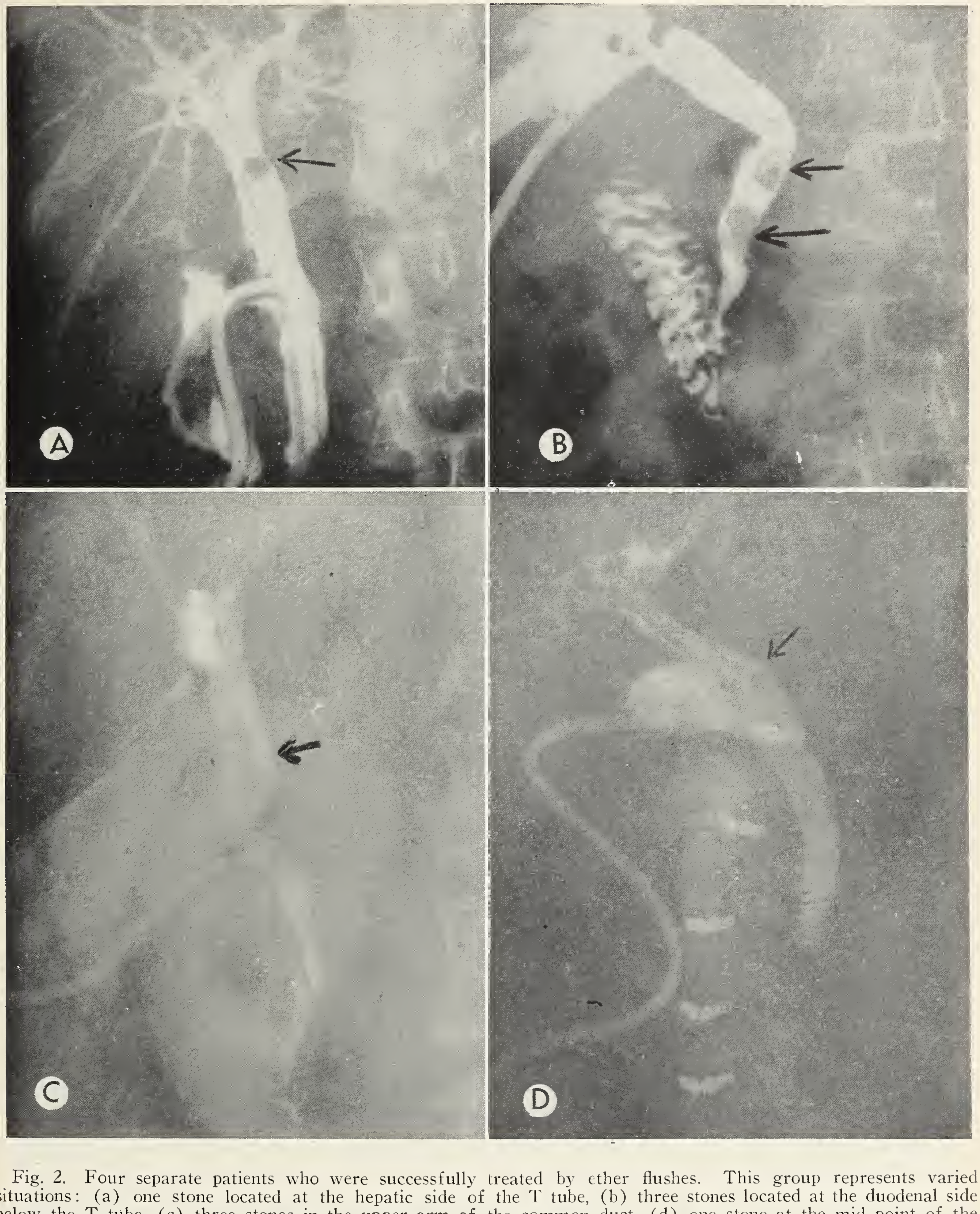

Fig. 2. Four separate patients who were successfully treated by ether flushes. This group represents varied situations: (a) one stone located at the hepatic side of the T tube, (b) three stones located at the duodenal side below the T tube, (c) three stones in the upper arm of the common duct, (d) one stone at the mid point of the T tube. Clearance time was 43, 19, 13, 26 days respectively.

Local Survey of Results

A canvas of the surgeons in and about Minneapolis indicates a high success ratio when a study of their cases revealed conscientious persistence. Four reported failures were not included in the total survey because flushes were not carried out at least once daily for three weeks. This exclusion seems justified since the attempt is hardly

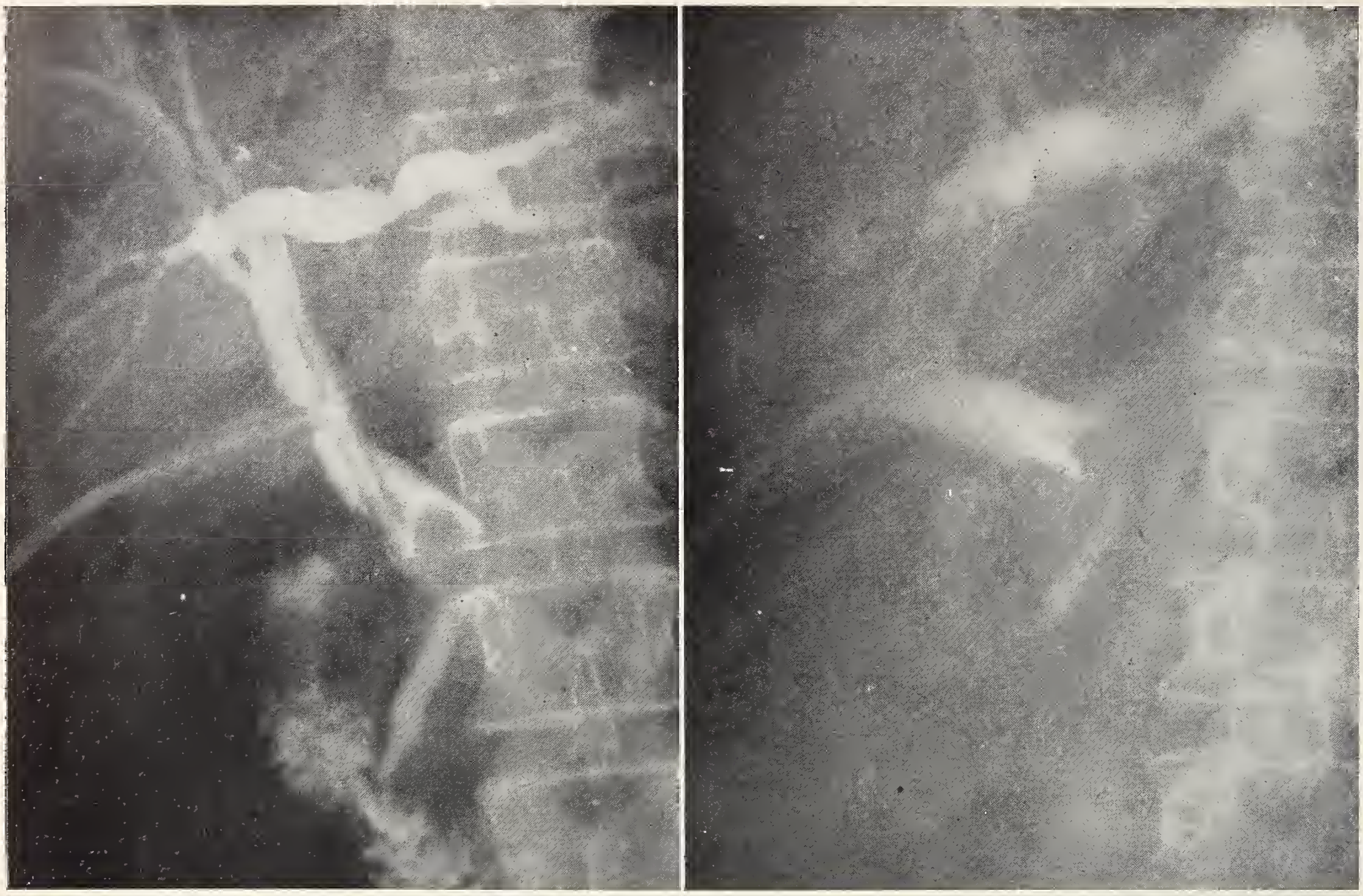

Fig. 3 represents a span of 157 days. Although the patient and surgeon finally decided upon reoperation, at that time the stone had decreased in size from 1.5 cm. to 0.75 cm. and was found to be quite mushy and soft at operation. This case exhibits reasonable evidence of the solvent action of the ether flushes.

sincere, nor is a just evaluation of the procedure obtained from a one or two week trial when the average time required in successful cases was forty-seven days.

Twelve surgeons produced twenty-six cases of reasonable trial with twenty-two successful cases and four failures (85 per cent success).

Results of Successful Cases (85 Per Cent)

Average time (22 cases)	47.1 days
Range	12 to 197 days
Clear in one month or less	9 cases
Requiring over 2 months to clear	3 cases
Size of stones	1 cm. or less
Complete obstruction	1 case
Stones above proximal or hepatic limb of T tube	2 cases
More than one stone	12 cases

Evaluation of Failures (15 Per Cent)

Average time before abandonment of flush 103 days.

One case flushed once daily for 119 days without success. (Gallbladder stones did not dissolve in ether bottle prior to flush and therefore should not actually have been tried.) One case developed a choledochoduodenal fistula as a result of an attempt to pass a ureteral catheter down the distal limb of the T tube (107 days). One case with a 1.5 cm. stone reduced to half its original size by four times daily flushes for 157 days. At reoperation a soft mushy stone was extracted. One case of a stone above the T tube was flushed for twenty-eight days and the T tube then was taken out with a current report of no symptoms at eighteen months. This group does not include four more cases of reported failures in which flushes were done less than three weeks.

The following case study exemplifies the features described within the body of this paper.

Case Report

This fifty-one-year-old moderately obese white woman was admitted to the hospital on June 12, 1952, with a twelve year history of mild, recurrent attacks of upper abdominal pain and a two week history of jaundice.

A G.I. series was suggestive of an active duodenal ulcer. The serum bilirubin was 2.2 mgm. per cent; the blood amylase was normal at 51 mgm. per cent; the urine was positive for bile and the hemograms were essentially normal.

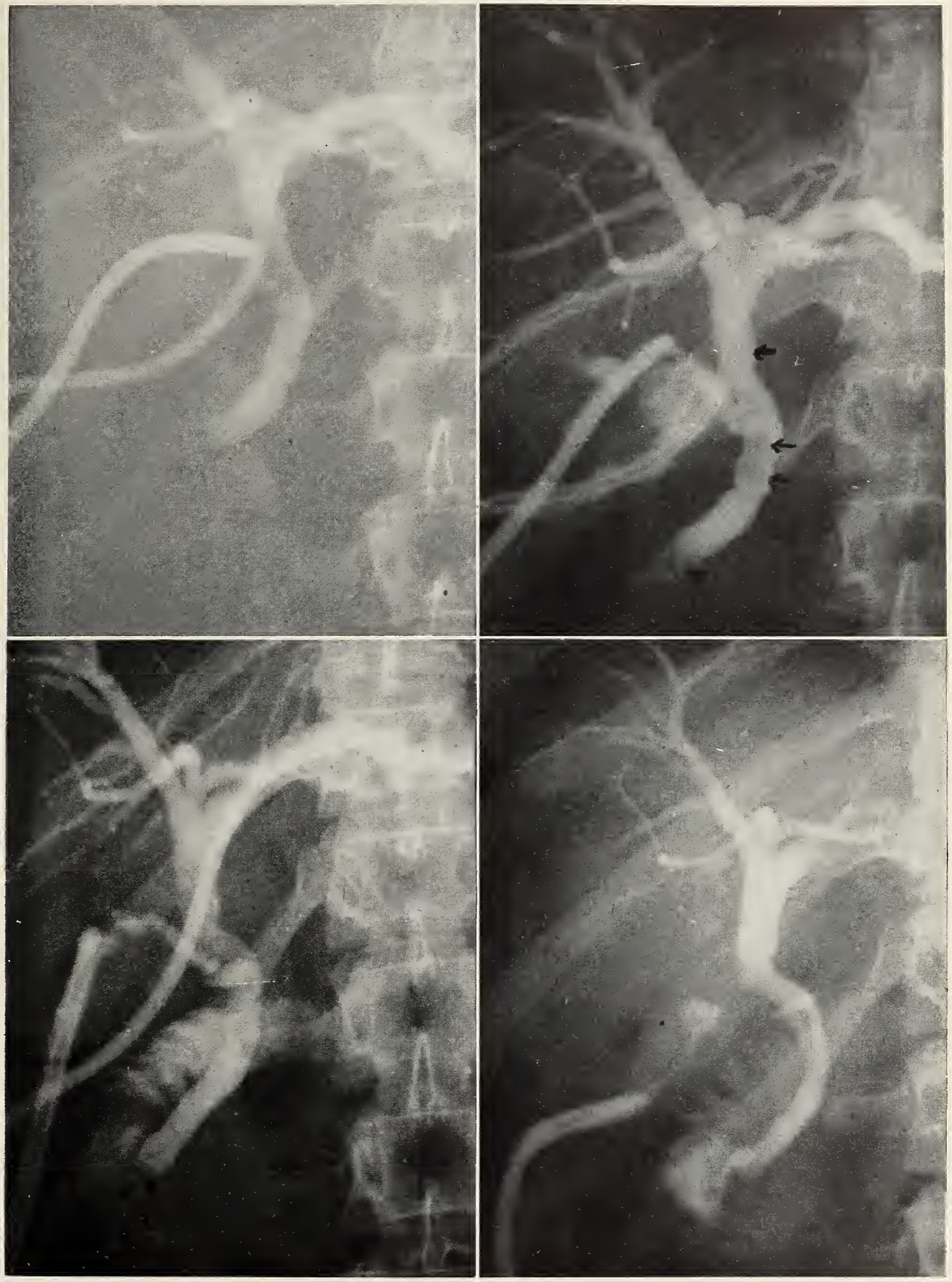

Fig. 4. Beginning with a total obstruction, this case became clear in twenty-three days of ether flushes done twice daily. Although a total obstruction was present, stones could not be separately visualized until one week later, suggesting that in addition to stones and debris obstructing the ampulla, it is likely that more stones migrated downward from the liver. In this instance two catheters were used, one of which was inserted in through the proximal arm of the common duct and the other through the cystic duct.

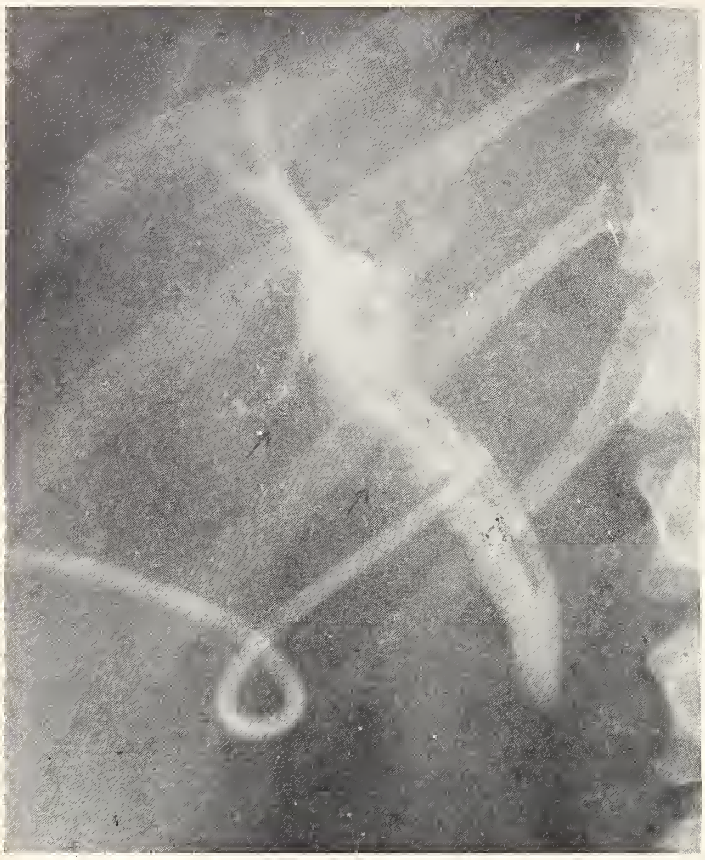

Fig. 5. A cholangiogram revealing at least two common duct stones lying just proximal to the uppermost limb of the T-tube.

A cholecystectomy, choledochotomy and appendectomy were performed on June 16. The gallbladder was contracted and contained stones. The common duct was markedly dilated and thickened. Twelve stones (0.7-1.5 cm. in diameter) were removed from the main hepatic and the main hepatic bile ducts, and five stones were extracted from the right hepatic duct. A cholangiogram was performed through the T-tube on the operating table and this was interpreted and later reported as normal.

Immediately after the operation several of the stones were placed in ether. Gentle agitation of the container readily fragmented the stones.

The patient's postoperative course was uneventful and on her discharge date of June 26, a cholangiogram (Fig. 5) revealed at least two common duct stones lying just proximal to the uppermost limb of the T-tube.

On June 27, 28, and 29, the patient was instructed as to the performance of the above technique (in this case ether-alcohol solution was used and this was injected only once daily into the T-tube). The patient performed the flushes once a day and at no time did she complain of pain. On several occasions transient nausea was noted.

Recheck cholangiograms on August 8, revealed two calculi in the common duct and two calculi in the hepatic ducts. Re-examination on October 17, showed a filling defect in the distal portion of the common duct and one in the hepatic radicle. However, the distal stone was smaller and thus the flushes were continued. On January 4 the patient experienced a transitory attack of upper abdominal pain with radiation into her back which was suggestive of common duct colic. On January 8, a cholangiogram showed the common and hepatic ducts to be clear, but the stones in the distal biliary tree remained unchanged.

Measurement of the sphincter resistance was normal and thus the T-tube was removed on January 16. The patient has had no abdominal distress since the removal of the T-tube one year ago.

Discussion

In this case daily flushes were carried out for 197 days. However, more frequent flushes may have produced more expedient results. The patient performed the flushes without outside help and had no distress other than several transient attacks of nausea. One small stone still remains fixed in an inaccessible position in a biliary radicle and, unless it migrates into the common duct, it is of no clinical significance.

Summary and Conclusions

1. A local survey into the efficacy of the ether flush technique of handling postoperative retained common bile duct stones indicates a rather high degree of success (85 per cent), provided an adequate trial time has been allowed.

2. We have recommended pure ethyl ether as the solvent of choice.

3. Stones in various locations within the duct are amenable to treatment.

4. Avoidance of pressure within the duct is important and may be avoided by the use of a two-way T tube or injection through an intravenous type polyethylene tube inserted into the standard type of T tube.

5. Use of a topical anesthetic such as 1:500 Nupercaine will decrease discomfort and prevent or act against sphincter spasm.

6. A stone larger than one cm. likely should be reoperated since the time required for success by flushing would likely be too long.

7. Stones removed at the initial operation should be tested in ether. The few that do not dissolve *in vitro* will likely not do so *in vivo*.

8. Persistence is essential. Plans should be made for a minimum of twice daily flushes for two months since the average time to achieve success was forty-seven days.

9. The patient or another lay person can safely conduct the flush after proper instruction.

References

1. Baker, R. R.: Choledochostomy—advantage of a modified T-tube. Ann. Surg., 113:1069, 1941.
2. Behrend, M.: The T-tube in surgery of the common duct. Surg. Clin. N. Am., 20:1839, 1940.
3. Bergh, G. S.: The sphincter mechanism of the common bile duct in human subjects; its reaction to certain types of stimuli. Surgery, 11:299, 1942.
4. Best, R. R.: Cholangiographic demonstration of the remaining common duct stone and its non-operative management. Surg., Gynec. & Obst., 66:1040, 1938.
5. Best, R. R.: The incidence of liver stones associated with cholelithiasis and its clinical significance. Surg., Gynec. & Obst., 78:425, 1944.
6. Best, R. R.: Choledochostomy—advantages of a modified T-tube. Surg., Gynec. & Obst., 90:295, 1950.
7. Best, R. R., Rasmussen, J. A., Wilson, C. E.: An evaluation of solutions for fragmentation and dissolution of gallstones and their effect on liver and ductal tissue. Ann. Surg., 138:570, 1953.
8. Boyce, F. F.: Hepatic and biliary tract disease. A review of recent significant advances. Ann. Surg., 109:351, 1949.
9. Burgess, C. M.: Solution of gallstones. J.A.M.A., 114:2372, 1940.
10. Copland, S. M.: The T-tube. South. Surgeon, 11:533, 1942.
11. Gertz, T. O.: Delayed cholangiography and the treatment of overlooked stones by Pribram method. Acta Chir. Scandinav., 90:249, 1944.
12. Glenn, W. R., and Hill, W. H.: Common duct obstruction relieved by injection of a topical anesthetic into T-tube. South. Surgeon, 14:3, 1948.
13. Goldman, B., Jackman, J. and Eastman, R. H.: The management of postoperative choledocholithiasis. Another use for solution G. Surg., Gynec. & Obst., 81:521, 1945.
14. Harris, F. I. and Marcus, S. A.: Common duct stone relieved by injection of nupercaine solution into T-tube. J.A.M.A., 131:29, 1946.
15. Hicken, F. N., McAllister, A. J. and Call, D. W.: The problem of intrahepatic calculi. Am. Surg., 19:695, 1953.
16. Howel, C. W.: Pancreatic duct filling during cholangiography; its effect upon serum amylase levels. M. S. Thesis, U. of Minn., 1950.
17. MacDonald, D.: A two-way T-tube for continuous postoperative perfusion of the biliary tree. The Canad. M. A. J., 62:481, 1950.
18. Michel, M. L.: Treatment of choledocholithiasis with intraductal injections of ether. New Orleans M. & S. J., 99:317, 1947.
19. Morton, H. B.: The treatment of common duct stone missed at operation. Surgery, 12:591, 1942.
20. Narat, J. K. and Cipolla, A. F.: Fragmentation and dissolution of gallstones by chloroform. Arch. Surg., 51:51, 1945.
21. Phemister, D. B., Aronsohn, H. G. and Pepinsky, R.: Variation in the cholesterol, bile pigment and calcium salts content of gallstones formed in gallbladder and in bile ducts with the degree of associated obstruction. Ann. Surg., 109:161, 1939.
22. Pribram, B. O.: New method in gallstone surgery. Surg., Gynec. & Obst., 60:55, 1935.
23. Pribram, B. O.: Ether treatment of gallstones impacted in the common duct. Lancet, 236:1311, 1939.
24. Pribram, B. O.: The method for dissolution of common duct stones remaining after operation. Surgery, 22:806, 1947.
25. Probstein, J. G. and Eckert, C. T.: Injection of ether into the biliary tract as treatment for choledocholithiasis. Arch. Surg., 35:258, 1937.
26. Raffl, A. B.: Experimental studies on the solvent action of ether on gallstones. Am. J. Surg., 52:63, 1941.
27. Strickler, J. H., Muller, J. J., Rice, Carl O. and Baronofsky, I. D.: Nonoperative treatment of retained postoperative common duct stones. Ann. Surg., 133:174, 1951.
28. Sweet, J. E.: Formation of gallstones. Ann. Surg., 101:624, 1935.
29. Walker, J. W.: The removal of gallstones by ether solution. Lancet, 1:874, 1891.
30. Walters, W. and Wesson, H. R.: Fragmentation and expulsion of common duct stone into duodenum by using ether and amyl nitrate. Surg., Gynec. & Obst., 65:695, 1937.
31. Weiser: Rice Institute Pamphlet 20, No. 3, 1933. Quoted by J. E. Sweet. Ann. Surg., 101:624, 1935.
32. Welborn, M. B.: The Management of common duct stone missed at operation. South. Surgeon, 12:185, 1946.

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NUMBER OF PHYSICIANS IN U. S. REACHES ALL-TIME HIGH

The total number of physicians—218,522—licensed to practice in the United States set an all-time record in 1953. Official figures from the fifty-second annual report on medical licensure of the AMA's Council on Medical Education and Hospitals indicate that 7,276 persons were added to the medical profession in 1953. During the same period, 3,421 physician deaths reported to the AMA Headquarters gives a net increase of 3,855 in the physician population of the country. In 1952, an increase of 2,987 was reported.

The report appearing in the May 29 issue of the *Journal of the AMA* shows that 14,434 medical licenses were issued in 1953 by the medical examining boards of the forty-eight states, the District of Columbia, Alaska, Canal Zone, Guam, Hawaii and Puerto Rico. Of this number, 6,565 were granted after written examination and 7,869 by reciprocity or endorsement of state licenses or the certificate of the National Board of Medical Examiners.

The present high level of medical education in this country is indicated by the fact that of the 5,646 graduates of approved medical schools in the United States to take examinations, only 3.8 per cent failed to pass. In comparison, however, of the 1,463 graduates of foreign medical faculties examined, 45.5 per cent failed.

Briefly, of the total number of physicians in the United States at the close of 1953—156,333 were engaged in private practice; 6,677 were in full-time research and teaching; 29,161 were interns or residents or physicians engaged in hospital administration; 9,311 were retired or not in practice, and 17,040 were in government service.
—*AMA News Notes*, June, 1954.

SYNDROME OF THE CEREBELLOPONTINE ANGLE

Report of Two Cases

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THE cerebellopontine angle is the region in the posterior cranial fossa between the petrous pyramid, tentorium, cerebellum and the pons. The syndrome arising from lesions in this area consists of unilateral involvement of the cerebellum associated with paresis of one or more of ipsilateral cranial nerves V, VII, VIII, IX and X.[1]

By far the most common lesion that produces this syndrome is the acoustic neuroma. Of 3,265 verified brain tumors at the Neurosurgical Clinic in Stockholm, 304 (9.3 per cent) were unilateral acoustic tumors.[4] Cushing's series of 1917 included 468 verified brain tumors, of which forty-two (9 per cent) were tumors at the cerebellopontine angle; thirty of these were acoustic neuromas and twelve were miscellaneous tumors. Thus, tumors of the cerebellopontine angle present a relatively frequent problem in neurosurgical operations.

Cushing emphasized the importance of the chronology of symptoms and signs in the diagnosis of acoustic neuromas. These manifestations usually occur in the following order: (1) signs of auditory and labyrinthine involvement (deafness, vertigo, tinnitus); (2) suboccipital and occipitofrontal discomfort; (3) cerebellar signs (ataxia, dysmetria, atonia); (4) involvement of adjacent cranial nerves; (5) increased intracranial pressure (headache, nausea, vomiting, papilledema) and (6) dysarthria, dysphagia, cerebellar crises and respiratory difficulties.

Thus it is seen that the earliest symptoms of acoustic neuromas are the same as those usually associated with Ménière's disease, and early in the course of an angle tumor it is often extremely difficult to differentiate such a tumor from Ménière's disease. It is interesting to note that in the Stockholm series, only one instance of a tumor at the cerebellopontine angle was diagnosed at this early stage, and that patient had come with a diagnosis of probable Ménière's disease.[4] In 560 cases of Ménière's disease in Dandy's series in which the posterior fossa was explored to perform section of the eighth cranial nerve for the relief of the disease, ten tumors of the eighth nerve (approximately 2 per cent) were found.

Laboratory tests may be the only means of differentiating Ménière's disease from angle tumors. Roentgenograms of the skull in the presence of angle tumors may reveal an enlarged or eroded porous acusticus, or erosion of the apex of the petrous pyramid. Studies utilizing air in angle lesions may show deformity of the fourth ventricle with or without hydrocephalus. In cases of tumor, the content of protein in the cerebrospinal fluid is usually increased. Recruitment of loudness is characteristic of the deafness found in Ménière's disease but is not characteristic of the deafness due to angle tumors.[5]

The following case reports emphasize the problems encountered in the differentiation of Ménière's disease from the syndrome of lesions at the cerebellopontine angle.

Case Reports

Case 1.—A fifty-seven-year-old white man was admitted to the Mayo Clinic in July, 1953, because of a two-year history of constant left tinnitus, constant dull ache behind the left ear and loss of memory and ambition. He had noted progressive loss of hearing on the left side. For two months prior to admission, he had experienced difficulty with balance and walking and had fallen several times. He also complained of lightheadedness.

Results of general physical examination and routine laboratory studies were within normal limits. Neurologic examination revealed grade 1 horizontal nystagmus on right and left lateral gaze, slight weakness of the left orbicularis oculi muscle and deafness of the left ear. The corneal reflex was slightly diminished bilaterally. Cerebellar function appeared normal. Otorhinologic examination, which included an audiogram and a caloric test, revealed loss of hearing of the perceptive type and

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The Mayo Foundation is a part of the Graduate School of the University of Minnesota.

The opinions expressed in this publication are not necessarily those of the Department of the Air Force or Department of Defense.

absence of recruitment on the left; however, the left labyrinth was more active than the right. Ophthalmoscopic examination disclosed normal fundi. An electroencephalogram was interpreted as showing grade 2 dysrhythmia, strongest in the left temporal area. Routine roentgenograms of the skull and also Stenver's views of the internal auditory meatus revealed no abnormalities.

Case 1; neurofibroma removed from the eighth nerve (hematoxylin and eosin; x100).

A pneumoencephalogram was interpreted as showing moderate hydrocephalus and slight rotation of the fourth ventricle to the right. The cerebrospinal fluid contained 100 mg. of protein per 100 cc. Left suboccipital craniectomy was done. A neurofibroma (see figure) the size of a large cherry was removed from the left eighth cranial nerve; the seventh cranial nerve was preserved. After an uneventful course in the hospital, the patient was dismissed twelve days following operation.

Case 2.—A forty-five-year-old white man had been seen at the clinic in 1946, at which time he had complained of left tinnitus and progressive deafness of the left ear, both of two years' duration, together with objective vertigo of one year's duration and left frontal and occipital headache of recent onset. Neurologic examination at that time revealed only deafness of the left ear. A diagnosis of Ménière's disease was made and medical treatment was instituted. He was relieved of symptoms, except for deafness, for one year. With recurrence of symptoms, a destructive labyrinthotomy was done. Three weeks after this operation the tinnitus returned but the vertigo did not.

He had remained well until January, 1953, at which time bifrontal headache with left occipital radiation recurred and was accompanied by projectile vomiting several times weekly. The patient experienced numbness of the left face in conjunction with the headache. Because these symptoms persisted, the patient came to the clinic in July, 1953.

Results of general physical examination and routine laboratory studies were within normal limits. Grade 1 nystagmus of "cerebellar type" was present on right and left lateral gaze. There was no papilledema. The corneal reflexes, facial sensation and muscular power were normal, as was cerebellar function. The left ear was deaf. The caloric test showed a nonfunctioning labyrinth on the left but produced a fairly active nystagmus from the horizontal canal on the right with a questionable response from the right vertical canal; this finding is sometimes seen in angle tumors. Roentgenograms of the skull, both routine and Stenver's views, disclosed nothing abnormal.

Left suboccipital craniectomy was done. Exploration of the angle revealed no abnormalities and needle exploration of the left cerebellar lobe failed to reveal a neoplasm. The postoperative course was uneventful. The patient was dismissed on the tenth day after operation. The projectile vomiting ceased but the headaches persisted. Three months after the patient was dismissed, word was received from the family physician that suicidal tendencies had developed and the patient had been committed to a mental institution.

Comment

The differential diagnosis of Ménière's disease and lesions of the cerebellopontine angle, primarily neurinomas of the acoustic nerve, remains a problem despite various laboratory aids in diagnosis. Usually the former condition is associated with a longer history, including remissions and exacerbations, while the latter lesion is usually progressive. Vertigo plus tinnitus, with or without deafness, is the rule in Ménière's disease, while tinnitus followed by deafness is the cardinal sequence in angle lesions. Medical treatment will help a large percentage of patients who have Ménière's disease but will not aid patients who have angle lesions. The latter condition produces progressive involvement of cranial nerves and increased intracranial pressure, whereas Ménière's disease does not. Postoperative mortality in angle lesions is 20 to 45 per cent. When other cranial nerves are involved and papilledema is present, the surgical mortality is increased by 20 per cent because of pontine edema after removal of the lesion. Early diagnosis and early surgical intervention, before pontine compression and pressure on the fifth, eighth and tenth cranial nerves occur, offer the best prognosis.

Suboccipital craniectomy is the usual approach in the neurosurgical treatment of Ménière's disease and angle lesions. In the management of Ménière's disease, total or subtotal (cutting the anterior two thirds) section of the eighth nerve relieves 80 per cent of the patients if medical methods fail. If the patient is deaf, the entire nerve is sectioned. In angle lesions, the eighth nerve is destroyed by the tumor and sacrificed; in total removal of the tumor and its capsule, the seventh nerve is frequently sacrificed because it cannot be isolated. This nerve can be saved if the lesion is small, as in the first case reported.

Case 1 illustrates several features that are interesting from an otologic standpoint. First, normal results of a caloric test are unusual in the presence of an acoustic neurofibroma. These tumors arise from the vestibular branch of the eighth cranial nerve and usually the function of the equilibrial labyrinth is more seriously impaired than is the hearing. Second, the audiogram showed pronounced impairment of high tones with a tendency for retention of the ability to hear low tones. This finding is compatible with acoustic neurofibroma and in questionable instances helps differentiate acoustic neurofibroma from Ménière's disease, in which there is apt to be more loss in low than in high tones. Absence of recruitment of loudness was present in this case, as would be expected in acoustic neurofibroma. As mentioned previously, recruitment of loudness is present in Ménière's disease. Tests for recruitment of loudness are becoming an important adjunct in differentiating acoustic neurofibroma from Ménière's disease; however, it must be remembered that probably 75 per cent of patients who have acoustic neurofibromas have little or no hearing left on the side of the tumor by the time the patient reports for examination. Some hearing must be present in order to demonstrate recruitment of loudness.

The findings in the second case appeared to represent Ménière's disease, with the anxiety overlay which so frequently accompanies the distressing symptoms of this condition. Medical management controlled the attacks for a short period but the symptoms recurred with increased severity and frequency. Because there was no practical hearing remaining in the diseased ear and no findings to suggest a lesion of the cerebellopontine angle, destructive labyrinthotomy was carried out. Destructive labyrinthotomy is an extremely useful and relatively safe means of handling unilateral Ménière's disease that does not respond to medical measures, provided the hearing is impaired to the extent of being of little value to the patient and provided there is nothing to suggest a tumor of the cerebellopontine angle. If the hearing is relatively good or if there is suspicion of a lesion at the cerebellopontine angle, intracranial exploration appears to be the procedure of choice. In this patient, a destructive labyrinthotomy controlled the symptoms for a considerable period, after which symptoms developed that were sufficiently suggestive of disease of the central nervous system to indicate exploration of the cerebellopontine angle.

In typical acoustic neurofibromas and in typical instances of Ménière's disease, the diagnosis is not difficult. However, a considerable number of patients present symptoms that do not fall into a well-defined category and then all available means must be used in an attempt to arrive at the proper diagnosis.

These two cases illustrate the advantage of early surgical exploration in diagnostic problems so that these tumors can be uncovered and removed before the brain stem and adjacent structures are compromised. Vertebral angiography has not substantially assisted in the diagnosis of angle lesions.

References

1. Bailey, Percival: Intracranial Tumors. Springfield, Illinois, Charles C Thomas, 1948.
2. Cushing, Harvey: Tumors of the Nervous Acusticus and the Syndrome of the Cerebellopontine Angle. Philadelphia, W. B. Saunders Company, 1917.
3. Dandy, W. E.: The Brain. In Walters, Waltman: Lewis' Practice of Surgery. Hagerstown, Maryland, W. F. Prior Company, Inc., 12:1-671, 1945.
4. Givre, A. and Olivecrona, H.: Surgical experiences with acoustic tumors. J. Neurosurg. 6:396-407 (Sept.) 1949.
5. Williams, H. L.: Ménière's Disease. Springfield, Illinois, Charles C Thomas, 1952.

ELIAS POTTER LYON—MEDICAL EDUCATOR OF VISION

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DR. ELIAS POTTER LYON, whom the University of Minnesota is honoring by associating his name with the new research building which we are dedicating today, served the University of Minnesota as Professor of Physiology and Dean of the Medical School from 1913 to 1936. Prior to coming to Minnesota, Dr. Lyon had served for six years as Dean of the Medical School of St. Louis University.

To have served for twenty-nine years as a medical dean, a vocation with the usual longevity of five to six years, is a remarkable achievement; particularly so since in neither of his posts did Dean Lyon assume the administrative responsibility for an established smoothly functioning organization, moving serenely along a well charted course. On the contrary, both at St. Louis and at Minnesota Dr. Lyon's appointment as dean followed a period of reorganization; and reorganization in a medical school is always a major surgical operation.

At Minnesota in 1911-12, the medical faculty listed eleven full professors of general surgery and, according to President Vincent, included most of the physicians in the telephone directories of Minneapolis and Saint Paul. To correct this situation, the entire faculty was asked to resign and a new faculty was organized: in part from those who had resigned and in part from new appointees who were brought to Minnesota from other institutions. Dean Lyon's first responsibility was to guide the Medical School through its convalescence from this operation, a convalescence which according to reports was a stormy one; unaided I might add, by "Wangensteen Suction."

Concerning this period the University's historian, James Gray, says: "For a moment after the reorganization plan was put into effect, peace seemed to have settled on the Medical School. But this was only for a moment. There began presently one of the most vivid, eventful and protracted fights of its history. This concerned the question of whether or not the University should become affiliated with the Mayo organization at Rochester, a matter which agitated the entire Minnesota community for nearly a decade."[2] President George Edgar Vincent and Dr. William Mayo led the fight for this affiliation which they believed would place a firm foundation under graduate training in the medical specialties. Strongly supporting this program and responsible for working out its details were Dean Guy Stanton Ford of the Graduate School and Dean Lyon of the Medical School. Most members of the medical faculty, some of them albeit halfheartedly, supported Dean Lyon and the official University position in this controversy. These were indeed stormy times for the Medical School and for its administrative officers, but both weathered the storms safely and moved steadily forward as an effective team for a total of twenty-three years. During this period sound policies in medical education were inaugurated, facilities improved and the staff strengthened.

But, what sort of man was this who was at the administrative helm of the medical school during this important developmental period? Dean Lyon was born and attended secondary school in Hillsdale, Michigan. For his college education "he went to Chicago where, according to historian Gray, he worked at delivering milk through the mean streets of Chicago's west side to put himself through. He first fitted himself for a career as a bookkeeper in a small commercial college but a chance encounter with botany opened a more stimulating world to him and he trained himself all over again in a new field. To finance his graduate work in Chicago, he taught in what was then a university preparatory school. This phase he interrupted to go as an experimental biologist on one of Frederick Cook's expeditions to northern Greenland."[2]

"My decision to become a teacher," Dean Lyon said, "was made quite early, by the age of twenty certainly, perhaps two or three years earlier. It was chance that determined that most of my years should be spent in medical education, but it was deliberate choice that led me to prepare myself to engage in teaching as a career.

Presented at the dedication of the Elias P. Lyon Laboratories, February 11, 1954.

Dr. Diehl is Dean of the Medical Sciences, University of Minnesota.

"My being a dean was as accidental and unplanned as my being in medical education. We who went from the University of Chicago and other schools to the newly formed St. Louis University Medical School in 1904 faced a strange situation. The old University—the oldest in the Mississippi Valley—had taken over two recently united proprietory medical colleges. The doctors who controlled these schools drove a hard bargain with the good Jesuit fathers by stipulating that they should retain their chairs for a period of nine years. For that time they were to constitute the faculty and no new members could be added without consent. The result was some of us fundamental science teachers were members of the University faculty but not of the Faculty of the Medical School in which we taught. It was only after two or three years that we were finally voted into the medical faculty. Even then we were looked upon with some hostility and suspicion.

"In 1907 the dean, a part-time clinical man, suddenly resigned. The old-time owners of the school, constituting a large majority of the faculty, claimed that they had the right to name the new dean. The University on the other hand pointed out that this prerogative had not been reserved to the faculty in the transfer. The latter view prevailed and the University authorities declared that one of us fundamental laboratory men—their own appointees—must take the job. The lot fell upon me.

"You can imagine the situation. I headed a faculty over whom I had no real jurisdiction. The only bright spot was the whole time laboratory group who, like myself, were professional teachers. Practically all our efforts had to be put forth in strengthening the fundamental departments. From time to time as death or resignation intervened, something could be done on the clinical side. As for the rest, it consisted in seeking able young men for subordinate positions and in improving the student body. I came to believe that only on a firm foundation in science and full-time teachers in both scientific and practical branches can a medical school be built or do the work for which it exists. I came to know student problems as very real individual human problems that deserve the best human judgment and the sympathy of a god."[6]

This in brief review was Dean Lyon's background before coming to the University of Minnesota. To those of you who did not know him and to those of us whose memory of him has dimmed over the years, possibly I can best give a picture of the sort of man that Dean Lyon was by quoting briefly from a few of his writings.

First, let us listen to him in February, 1916, as a participant in a symposium at the Annual Congress on Medical Education of the American Medical Association. The title of this symposium was the "Relation of the Laboratory Courses to the Work in the Clinical Years." "First," he said, "I tried to work out this relationship on a family basis. Medicine, I said, is the mother of physiology, and I prepared to come here and paint a pretty picture of physiology taking care of mother in her old age. But when I looked around for father, my trouble began. I found that medicine had a promiscuous lot of children—quite a heterogeneous household ranging all the way from substantial oldest son, anatomy, to sapheaded chiropractic. I became fearful of discovering a scandal and gave up the idea of family relationship.

"I then saw that if I were intelligently to discuss the relation between the laboratory sciences and the work of the clinical years I needed to know something about the clinical years. So I followed a master of diagnosis in his day's work. I saw him percuss and auscultate; I saw him count corpuscles and analyze excreta; I saw him elicit reflexes and measure temperature; I heard him speak of valves and pressure and enzymes and neurons and calories. I said: 'That man is not practicing medicine. He is practicing physiology.' I watched a therapist at the bedside and found that he was not practicing medicine but pharmacology. I saw a surgeon earning a thousand dollars. I discovered that he was not practicing surgery but anatomy, pathology, physiology and —high finance! Finally, the relation I was seeking came suddenly into mental view. Anatomy, physiology, pharmacology, bacteriology, chemistry and the rest are not children of medicine; they are not the branches of an evolutionary tree; they are not handmaids; they are not stepping stones or preparatory stages. They are it. They are medicine itself.

"Every correct observation of function or disorder, every established fact of structure, every corroborated discovery in the field of causation or in the domain of restitution, every correctly interpreted experiment in test tube, animal or man, whether made by layman, practitioner or professional scientist, is describable in the terminology of the fundamental sciences, and is really a constituent part of those sciences. These make up medicine as a real living body. For clothing there is folk psychology, a modicum of ritual, that indefinite confidence-producing mantle called personality, a veil of magic, and a vanishing hat, labeled 'empiricism,' with a hole in it through which talking may be done. But all these are outward habiliments. The warm, breathing, vital entity is science—the medical sciences or the science of medicine.

"Anatomy, chemistry and the rest are not preparatory to medicine, they are medicine itself. They are provinces, not independent kingdoms. Whatever is good anatomy is good medicine. Whatever is good physiology is good medicine. In giving the well-rounded training which will make his science a living entity, the anatomist or the physiologist or the pathologist or the chemist may be trusted to include the so-called practical matters which are the outward manifestation of medical practice, just as he may be trusted to do his best to instill the methods and powers of observation, experimentation and orderly reasoning which should be the inward essence of medicine itself. He may be trusted, I add, so to steer the ship of research that, though it skirt uninhabitable shores and though it penetrate many an unpromising inlet, in the end it will discover safe anchorage and will chart out passages by which the commerce of applied science may pass into now unknown seas.

"Moreover, if my thesis is even approximately current, the professors of surgery and medicine should expect to teach anatomy, physiology, pathology, et cetera, as part of their regular work. Instead of complaining because the students who come to you do not know it all, you should look on yourselves as duly appointed to teach these sciences and to continue the training of the juniors and seniors along the very lines laid down in the first two years. Thereby the marked break at present existing between the first two years and the last two years would in large measure be avoided.

"In the text of this homily lies, furthermore, the best argument for the unity of medicine. Did anyone ever hear of chiropractic chemistry or osteopathic physiology or even homeopathic pharmacology? There are no sects in science. There will be none in medicine when medical men and their patients appreciate what medicine is and what it can do and cannot do. That joyful but distant millennium awaits the universal development of the scientific mind. It may come. Who knows? Think of the progress of the last four hundred years. Let us begin by making the doctors more scientific.

"This is my thesis, my creed, my optimistic hope. It is in the failure of us science teachers to realize our part of it, that I find my moments of discouragement. In how many of our students do we fail to establish logical habits of thought, fail to found a confident reliance on observation accompanied by an appreciation of the limitations and pitfalls of the senses, fail to awaken the imagination and to kindle such a fervor for science that the constant cry shall be, 'What, How and Why,' fail to establish sleepless guard against that insidious enemy of the understanding, *post hoc ergo propter hoc*—in how many of our students, I say, do we science men fail to do these necessary things? The number is large, I am sure."[5]

Next, I would like to quote from a paper which Dean Lyon gave in October, 1917, at the Conference on Hospital Standardization of the American College of Surgeons: "I am pleading," he said, "that hospital staffs consciously undertake their teaching obligation to the intern, as they consciously take up their other duties connected with the hospital and with the practice of medicine. I do not say that the intern needs formal lectures, but I do say he needs inspiring leadership. He needs to have his eyes opened to all the world around him; his imagination quickened to perceive what is beyond present seeing. He needs to have his footsteps led now forward to the new and the unknown, now backward to that repository of truth, the library, with its records of research, biography and medical history. I do say that every hospital staff should be a medical faculty. I do say that the man who has nothing to bring to others, has nothing to bring to a hospital, and has no place on its staff. I plead for the teaching function of a hospital as co-ordinate and co-equal with its other functions, and I demand a high valuation of this function in the standardization of hospitals. I demand that data on the education of interns be sought and evaluated by your committee and investigators."[4]

I might comment that since 1917, considerable progress has been made in the education and training of interns. On the other hand, this statement as well as many others that Dean Lyon made in his early years are just as pertinent and just as applicable today as they were when he made them.

One of Dean Lyon's very special characteristics was his social and educational philosophy. For a glimpse of this, I quote from a commencement address he gave to the graduating medical class at St. Louis University in 1920. To these graduating students he said, "It is going to be an interesting age for you to live in. I was told that when I graduated. Every age is interesting for those who live in it. Your age is going to be 'different.' I was told that also. Every age is different; for civilization, like life, is a dynamic affair and always attaining—never completed.

"Some people think the social power-house is going to blow up and smash things generally. It is true the temperature just now is pretty high, and some foaming takes place in the steam-chamber. I think the heavy weight of war-time necessity ought to be taken off the safety valve. The excess pressure ought to be allowed to escape through our time-tested mechanism of free speech. I think the governor on the engine should be readjusted with tolerance and mutual sympathy. But I think the machine will presently cease to creak so much, and that evolution rather than catastrophe will describe the changing order, as you view it in your lifetime. No one is going to burn down the universities nor tear up the railroads nor destroy the factories nor kill all the people who wear good clothes.

"A new equilibrium will tend to be established. I say advisedly 'tend to be established'; for the social organism, as I just said, is like the biologic organism—never static but always in process of adjustment. And the forces of social metabolism are going to be the old, wholesome ideals of the home, the family, property, religion, law, regard for our neighbors, love of country, self-respect and hard work. Some of these ideals are going to be broadened. I suspect that our concept of neighborliness will undergo very striking modifications in the form of social legislation. I suspect our notions of property may change. I suspect that our ideal of patriotism may become different, as did the ideal of a citizen of Massachusetts or of Virginia in the early days of our constitutional government.

"Perhaps the hide-bound conservative with his blindness to human needs and his repressive tendencies is valuable as a drag upon the ship of state. I am doubtful about that. Perhaps the blatant radical with his frequent disregard for truth and his impractical schemes is valuable as a ferment and stimulus in the body politic. I am doubtful about that also. But I have no doubt of the value of the man who, with scientific candor, a love of truth and a calm mind, examines the phenomena of human relationship and forms strong opinions on political and social and moral and religious questions. That is the kind of men I think your education has fitted you to be.

"Be open-minded. Be not afraid to speak out, but have the facts first. Look to the past; but live in the present—just a little ahead of the present. Hold what is good; but reach out for something better.

"These remarks you may recognize as a paraphrase of the writing of a man named Paul, who lived a long time ago. He was a professor in the University of the World and a very wise man. Nowadays we might call him an exchange lecturer; for he went from place to place, speaking to those who wished to learn. 'Prove all things,' he said; 'hold fast that which is good.'

"I think Paul made the best commencement speech ever delivered. The gist of it is in the first three words: 'Prove all things.' That's the task. Most people do not prove things. They jump at conclusions. They hitch effect in front of cause. They see cause and effect as unrelated phenomena. They magnify the unimportant. The rabbit's foot and the ouija board are emblems of loose mental processes, from opposite ends of society. 'Prove all things,' said Paul. That is the scientific ideal! and its basis is the love of the truth. It takes a trained mind to prove things. You are trained and proving things should be your life work. 'Hold fast that which is good.' That is easier to do. It's just good common sense. Educated people ought to have common sense.

"The amalgamated experience of trained minds constitutes that 'common sense of most,' of which Tennyson speaks. It is the 'enlightened public opinion' on which democracy is founded. If you of this graduating class and of those others by thousands who in America receive, through the years, the benefits of higher education will, first of all, hold fast those nine simple words of Saint

Paul, we need have no concern for our country, nor for the world."[3]

Finally, a few quotations from an address Dean Lyon gave at the Congress on Medical Education just prior to his retirement in 1936. The title he gave to this address was "Swans Sing Before They Die." "This title," he said, "may need explanation. It should be recalled that this is only a partial quotation. The full stanza runs as follows:

> Swans sing before they die,
> 'Twere no bad thing
> Did certain persons die
> Before they sing.

I suggest the hypothesis that I had in mind in the latter part of the verse rather than the doubtful biology of singing swans.

"I am one of the oldest, perhaps the oldest, of medical deans in point of continuous service—the dean of deans! This connotes no special merit but points to a certain agility, adaptability and power of endurance.

"I first planned to portray the personages who have acted in the panorama of medical education, great men who have done much for medical education, have finished their parts and left the stage. However, I decided that the time is not ripe for history, that we are still in the midst of doing and that it is better to keep the eyes fixed ahead, therefore I shall speak of some of the problems of medical education which I consider important and which I hope the future may solve.

"The first concerns the choice and admission of students. This involves a responsibility on the part of college officers which they have rarely sufficiently realized and faced. It predicates decisions on which the whole life and work of young men and women may depend, and in which the future standards of medical practice may be foreshadowed.

"The great question emerges—can we get any better basis for admission than college grades, intelligence tests and interviews, for such as believe in that technic?

"We medical educators should be concerned; the American Medical Association should be concerned, for the individuals that will compose this great body are the product that is being manufactured in the medical schools. Until we are surer about our raw material, more exacting in our inspections, more clear as to what our product should be, the medical profession will average lower than it should in the great field test that goes on day by day in the fight for better health.

"Another feature which causes concern is the fact that medical education is in general restricted to those who have money. This fact means inevitably that some superior men cannot enter the medical profession and that the average standard is lowered thereby.

"When one remarks the hosts of inferior individuals who throng to college halls, bungle their opportunities and depart little better than they came, the tragedy of the situation becomes titanic. Under our system we can suggest nothing better than scholarships with a well organized arrangement for their distribution to the most able candidates. I firmly believe that at present ten million dollars would at no other point create such dividends of medical progress as in the search for and assistance of submerged superior ability.

"In general I am favorable to increased requirements. But before the three-year premedical course becomes embedded in actions of the Council on Medical Education and Hospitals or in the requirements of state boards I advocate that an alternative proposal be experimented with by some medical colleges. I would add another year to the medical course itself. I would transfer to the medical curriculum some of the courses now classed as premedical, such as organic chemistry, sociology and psychology. I would leave the mimimum entrance requirement at two years of college as now, but the transfer described would permit of a wider sampling of college offerings in the direction of cultural background. I think the added medical year would enable faculties to plan a better balanced medical curriculum. I would begin anatomy, biochemistry, perhaps bacteriology in this year. But I would devote at least half the time to subjects now considered premedical. Psychology, adapted to medical students and thought of as the basis of mental medicine in the same way as anatomy and physiology are basic to physical medicine, would certainly be required and in the medical curriculum.

"The question of overproduction of doctors is of the very greatest importance to medical students, to the medical profession, to the maintenance of standards of ethical practice and to the public welfare. I contend that there are already too many doctors, even if they were properly distributed and even if their services were available to all people, neither of which contingencies is true nor likely to be true under American conditions. If it is claimed that competition is the life of trade, I retort that competition may be the death of a profession. These are just bald statements. In many years at dean's desk, I have watched the increasing difficulty and discouragement experienced by young doctors in earning a living."[7]

There is much more that I would like to give you from Dean Lyon's writings and many other things that I would like to say about him. The limitation of time, however, precludes my saying much more. I just hope that I have been able to give you a bit of a picture of this man who served as administrative head of our medical school for almost a quarter of a century. Concerning his administration, President Lotus D. Coffman said at the dinner honoring him upon retirement that "Dean Lyon was always kindly and generous in dealing with others, professional and high minded in discussing the problems of the medical school, and friendly to students and staff. In his administrative capacity he kept his face to the future rather than to the past. A scientist in his own right, he knew how to evaluate the scientific efforts of others. He has been a university dean who administered the affairs of his college with no thought of gain to himself and a spirit of selflessness which others would do well to emulate, and his contribution to the university is as enduring as the benefits to medical science are to humanity."[1]

In medical education Dean Lyon long occupied a position of national leadership. He had the vision of new horizons and the courage to depart from traditional methods, yet he was always wisely conservative. He left enduring footprints in the sands of medical education and his imprint indelibly upon this medical school. This is in the records, but to the students who were privileged to know him, Dean Lyon's outstanding qualities were those of kindliness and personal, human interest. He always considered the welfare of the student of first importance and helped many a one over the rough spots on the road to a medical career.

Years ago I remember hearing a verse which it seems to me is applicable to my appraisal of Dean Lyon here this evening. This verse was something like this:

> A King once said of a prince struck down,
> 'Tis taller he seems in death:
> And this speech holds truth for now as then,
> 'Tis after death we measure men.

And so a decade and a half after his death we can say of Dean Lyon that the shadow of his accomplishments and influence have lengthened rather than shortened with the passing of time.

Certainly, therefore, it is most fitting that Dean Lyon's name should be associated with the future as it has been with the past of this Medical School. And it is a great privilege for me and for all of us to dedicate this new research building in his honor as the Elias Potter Lyon Research Laboratories.

References

1. Coffman, Lotus D.: On Elias P. Lyon's Retirement. Minn. Med., 19:791-792 (Dec.) 1936.
2. Gray, James: The University of Minnesota, pp. 172-176, 1851-1951.
3. Lyon, Elias P.: Commencement Address, St. Louis University, June 7, 1920.
4. Lyon, Elias P.: Conferences on Hospital Standardization, American College of Surgeons, October, 1917.
5. Lyon, Elias P.: The relation of the laboratory courses to the work of the clinical years. J.A.M.A., 46:629-631 (Feb.) 1916.
6. Lyon, Elias Potter: Retirement Address. Minn. Med., 791 (Dec.) 1936.
7. Lyon, Elias P.: Swans sing before they die. J.A.M.A., 107:1498-99 (Apr.) 1936

AUXILIARY LAUNCHES "TODAY'S HEALTH" TEST

As a special test program of the national *Today's Health* committee of the Woman's Auxiliary to the AMA, each physician and dentist in Virginia not now subscribing to the magazine will receive a six-month subscription for his reception room. The offer will begin with the July issue and run through December.

During the fall months, members of the local auxiliaries in the state will personally contact each physician and dentist to take subscription orders for next year. It is hoped that as a result of this test, *Today's Health* will win many new friends during the coming months among both patients and the professions.

MID-PELVIC CONTRACTION AND LABOR

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IN RECENT years, the active obstetric staff of Maternity Hospital of Minneapolis has been disinclined to recommend elective cesarean section for patients on the sole indication of contracted mid-pelvis. As a result, many such patients have been given a trial of labor.

The Mid-Plane of the Pelvis

The mid-plane is generally regarded by obstetricians today as one extending from the lower inner border of the symphysis pubis posteriorly through a line connecting the ischial spines to a point on the anterior surface of the sacrum near the junction of the fourth and fifth sacral components, depending on the length and shape of the sacrum. The distance between the ischial spines is called the bispinous diameter. The anterior-posterior diameter of this plane extends from the lower inner border of the pubis posteriorly to the point on the sacrum mentioned above. The posterior sagittal of this plane is that portion of the anterior-posterior diameter extending from its intersection with the bispinous to the point on the sacrum mentioned above.

Hanson[4] was one of the first to recognize that certain difficulties connected with pelvic delivery might be related to abnormalities present in the mid-pelvis. In 1930, he described an instrument he had invented with which he could manually measure the distance between the ischial spines. The instrument was never generally popular, although its inventor was able to obtain quite accurate measurements with it. DeLee also invented an instrument for the same purpose.

There is a great range of interest in x-ray pelvimetry. It varies from those who never use it at all, to those who advise elective cesarean section for patients having small mid-pelvic measurements. Of the latter group, McKelvey[7] advised section in patients with a bispinous diameter of less than 9 cm., who seemed to have a fetus weighing 3500 grams or more. Perhaps the most concise evaluation of the place of x-ray pelvimetry in obstetrics was done by DeLee,[13] who is quoted as saying: "Since the x-ray cannot measure the strength of the pains, the moldability of the head, or the expansibility of the pelvic joints, or the mutation of the sacrum, or the rigidity of the soft parts, or the metabolism of the parturient, et al, you can see what a small part it plays in the routine of delivery. But I would not be without it."

Patients Who Should Have X-Ray Pelvimetry

The following are indications for x-ray pelvimetry:

The nullipara at term with an unengaged fetal head.

Most breech presentations, particularly those in nulliparae.

In patients where there has been a history of a previous dystocia or an unexplained stillbirth.

When on bimanual examination the sacral promontory can be reached.

When the sacral cavity seems shallow or flattened.

When the ischial spines are unusually prominent.

When the bituberous diameter (the transverse of the outlet) is 8.5 cm. or less.

During labor if progress is unsatisfactory.

Criteria for Contraction of the Mid-Plane

Eller and Mengert[3] gave the average measurements for the mid-pelvis as follows: bispinous (B.S.) 10.5 cm., anterior-posterior (A.P.) 11.5 cm., and posterior sagittal (P.S.M.) 5 cm. Weinberg[12] gave the same averages. Most authors are agreed that a bispinous of less than 10.0 cm. is evidence of some degree of contraction of that diameter. Dyer[2] found that in 197 patients with a bispinous of less than 10 cm., 30.4 per cent eventually had to be delivered by cesarean section. There is less agreement as to what constitutes contraction in the A.P. diameter of this plane. Thoms and Wyatt[11] consider this diameter as being contracted at different figures, according to the shape of the pelvis. With the dolichopellic type, if the A.P. of the mid-plane is less than 12 cm., with the mesatipellic, if less than 11.75 cm., and with the brachypellic, if the anterior-posterior diameter is less than 11.5 cm., contraction is present.

Pelvimetry Methods Used in This Study

The majority of the patients in this group had their x-ray measurements made of their pelves employing the Steele-Javert technic.[10] The last third of the patients were measured by the Hodges-Dippel direct parallax method, somewhat modified.[8] Any of the standard techniques will give comparable accurate measurements. Weinberg and Scadron[14] compared the results in 100 patients each measured with four different methods, and found each one accurate to within 0.1 cm.

Materials Used

For the purpose of this study, the records of all nulliparous patients who had had x-ray pelvimetry done at Maternity Hospital and delivered there, between the dates October 1, 1951, and October 1, 1953, were reviewed. There were 119 patients in this category whose mid-pelvic measurements were below the critical levels selected. Those levels were arbitrarily chosen as being less than 10 cm. for the bispinous and/or less than 11.5 cm. for anterior-posterior diameter of the mid-pelvis. All of these patients were in labor for some period of time and all of them eventually delivered either vaginally or abdominally in this hospital.

All of these patients had minimal amounts of analgesia during labor, usually one dose of Demerol or Nisentil with or without scopolamine. Sometimes in some of the longer labors, of course, the sedation was repeated. Occasionally, Trilene was also used in the first stage. For delivery, pudendal block with 1 per cent Procain (or Cyclaine) supplemented with nitrous oxide-oxygen inhalation administered during contractions and also during birth of the head. The sections were done either with spinal or local supplemented with gas-oxygen after delivery. It is felt that analgesia and anesthesia were not a factor in the production of the asphyxia present in some of the babies.

For this study, the term "Spontaneous Delivery Group" includes low forceps deliveries as well as the strictly spontaneous variety. The definition of low and mid-forceps is the same as that agreed upon by Eastman, Greenhill, Titus, and McCormick. The term "Difficult Delivery Group" as used in this study includes the mid-forceps, the breeches whose heads were delivered with some difficulty with forceps, and the cesarean sections.

Bases for Comparison

There are two methods currently popular which are claimed by their originators to be helpful in prognosticating the outcome of labor through the mid-pelvis. It is felt by the author that in some instances too much weight may be given to the prognosis based on radiologic findings in selecting patients for elective cesarean section, since there are so many other factors involved in a successful and atraumatic vaginal delivery. One can hardly believe that the originators of the methods to be discussed ever intended them to be used as the sole basis of predicting the outcome of labor.

Weinberg and Scadron[14] devised a method which used the sum of the bispinous and posterior sagittal diameters. They found that a narrow bispinous carried a good prognosis if it were compensated for by a generous posterior sagittal. They felt that delivery from below rarely occurred with sums of 13.5 cms. or less. The other method was devised by Mengert,[9] who considers the mid-plane roughly as an area and estimates it by multiplying the bispinous measurement by the anterio-posterior measurement. The figure obtained is then compared to his average one of 125. Eighty-five per cent is considered the borderline between adequacy and contraction. Mengert found by trial and error that mid-pelves having an index greater than 85 per cent were usually uncomplicated. Below this critical level they were much more likely to terminate in operative deliveries. Kaltreider,[6] using Mengert's index for the mid-pelvis found that when the inlet was adequate and the index for the mid-pelvis was over 85 per cent, in only 6.1 per cent was there difficulty. When the inlet was all right and the mid-plane index was less than 85 per cent of average, there was difficulty in 40 per cent of the deliveries. He had about the same results when applying Weinberg and Scadron's sums. When the inlet was adequate and the mid-plane sum was above 13.5 cm., there was difficulty in 6.3 per cent. If the sum was less than 13.5, there was difficulty in 30 per cent of the deliveries.

Results in Our Series

The length of labor for all patients delivering vaginally averaged fourteen and a half hours, and ranged from four to seventy-one and three-quarter hours. The average length of labor for the operative vaginal deliveries (mid-forceps and

TABLE I. MENGERT'S MID-PLANE INDEX

	Number	Difficult	% Difficult
Above 85% of normal	78	15	19
Below 85% of normal	41	17	41

TABLE II. WEINBERG-SCADRON MID-PLANE SUMS

	Number	Difficult	% Difficult
Over 13.5 cm. (BS plus PSM)	73	15	21
Below 13.5 cm.	46	17	37

forceps on the after-coming head) was twenty-two hours. There were eighty-seven spontaneous (or low forceps) deliveries, nineteen mid-forceps operations, four cases in which there was some difficulty in delivering the after-coming head with forceps, and there were nine cesarean sections. There were 109 vertex presentations, seven breeches, one brow, and two face presentations. The average length of trial labor before cesarean section was nineteen and a half hours. There were sixty-one patients with a bispinous of less than 10.0 cm. and only 11.4 per cent terminated in cesarean section. This was considerably less than Dyer's[2] rate of 30.4 per cent. There were five patients in this series who had bispinous diameters of less than 9 cm. and babies who weighed more than 3500 grams. All five delivered vaginally, although it is possible the largest might well have been better delivered abdominally. The babies in this group ranged in weight from 7 pounds 12 ounces to 9 pounds 5½ ounces, two were delivered spontaneously and the other three were delivered by mid-forceps. Two babies were mildly asphyxiated and in one this was classified as being severe. All lived and seemed quite normal when they were discharged.

Of the nine sections that were done, one was on a patient with a frank breech and poor labor, another had a face presentation with poor labor, and there were five patients who had vertex presentations but in whom the fetal head was not yet engaged at the time of the operation. Also, one section was done for a brow that had not engaged. Thus, there was only one patient who required a cesarean section because of difficulty incurred while the fetal head was attempting to traverse the contracted mid-pelvis. This patient had a 7 pound 15 ounce baby in LOT position with a bispinous of 9.5 cm. During the last ten hours of labor there was no progress, with the cervix at 5 cm. and the fetal head failing to descend lower than a plus 1 to 2 station.

TABLE III.

	Spontaneous	Difficult	% Diff.	Asphyxia	% Asphyxia
APM less than 11.5 cm. BS 10 or more cms.	47	10	21	7	15
BS less than 10 cm. APM 11.5 or more	19	7	37	4	21
BS less than 10 cm. APM less than 11.5 cm.	21	15	71	11	52

TABLE IV. NEONATAL ASPHYXIA

Type of Delivery	Number Cases	Asphyxia	% Asphyxia
Spontaneous or low forceps	87	7	8
Mid-forceps	23	11	48
Cesarean sections	9	5	56

As can be seen from Tables I and II, the incidence of difficulty increased markedly when the mid-plane index was less than 85 per cent of average, or, when the sum of bispinous and posterior sagittal diameters was less than 13.5 cm. However, it will be noted that the majority of the patients whose measurements fell below these critical levels still had uncomplicated deliveries.

As can be seen from a study of Table III, the degree of difficulty in delivery and the incidence of asphyxia are closely correlated. The lowest incidence of both was found in the group of patients who had an APM less than 11.5 cm. and whose BS was 10 cm. or more. The next higher rate of difficulty and of asphyxia was in the group where the BS was less than 10 cm. and the APM was 11.5 cm. or more. The group responsible for the highest incidence of difficulty and of perinatal asphyxia was that one in which the BS was less than 10 cm. and whose APM was less than 11.5 cm.

Maternal Results

There were no maternal deaths in this group. One of the patients who had a cesarean section had a febrile course and there were two patients who were febrile following spontaneous delivery (or outlet forceps). None of the patients who had been delivered by mid-forceps had febrile puerperia but one had dribbling of urine for a few days, and another suffered a complete tear into the rectum as well as atony of the bladder for a few days. Two other patients had atonic bladders for a day. These had also been delivered by mid-forceps. One of the patients who had a cesarean section vomited partially digested food during the procedure and subsequently developed aspiration pneumonia. Bronchoscopy was performed and the patient eventually recovered.

Fetal and Neonatal Statistics

There were no neonatal deaths. There was one stillbirth, the baby dying during delivery. A 5 pound 12 ounce baby was delivered by breech extraction and forceps to the after-coming head through a borderline pelvis.

There were twenty-three babies who showed some degree of asphyxia. Mild asphyxia was considered to be present when the child did not cry and/or his color and respiration were poor for one and one-half minutes or more. Severe asphyxia was said to exist when the above findings were still present after five to six minutes of treatment.

In the spontaneous or outlet forceps group there were six with mild and one with severe asphyxia. In the mid-forceps group, there were three with mild and eight with severe asphyxia. In the cesarean section group there were two with mild and three with severe asphyxia. Also, Table III should again be referred to, since it shows the incidence of asphyxia of the baby in relation to the degree of contraction of the maternal pelvis.

Summary and Conclusions

The records of 119 nulliparae delivering at Maternity Hospital between October 1, 1951 and October 1, 1953, and who had mid-pelvic measurements below certain arbitrary critical levels, were reviewed. The indices for the mid-planes were calculated as suggested by Mengert and these were compared in terms of percentages of his average. It was found that while there was a much higher incidence of operative deliveries below 85 per cent of Mengert's average value, there were some above this level with operative deliveries. Also, the majority of those below the 85 per cent level had uncomplicated deliveries. The same group of mid-pelvic measurements were then considered as recommended by Weinberg and Scadron, i.e., using the sum of the bispinous and posterior sagittal diameters. Although these authors had stated that delivery from below rarely occurred when this sum was less than 13.5 cm., it was found in this study that 63 per cent of the patients with a sum less than this had spontaneous or low forceps deliveries. Of the 119 patients only 9 required cesarean section for delivery or an incidence of 7.5 per cent. There was an increasing incidence of neonatal asphyxia in going from the spontaneous to the mid-forceps group. Curiously, there was an even higher rate of asphyxia in the babies delivered abdominally. When both the BS and APM diameters were below the critical levels chosen, the incidence of difficult delivery and of neonatal asphyxia were more than three times what they were when only the APM measurement was reduced and about twice as much as when only the BS diameter was contracted. When both of these diameters are reduced below the critical levels used in this study, one may expect 71 per cent of the deliveries to be difficult and 52 per cent of the babies to exhibit some degree of asphyxia. All of the babies who were asphyxiated, left the hospital in good condition. According to Benaron et al,[1] if the child survives the neonatal period it may be expected to develop normally, mentally and physically.

Measurements of the mid-plane of the pelvis obtained by x-ray are helpful in evaluating the probable outcome of labor in parturients but *only* when viewed in their proper perspective: i.e., when considered along with the estimated size of the fetus, its presentation and position, and the quality of labor the patient is having. Given a normal pattern of labor with contractions of good quality, the majority of women can deliver an average size baby safely through a mid-pelvic plane which is moderately contracted. As a result of the decreased space available in this area, there may be an increased incidence of the fetal head becoming arrested in either an occiput posterior, or transverse position, thus necessitating rotation and forceps delivery.[5] A follow-up on the babies who had some degree of asphyxia is planned.

References

1. Benaron, H. B. W., Brown, M., Tucker, B. E., Wenty, V., and Yacorzynski, G. K.: Am. J. Obst. & Gynec., 66:551, 1953.
2. Dyer, I.: Am. J. Obst. & Gynec., 60:302, 1950.
3. Eller, W. C., and Mengert, W. F.: Am. J. Obst. & Gynec., 53:252, 1947.
4. Hanson, S.: Am. J. Obst. & Gynec., 19:124, 1930.
5. Hanson, S.: Surg., Gynec. & Obst., 59:102, 1934.
6. Kaltreider, D. F.: Am. J. Obst. & Gynec., 63:392, 1952.
7. McKelvey, J. L.: J. Michigan Med. Soc., 50:736, 1951.
8. McLennan, C. E.: West. J. Surg., 58:48, 1950.
9. Mengert, W. F.: J.A.M.A., 138:169 (Sept. 18) 1948.
10. Steele, K. B., and Javert, C. T.: Am. J. Obst. & Gynec., 43:600, 1942.
11. Thoms, H., and Wyatt, R. H.: Am. J. Obst. & Gynec., 59:424, 1950.
12. Weinberg, A.: Obst. & Gynec. Surv., 7:455, 1952.
13. Weinberg, A.: Obst. & Gynec. Surv., 7:455, 1952 (quoting Swenson, P. C.).
14. Weinberg, A., and Scadron, S. J.: Am. J. Obst. & Gynec., 52:255, 1946.

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Laboratory Aids to Medical Practice

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THE SURGEON AND THE BACTERIOLOGIST

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THE PRACTICE of making a bacteriologic diagnosis by having a technician in a general laboratory do a few stains and cultures on any type of specimen, suitable or not, depending on the whims of the clinician, is obsolete. Bacteriology as applied in modern clinical medicine is an extensive and complicated field and accuracy is important. The technical work must be done by, or under the direct and close supervision of, one who has had extensive training and experience in the field and who has the time and interest to evaluate new discoveries important in the field. In order to provide satisfactory bacteriologic studies, certain conditions must obtain, namely, the proper specimen must be provided, it must be adequate in amount to allow the desired studies as indicated by the history and pathologic examination, and the laboratory personnel must be prepared and willing to make whatever studies are necessary to determine the nature of the etiologic agent.

Proper Specimens

In order to facilitate provision of the proper material, a few suggestions may be helpful. It is well to aspirate the pus from an abscess and save a generous portion in a sterile, wide-mouthed, screw-capped bottle. Pus collected on a cotton swab is likely to be contaminated from the skin and will likely be inadequate in amount. It is unfair to the patient to send a sample of pus on a swab when large amounts are obtained during drainage and then discarded. It is desirable also to save a portion of the wall of the abscess for culture since it may contain more organisms than the pus. Draining sinuses should be curetted and in some cases extirpated. This avoids contamination from the cutaneous orifice and provides a more adequate specimen. Ulcers of the mucous membrane or skin should be biopsied after careful selection of areas of probable activity. A punch biopsy is easy to perform but may not provide an adequate amount of material; biopsy by means of a scalpel is generally preferred. Lymph nodes should be selected to obtain those showing evidence of recent activity; if available, several should be removed. A small portion of each node should be saved for histopathologic study and the remaining larger portion kept without contamination so that bacteriologic studies may be done if desired. Lesions of deep organs, such as lungs, liver, spleen, kidneys, salpinges, epididymides, bursae and bones, may be saved after histopathologic studies indicate they are likely due to an infection. A satisfactory policy is to place a generous portion in a wide-mouthed bottle in the operating room. This specimen may then be used for culture if needed.

Amount of Specimen

The specimen must be adequate in amount because experience has shown that in chronic infections one must always consider the possibility of tuberculosis, brucellosis or some mycotic condition, in addition to such common pyogens as micrococci, streptococci, *Salmonella, Shigella, Pasteurella, Klebsiella* and others. It is known that acid-fast stains frequently may fail to demonstrate tubercle bacilli and that positive results of stains are sometimes due to saprophytic acid-fast bacilli; therefore, this technique is unreliable for diagnostic purposes. "Sulfur granules" are not always present in actinomycosis and structures grossly similar to them may be due to many

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From the Section of Bacteriology of the Mayo Clinic.

other organisms. Fungi frequently cannot be found in the histologic sections although they are easily demonstrated by culture. Conversely, sections of tissue sometimes contain structures resembling certain fungi but not in characteristic form reliable for diagnosis. There is no characteristic histopathologic picture in chronic lesions due to *Brucella.* Therefore, in order to make adequate bacteriologic studies for such a wide variety of organisms, keeping in mind they may be few in number, a generous specimen must be provided.

Sensitivity to Antibiotics

It is frequently important to isolate the causative organism to determine whether or not it is sensitive to certain antibiotics. It is desirable many times to reisolate the organism after a certain amount of treatment to determine if it has become resistant to the drug. The methods for these tests are varied; they present many technical problems for accuracy and are constantly changing. Each new antibiotic presents a new set of problems for evaluation.

Accessory Aids

In chronic infections, many accessory diagnostic aids may be of little value and even may be misleading. Positive results of a tuberculin test do not establish that a given lesion is tuberculous, and negative results do not entirely rule out the possibility of tuberculosis. Approximately a third of the patients seen at the Mayo Clinic who have localized lesions due to *Brucella* have no specific agglutinins against this organism. Patients who have osteomyelitis due to *Salmonella* also may have negative agglutination reactions for those organisms. Skin tests for the diagnosis of fungous infections are, in general, rather unreliable for establishing a positive diagnosis.

Histopathologic Examination

Histopathologic examination is of limited diagnostic value in most chronic infections. The pattern for tuberculosis is extremely varied and may be imitated by many other conditions due to certain viruses, bacteria and fungi. The histopathologic response in chronic brucellosis may range from one closely simulating caseous tuberculosis to one typical of extremely mild nonspecific inflammation. The problem related to the limitations of the histopathologic diagnosis of fungous infections already has been considered.

Bacteriologic Emergencies

Certain emergencies of a bacteriologic nature must be recognized by the surgeon. The development of pseudomembranous enterocolitis resulting from antibiotic therapy must be recognized early and a specimen of stool obtained immediately for Gram's stain and culture. The patient's future may depend on the alertness of the surgeon and the prompt co-operation of the bacteriologist. A delay of a few hours may cost the life of the patient. Tetanus and gas gangrene are also emergencies but the bacteriologist is of little or no assistance in these conditions. The surgeon must carry the entire responsibility, basing his judgment on the history and clinical findings.

Satisfactory bacteriologic studies of material obtained from surgical patients are not only extremely important in order to permit proper treatment but depend in a large measure on the co-operation of the surgeon in supplying plenty of the proper material.

AMA PREPARES TWO CIVIL DEFENSE BOOKLETS

What part the physician should play in civil defense activities is aptly depicted in a series of six articles which AMA's Council on National Emergency Medical Service currently is offering in booklet form. Reprinted from the *Journal of the AMA,* these articles discuss such aspects as organizing for civil defense, developing medical participation in civil defense, the physician's civil defense responsibilities, and the doctor and the improvised hospital.

In addition, the Council now has available the "Proceedings of the Medical Civil Defense Conference" which was held in February in Louisville, Kentucky. This should prove a valuable sourcebook inasmuch as it contains rather thorough discussions of atomic bombing, the threat of biological warfare, civil defense against chemical warfare, psychological warfare, and a case study of a typical state's civil defense organization.

Both booklets may be secured on request from the Council.

President's Letter

VETERANS' CARE REVISED

As of June 30, 1954, the contract of the Minnesota State Medical Association with the Veterans Administration, to provide home-town medical care for veterans in Minnesota, was cancelled by the Veterans Administration.

Physicians of the state association have already been notified of the changes now in effect, but a summary here may help to supplement this information.

At the implementation of the original contract in 1946, all members were automatically included as participating doctors, but that participation was not compulsory. The Veterans Medical Service Division in the State Office served as a clearing house for processing bills of members who provided medical service for veterans. Processing was speeded up by this method, and physicians did not need to deal directly with the local offices of the Veterans Administration. The handling of vouchers, authorizations and billing were done on a group basis, thus eliminating the need for individual physicians to work directly with the Veterans Administration. All questionable cases were reviewed by the Veterans Medical Service Committee of the State Medical Association.

On an over-all picture, this arrangement has been workable, and, with a few minor exceptions, has worked satisfactorily for all concerned.

As of June 30, however, this arrangement was terminated. The Veterans Administration has been in contact with all members of the Association in an attempt to secure their co-operation as "designated physicians." Those who become such participating doctors will have to deal directly with the Veterans Administration, submitting bills, requesting authorizations for service, and receiving payment for service.

The Association feels that through this contract, it has rendered immeasurable service to the physicians of the state and to all veterans using this service. The State Office appreciates the degree of co-operation which physicians of Minnesota have given in making this service valuable.

It is felt that this contract was also of great help to the Veterans Administration in assisting them in securing adequate treatment and examination for veterans at government expense. Now that this relationship is terminated, physicians should realize that the state association has no official position to maintain, nor any obligation to mediate disputes over provision of service or payment for service with the Veterans Administration, as was the case under this contract.

President, Minnesota State Medical Association

Editorial

Arthur H. Wells, M.D., *Editor;* Henry G. Moehring, M.D., and John F. Briggs, M.D.

BACTERIAL ASPECTS OF ACUTE GLOMERULONEPHRITIS

THE HEMOLYTIC streptococcus is a versatile organism. It infects a number of animal species and in man results in such diverse clinical pictures as scarlet fever, erysipelas, rheumatic fever, and acute glomerulonephritis. For some time, investigators have been attracted by the possibility that some of these manifest variations in kind of illness might be explained by differences in the infecting strains. In the early days, bacteriologists went too far in this direction. Each strain was named according to the type of clinical disease with which it was associated. With the development of serological techniques for classification, it soon became clear that most hemolytic streptococcal infections in man are caused by organisms belonging to a single serological group, designated as group A, but it has been difficult to correlate clinical entities with any of the forty-odd serological types which are subdivisions of group A and are designated by number.

In scarlet fever, for instance, it was originally thought that only certain types of group A streptococci might result in a skin rash. It now seems that, although strains may at times vary in their ability to produce erythrogenic toxins, scarlet fever can be produced by most of the common types of group A streptococci.

There has been considerable speculation concerning the possible existence of rheumatogenic and nephritogenic strains of group A streptococci. Outbreaks of streptococcal infection have been described in which an unusually low or high incidence of these non-suppurative complications has seemed to occur. One of the difficulties in evaluating such reports is that, in any one epidemic, one type usually predominates. Without a number of types producing simultaneous infections, it is impossible to be sure that some other factor was not operating. Another difficulty is one of definition. Without sufficient clinical and laboratory data and without careful definition of both the preceding streptococcal infection and the non-suppurative complication, it is impossible to arrive at an accurate attack rate. Furthermore, it is hazardous to compare attack rates obtained by different reporters who have used quite different criteria for determining the incidence of streptococcal infections and of their sequaelae.

There is little evidence that strain variation plays a significant role in the incidence of acute rheumatic fever. Careful studies of populations in which infections with a number of types were occurring simultaneously have shown a remarkable constancy of the attack rate for rheumatic fever regardless of the infecting type of streptococcus.

In contrast to rheumatic fever, the attack rate of acute glomerulonephritis appears to be quite variable. Large-scale epidemics of streptococcal infection occurred in the Armed Forces during World War II with little or no acute nephritis. Both acute glomerulonephritis and acute rheumatic fever tend to occur in families, but acute nephritis may develop simultaneously in several members of a family, whereas acute rheumatic fever tends to appear at different times in the same family or in different generations. The former suggests a bacterial factor, whereas the latter suggests a genetic or host factor.

Interest in bacterial factors in the pathogenesis of acute nephritis has been revived by the evidence produced by Rammelkamp and his associates that several epidemics and many individual cases of acute glomerulonephritis have—in recent years at least—been preceded by infections with type 12 streptococci. Whether the nephritogenic property of this strain is dependent upon the serological property which makes it a type 12 is not known. In the epidemic at Red Lake Indian Reservation, described elsewhere in this issue, a group A streptococcus was found which so far is serologically non-typable. Concurrently a non-typable group A streptococcus was isolated from several members of a family in Minneapolis in whom five of the seven members developed acute glomerulonephritis. If these strains prove to be a new type, their nephritogenic potentialities may possibly be explained by some biological property which they hold in common with type 12.

The observations at Red Lake seem to support the opinion of some clinicians that streptococcal infections of the skin are particularly prone to result in acute glomerulonephritis. However, even with careful bacteriological studies, it is impossible to be certain whether such skin infections are primary or secondary since most patients with skin infections will show similar streptococci in the nose and throat, and conversely many patients with acute streptococcal infections of the upper respiratory tract will show large numbers of streptococci in cultures of the skin. This aspect of the problem warrants further study. It is an intriguing possibility that certain strains of streptococci which are nephritogenic may also have a predilection for infecting the skin.

The bacterial aspects of acute glomerulonephritis and acute rheumatic fever are not a matter of purely academic interest. To the bacteriologist and the epidemiologist, the association of these diseases with hemolytic streptococcal infections is quite regular. To the practicing physician, the association appears to be somewhat inconstant because close to half of all streptococcal infections are subclinical or so mild that they are never brought to his attention. It is nevertheless important for the clinician to be aware of the role of streptococcal infections in the etiology of these diseases because their control is primarily dependent upon the proper treatment and control of streptococcal infections.

Lewis W. Wannamaker

PRESENT TRENDS IN THE TREATMENT OF HYPERTENSION

THE AVAILABILITY of drugs which reduce the blood pressure of patients with essential hypertension has changed methods of treatment and permitted optimism concerning the results of medical treatment. A physician considering a patient with hypertension will of course search for pheochromocytoma, coarctation of the aorta or unilateral renal disease; surgical treatment of these conditions relieves hypertension in most instances.

Sympathectomy for hypertension has largely been abandoned because medical treatment relieves hypertension more frequently than does sympathectomy. However sympathectomy occasionally is advisable. Restriction of sodium in the diet also has been largely abandoned except in instances of congestive heart failure. The percentage of good results in reducing blood pressure resulting from restriction of sodium is small and other methods of treatment are usually much more productive. Too many patients are using a so-called salt-free diet without evidence of benefit.

It is difficult for a student of clinical hypertension to avoid annoyance because of extravagant claims made for preparations which sometimes reduce blood pressure by some manufacturers. According to the advertisements of some manufacturers, solution of the problem of essential hypertension is simple; actually it is more often than not complex. Annoyance is accentuated by the observation that many more physicians will read and believe advertisements than will read more honest and informed articles in medical journals.

Drugs are available which reduce blood pressure and which in general prevent the complications of uncontrolled hypertension. The hypertension of each patient is an individual problem; a drug which may be used successfully for one patient may be wholly useless for another. Dosage varies widely. Unfortunately physicians tend to prescribe an antihypertensive drug and ignore the situation subsequently. This is bad therapy. The alert and skilled physician will persist in treatment; he changes medication and dosage until the hypertension is adequately controlled or until it is clear that medical treatment is valueless. Adequate treatment for hypertension requires co-operative patients and interested, informed and persistent physicians.

Drugs which reduce blood pressure in essential hypertension although they do not do this uniformly are: (1) Veratrum viride (example, protoveratrine); (2) Rauwolfia serpentina (examples, Serpasil and Raudixin); (3) ganglion blocking agents (examples, dibenzyline, hexamethonium chloride and pentapyrrolidinium) and (4) potassium sulfocyanate. They are all useful in treating hypertension.

No preparation has effects solely on blood pressure; they may cause distressing symptoms (which may be alleviated or prevented by appropriate treatment or provoke serious complications as, for example, lupus erythematosus.

No physician should use antihypertensive agents unless he has knowledge of their effects. The informed physician will be rewarded by doing a good job in the treatment of a disease which all too frequently is a serious one.

Edgar V. Allen, M. D.

IMMEDIATE BLOOD REPLACEMENT IN OBSTETRICAL HEMORRHAGE

In THE last annual report of the Minnesota Maternal Mortality Committee,[2] there were thirteen maternal deaths from post-partum hemorrhage (1951). That this is the greatest single cause of maternal mortality is pointed out by James J. Swendson[4] in an editorial in the March 1954 issue of *Minnesota Medicine,* in which he states, "it is a matter of concern that blood replacement is still often too little and too late."

With the increasing availability of blood during this past decade, largely accelerated through blood bank programs, the number of maternal deaths due to hemorrhage has decreased remarkably, but it is obvious that we will soon arrive at an irreducible minimum unless the blood bank program is further extended to the delivery room itself. This means maintaining Group O Rh negative blood in a refrigerator in the delivery room at all times for immediate emergency use.

Such a system has prevailed for the past ten years at St. Mary's Hospital in Rochester, Minnesota. Two bottles of Group O Rh negative blood of low anti-A titer are refrigerated on the obstetrical floor. When the blood is sixteen or seventeen days old it is brought back to the surgical section to be utilized before its expiration date. A new bottle of blood then takes its place. The results have been very satisfactory, and it is reported that the transfusion reaction rate is no higher in patients who have not received cross-matched blood than in those who have received type specific cross-matched blood.

In 1950 a similar system was instituted at St. Luke's Hospital, Duluth, Minnesota, and more recently at St. Mary's Hospital of the same city. To the writers' knowledge, at the present time, there are but two delivery-room suites in the Twin City Hospitals that are so equipped. Group O Rh negative blood is available at other hospitals but is not kept in the delivery room. In many instances, there is unavoidable delay in obtaining it from the laboratory, particularly at night: i.e. the night supervisor must be called to unlock the laboratory door, blood is located with difficulty by changing personnel, and so forth. In some instances, night technicians must even be called from their homes so that their travel time is superimposed upon the time involved in cross-matching.

That all obstetrical patients should have blood typing and Rh determinations prior to delivery is an accepted requirement of pre-natal care. We readily concede to the argument that most obstetrical emergencies can be anticipated in time to allow for the routine cross-matching and the use of type specific blood. That this is a far safer procedure and much to be preferred is obvious, but there is also the occasional unanticipated and exsanguinating hemorrhage in which seconds lost in replacement of blood may mean serious damage from shock and even death.

Patton[3] has emphasized the fact that in small hospitals a Group O blood bank is adequate, using only O positive and O negative blood without cross-matching. Blood is preserved in ACD solution (citric acid, sodium citrate, and dextrose) and may be used up to twenty-one days after it is drawn. It is interesting to note that in his experience the incidence of transfusion reaction was less with the uncross-matched blood than when the type specific cross-matched blood was used.

Cole[1] of Cornell University, has pioneered this problem of early and adequate blood transfusions in obstetrics, and as early as 1947, he urged the immediate availability of Group O Rh negative blood in every delivery room. With the application of this policy in 68,000 deliveries at the New York Lying-In Hospital from 1944 to 1949, only one private obstetrical patient died of hemorrhage. We note that in 80,000 deliveries in Minnesota during 1951 there were thirteen hemorrhagic deaths.

We Minnesota obstetricians may well be proud of our low maternal mortality rate of .45 deaths per 1,000 live births. But as long as there is a single preventable death due to hemorrhage we are guilty of laxity. Medical centers throughout the country are now providing immediate blood replacement, as well as a recovery room for obstetrical patients. If Minnesota is to maintain its leadership in the reduction of maternal mortality, we must strive to attain equal or higher standards of medical care. It should not be necessary to lose one of our own obstetrical patients to spur us into action.

1. Douglas, R. Gordon, Landesman, Robert, and Cole, John T.: Hemorrhage of late pregnancy. California Med., 74:233-227 (April) 1951.
2. Maternal Mortality Committee, Minnesota State Med. Assn.: Minnesota maternal mortality study. Minn. Med., 37:131-135, 1954.
3. Patton, George D.: Operation of a Group O blood bank. Am. J. Obst. & Gynec., 59:693 (March) 1950.
4. Swendson, James J.: Maternal mortality factors. Minn. Med., 37:212, 1954.

Jane E. Hodgson, M. D.
P. Theodore Watson, M. D.

ROUTINE CHEST X-RAY EXAMINATIONS IN HOSPITAL ADMISSIONS

THE HOUSE of Delegates of the Minnesota State Medical Association at its 100th Annual Session, May 17, 1953, acting upon the recommendation of its committee on tuberculosis, approved the following recommendation:

> "Approval of a program of routine admission chest x-rays to be instituted in all hospitals in the state with the stipulation that methods of initiation of such a program be decided by the roentgenologist, the staff, and the administrator of the individual hospital."

Here again, medicine in Minnesota reiterates one of its early fundamental principles, "that the practice of medicine includes both preventive as well as curative medicine." The family physician here is, as he should be, the keystone in planning for better health in his community.

The role of the official and voluntary health agencies has become increasingly that of assisting and supporting the physician in carrying out his responsibilities in the field of preventive medicine through assistance in program planning and in development of facilities at the community level. Admission x-ray procedures now have the endorsement of many organizations interested in the health of the individual and the community, including the American Medical Association, the American Hospital Association, the American College of Radiology, the American Public Health Association, the American Nurses Association, the National Tuberculosis Association, the American Cancer Society, the American Heart Association, and the Minnesota chapters of each of these societies.

When programs were first developed to meet the problem of finding and assisting in the diagnosis of early tuberculosis, it was discovered that of procedures available, the x-ray was the one most easily applied for mass use. As a result, since World War II, when equipment became readily available, the mobile photofluorographic x-ray unit became a very useful tool in the detection of many previously unknown cases of tuberculosis through surveys of the adult population of entire communities, cities and counties. In addition, it served to develop a recognition on the part of the public of the importance of such procedures.

What is equally significant is the fact that the public as well as the medical profession came to recognize that an equal or even greater number of significant nontuberculous conditions were also found by such procedures. This is all the more significant these days when heart disease and cancer of the lung are assuming such important roles in public health. Unquestionably, this condition was a factor in the recent recommendation of the American Cancer Society that all men over forty-five years of age have a chest x-ray examination at least once every six months.

The development of routine chest x-ray examination in hospital admissions has still greater significance in view of the retrenching action of Congress during the past year in reducing federal appropriations in the field of public health and specifically in tuberculosis control. A severe reduction in the federal funds for tuberculosis control places the Minnesota State Board of Health in a position where it can no longer continue the operation of its three mobile photofluorographic units.

These units were taking approximately 180,000 x-ray pictures of Minnesota's population annually. In comparison, hospital admissions in Minnesota's 193 general hospitals total approximately 450,000 annually.

Routine hospital admission x-ray examinations disclose more than twice as many pathologic conditions in the chest as does the routine chest x-ray examination of the apparently well adult population. In Minnesota this procedure has additional importance to the hospital, in that the compensation laws of the State of Minnesota specifically provide that employes of sanatoria or institutions of the state, or any county, or municipal subdivision of the state, who contract tuberculosis while in the employ of such institution or subsequent to such employment, are entitled to care and compensation. This means that the hospital must pay for the entire sanatorium care of the employe and for three-fourths of his wages during the entire period of his disability. The courts in Minnesota, by their action have indicated that employes of all other hospitals and institutions are entitled to the same protection.

Many hospitals in Minnesota are already using the routine chest x-ray examination in all hospital admissions of adult patients. Experience indicates that in no instance where such routine procedures were instituted have they ever been discontinued. Charges for such services meet with general acceptance from both the patients and insuring agencies. For the cost involved, it is the most productive and economical of all hospital routine laboratory procedures. Staff members also find that, as a result of these programs, patients more readily accept the inclusion of the routine chest x-ray examination as part of a physical examination and evaluation in the hospital and the physician's office.

The establishment of such programs is a complicated procedure and varies with the number of admissions to the hospital. Detailed information is available from the agencies, both official and nonofficial, which are endorsing and supporting this type of procedure. Voluntary agencies have contributed financial support for these programs, and it may be safe to assume that they would welcome the opportunity to assist in the planning and development of a local hospital admission chest x-ray service.

R. N. Barr, M. D.

ADMINISTRATIVE EXPERIENCES WITH ROUTINE CHEST FILMS

FROM an administrative point of view, the policy of requiring routine chest examinations on admission to the hospital, as well as on entering the employment of the institution, presents no insurmountable difficulties. This journal* has given a detailed account of the setting up of such a routine in a long-existing hospital.

Space limitations forced us to choose for the photoroentgen unit a room some distance from the admitting office. This was a disadvantage while we were attempting to cover the service with admitting office personnel. It was soon found essential, however, to have a technician on hand during routine admission hours. Except for obstetrical cases and emergency, few patients are now missed on admission. Excellent co-operation from the obstetrical floors makes possible the x-raying of all their patients within forty-eight hours because of early ambulation.

The overall picture shows 85 per cent success in getting all patients x-rayed. This administrator will never be satisfied that there is adequate protection of personnel or other patients until the score has reached 100 per cent. The short-stay cases are probably the greatest weak spot. At the same time potential exposure of personnel is least protracted in this instance, a circumstance which allays our fears to a small degree.

In any new hospital program some medical staff opposition is to be expected. The doctors responsible for inaugurating the program at St. Joseph's are to be congratulated on their skill in the laying of the groundwork for acceptance of the plan. Only a minimal number of objections were made to the administrator. These were on the basis of "taking away work from the doctor's office". When it was pointed out that only rarely would the chest x-ray be indicated in the doctor's office and that he would in all likelihood send the patient into the hospital with orders for a standard film, objections ceased. Since no small film would be taken under these circumstances, the patient would have the same expense as before the introduction of the routine chest film on admission.

A word should be said about the advantage of using the photoroentgen unit for screening all employes, students, house staff—in fact all who come in contact with the patients. We have found the unit to be a tremendous saving in this regard, while it creates a feeling of security in the face of the ever-present problem of hospital liability. The very convenience of the unit makes it readily acceptable to new and old employes.

Three years' experience has given ample evidence of the indispensability of the routine chest film program. Tabulated summaries of results such as the one given in the article referred to above may be duplicated wherever the plan is being used. The administrative and medical staffs of St. Joseph's Hospital are convinced of the solid merits of the policy of requiring routine chest films.

Sister Antonius
St. Joseph's Hospital
St. Paul, Minnesota

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*Minnesota Medicine, 35:340-344 (April) 1952.

Medical Economics

Edited by the Committee on Medical Economics
of the
Minnesota State Medical Association
George Earl, M.D., Chairman

TOTAL OF LICENSED DOCTORS ESTABLISHES RECORD

According to a recent report on medical licensure of the Council on Medical Education and Hospitals of the American Medical Association, an all-time record number of physicians were licensed. A total of 218,522 physicians were licensed to practice medicine in 1953.

The report states that "of this total, 156,333 were engaged in private practice, 6,677 were engaged in full-time research and teaching and were physicians employed by insurance companies, industries and health departments, 29,161 were interns and residents in hospitals and those engaged in hospital administration, 9,311 were retired or not in practice, and 17,040 were in government service.

Net Gain Noted

The data presented in the report showed that last year 7,276 physicians received their first license to practice medicine. In the same period there were approximately 3,421 deaths of physicians reported, thus showing a net gain of 3,855 in the physician population of the country and its territories and outlying possessions. This compares with the net gain for 1952 of 2,987.

From 1935 through 1953, the report stated, a total of 207,744 licenses to practice medicine was issued in the United States. During the same period there were 119,510 additions to the medical profession—an increase reflecting accelerated programs in medical schools, expanded facilities, and the licensure of foreign trained physicians.

Schools Rated Excellent

The report emphasized that the excellent rating of the nation's and Canada's approved medical schools was pointed up by the number of applicants who successfully passed examinations. It noted:

> "Thirteen per cent of the total number of applicants who took written examinations for licensure failed. . . . Only 3.8 per cent of the graduates of approved medical schools in the United States and 4.1 per cent of those of approved Canadian medical schools failed. In contrast, 50 per cent of those graduated from now extinct medical schools in the United States failed, as did 45.5 per cent of the graduates of foreign medical faculties, 70.2 per cent of graduates of unapproved U. S. medical schools no longer in existence, and 13.4 per cent of graduates of schools of osteopathy."

Reviews Foreign-Trained M.D.'s

The report also revealed that in many states the licensure of foreign trained physicians has been given serious consideration by the authorities and that methods are being developed to provide for the licensure of such physicians which will not lower the standards of medical practice in the United States. Several groups, such as the Council itself, the Association of American Medical Colleges and the Federation of State Medical Boards, are now engaged in discussions looking toward a re-evaluation of the problem created by the influx of foreign-trained physicians migrating to the United States to pursue their profession.

The report traces the increase in foreign-trained physicians thusly:

> "The number of graduates of foreign faculties of medicine examined began increasing in 1936, and by 1940 there were over three times as many tested as in 1936. . . . Beginning in 1944 the numbers examined began to decrease until 1951, when there was a noticeable increase, an increase again recorded in 1952 and 1953.
>
> "During 1953, 1,463 graduates of 175 foreign medical schools and seven licensing corporations of foreign countries were examined by 36 licensing boards. A total of 796 successfully passed the examinations; failures numbered 667, or 45.5 per cent. At no time during the last 24 years did fewer than 30.7 per cent of such graduates fail in a given year. . . .
>
> "This extremely high percentage of failures is a primary factor in the cautious attitude that has been main-

tained by licensing boards in admitting foreign graduates to the licensing examination," the report said.

Breaking the figures down by states, the report noted that the largest number of foreign graduates were examined by New York—450; Illinois examined 411, California 148, and Ohio 105. Foreign-trained physicians may apply for licensure to all but 11 licensing boards; most boards have stipulations which must be complied with prior to licensure examination, the report concluded.

Few Doctors Retire

Another report, issued by the Bureau of Medical Economic Research of the American Medical Association, has some relation to the above statistics. This report shows that 84.2 per cent of the physicians in the 65-74 age group were in active practice of medicine. There were, in 1950, 22,296 physicians in that age group, with 18,770 in active practice, according to the report.

The *Journal of the AMA* commented editorially on this information in a recent issue:

> "Only 15.8 per cent were not engaged in active private practice. Among these 3,526 not in active private practice were housewives and others who had practiced for only a few years, if any, and several hundred who were still employed by private or public employers; still others had retired from private or public employment, probably on a pension financed in whole or in part by the employer. . . .
>
> "If the pattern of 1950 is continued, eight or nine physicians out of 10 would be required to pay social security taxes but would receive no pension from age 65 to 75."

HOSPITALS SHOW ADMISSIONS INCREASE

A new record was set by American hospitals in admissions during 1953. According to the thirty-third annual report on hospital services of the AMA's Council on Medical Education and Hospitals, 19,869,061 patients were admitted, compared with 18,914,847 in 1952. A continued increase in the volume of hospital services was noted in the report. It states: "For the second time in history, the number of hospital births exceeded the three million mark—3,307,182 in 1953 as compared with 3,170,495 in 1952. This represents approximately 84 per cent of the estimated 3,910,000 annual births in the United States."

Other highlights of the report were:

1. 6,840 hospitals registered by the American Medical Association—2,136 classified as governmental and 4,704 as nongovernmental.

2. Gains in patient admissions noted in both groups but more pronounced in the nongovernmental division credited with 74 per cent of all admissions.

3. Bed capacity in registered hospitals increased by 31,399 over the previous year with a total of 1,573,014—1,113,004 in governmental and 460,010 in nongovernmental hospitals.

4. Average length of stay per patient was reduced in general hospitals from 9.8 to 9.3 days. Also included in this group are federal general hospitals which showed a reduction from 26.2 to 23.6 days.

In addition, the report states, many registered hospitals are supporting educational activities connected with training of interns, residents, student nurses, technical and other hospital personnel. 850 hospitals approved for internships; 1,146 for residencies, and 1,071 for professional nursing education.

NEW PAMPHLETS NOW AVAILABLE

Available in quantity from the State Office, 496 Lowry Medical Arts Building, St. Paul, are four new pamphlets suitable for doctors' offices or for distribution.

The pamphlets cover various aspects of medical public relations, and include one on research, one on choosing a family doctor before illness strikes, one on how the AMA stands guard behind the doctor's prescription, and another on warnings about healers who guarantee cures. Physicians may order these pamphlets by writing to the above address.

MATCHING INTERN PROGRAM EXPLAINED

A recent Secretary's Letter from the American Medical Association carries an explanation of the purpose, attitude and philosophy of the National Intern Matching Program which has now been operating for three years. In order to explain how the program works and its purpose, the following information was submitted:

> "The National Intern Matching Program has now completed three years of successful operation. This agency is operated jointly by the AMA, the hospital associations and the Association of American Medical Colleges. Its sole purpose is to facilitate the orderly

placement of senior medical students in first-year internships.

"The plan preserves in the American tradition free choice by both hospital and student as well as their bargaining rights. It does not interfere with, but gives effect to the expressed preferences of both groups. The program does not approve, restrict, or in any way control the internships offered by hospitals. It does not allocate interns. Each student gets his most desired internship if that hospital wants him. Each hospital gets every man that it wants, up to the number it specifies, who wants to go there."

SECOND SCHOLARSHIP AWARDED IN DULUTH

The second annual Rural Medical Student Scholarship of the Minnesota State Medical Association was presented to Leland R. Christenson of Marshall, Minnesota, at the annual meeting in Duluth on June 8, 1954.

The award, begun last year by the Association, is given with the stipulation that the recipient spend the first five years of his practice in a Minnesota community of 5,000 population or less.

The 1954 award was presented at the annual banquet by Dr. F. J. Elias, chairman of the Association's Rural Medical Student Scholarship Selection committee. In presenting the scholarship to Mr. Christenson, Dr. Elias said, in part: ". . . we are pleased to know that this young man will be joining our ranks soon. The very fact that we have made this scholarship available, and have seen that the best qualified student should receive it, evidences our faith in the youth of this state."

FILM ON ALCOHOLISM ADDED TO AMA MOTION PICTURE LIBRARY

Case studies of three types of alcoholics tracing the development of the disorder from origin are incorporated in a motion picture film which recently was added to the AMA's Motion Picture Library. Entitled "Alcoholism," this film attempts to show how the roots of this illness are imbedded personality difficulties often relating back to the formative years of the victim's childhood and how it can be treated through psychology. This black and white, sound, 22-minute film may be obtained from the Committee on Medical Motion Pictures for a service charge of two dollars.

MINNESOTA STATE BOARD OF MEDICAL EXAMINERS

230 Lowry Medical Arts Building
Saint Paul, Minnesota

E. M. Jones, M.D., Secretary

CALIFORNIA MAN GIVEN WORKHOUSE TERM IN MINNEAPOLIS FOR ABORTION

Re: State of Minnesota vs. Vern E. Jorgensen

On June 3, 1954, Vern E. Jorgensen, forty-one, 543 Eureka Street, Lodi, California, an employe in a liquor store there, was sentenced by the Hon. John A. Weeks, Judge of the District Court of Hennepin County, to a term of not to exceed eight years in the State Prison at Stillwater, following his plea of guilty to an information charging him with the crime of abortion. The sentence was stayed, however, and the defendant placed on probation for a period of three years, provided that he serves the first year in the Minneapolis Workhouse, and in addition pays the sum of $1,000.00 to the probation officer of Hennepin County. Out of that money, $700.00 is to be paid to Hennepin County and $300.00 to the Minnesota State Board of Medical Examiners to defray the expenses of apprehending the defendant who fled Minneapolis last August. Just prior to being sentenced, Jorgensen admitted having been previously convicted of a felony. On February 5, 1948, he was convicted on an abortion charge in the District Court of Hennepin County.

In the present case, a complaint was issued on August 19, 1953, in the Municipal Court of Minneapolis charging the defendant Jorgensen and Lillian M. Mullane with the crime of abortion, following the hospitalization of a 26-year-old Minneapolis divorcee in General Hospital who was ill as the result of having submitted to a criminal abortion. She had paid $150.00 for the abortion. Mrs. Mullane, who acted as the contact between Jorgensen and the patient, was arrested and subsequently sentenced to serve a two-year term in the Women's Reformatory at Shakopee. Although a warrant was issued for Jorgensen's arrest, he was not apprehended until last April, at which time he was located in Lodi, California, where he was arrested on April 26, 1954.

Both Jorgensen and Lillian M. Mullane, fifty-seven, 9949 Maple Avenue, Bloomington, Minnesota, were previously charged with the crime of abortion in another case in the District Court of Hennepin County and on February 5, 1948, each was sentenced to a state penal institution. The defendant Mullane was required to serve part of her sentence in the Minneapolis Women's Detention Home. Jorgensen was placed on probation. Although neither of them has had any medical training of any kind, in 1947 Jorgensen posed for several months in Minneapolis as "Dr. Vern Roberts."

The Minnesota State Board of Medical Examiners wishes to express its appreciation for the splendid cooperation given in this case by Mr. Michael J. Dillon, County Attorney of Hennepin County, and also for the extra effort put forth by the Minneapolis Police Department under Chief Thomas Jones.

The Dean's Page

SOME WISE WORDS FROM DOCTOR BELL

Last month the Seniors about to graduate from the Medical School, were guests at a luncheon sponsored by the Minnesota Medical Alumni association. An annual event, the luncheon serves to introduce the students to the alumni association and its activities.

Officers of the alumni group asked Dr. E. T. "Tommy" Bell, our beloved Emeritus Professor of Pathology, to talk to the students briefly on this occasion. His simple, wise, and sincere words were an inspiration to all who heard them. All of us—students, practitioners, teachers—would do well to study them not once but repeatedly. In order that those who did not have an opportunity to hear him may read his comments, Dr. Bell has agreed to let them be recorded on this page. The following then is a direct quotation from Dr. Bell's address to members of the 1954 Senior Class in Medicine.

"Your long course of training has been completed, and you are now ready to start out in life. This is a happy day for you and also a serious day, because you must now assume responsibilities that are new. Although I have taken no part in your education, I still feel almost as much interest and pride in you as I have had in classes which I helped to educate. I think I shall always feel the same way about Minnesota graduates.

"What I say to you is given in the spirit of a father counseling his son when he leaves home to start out in life. Be honorable and true to the best standards of our noble profession.

"The Hippocratic oath is a solemn promise which has guided the best men in our noble profession for over 2000 years. Translated into compact modern language it means that you pledge yourselves to rigid honesty in the practice of medicine. It means that you will treat your patients to the best of your ability, that you will not neglect them or do anything not in their best interest.

"Your patient puts his health and even his life in your hands. You must justify his confidence and faith.

"You have a debt to society. The actual cost of your four years of training to the people of Minnesota is $12,000. Colorado charges the State of Wyoming this amount for training their students ($3,000 per year). You have paid only about 10 per cent of this cost. You were selected from many applicants because of superior scholarship. A great many young men and women were denied the opportunity to study medicine because of our limited facilities. This is another cogent reason for you to do your best.

"Your skill in the diagnosis and treatment of disease should increase with your years of experience, but you must work continuously for greater knowledge and skill. You must study all your life. You will never be too old to learn. Any older doctor will tell you that the greater part of his knowledege was acquired after graduation. Medicine is moving forward rapidly. You must keep up with the procession. The doctor who quits studying soon falls behind. What would a doctor be like today if he practiced with only the knowledge we had twenty or thirty years ago?

"Read medical journals, attend medical meetings whenever possible. Find time for an occasional postgraduate course. The practice of medicine is a pleasure to the man who understands what he is doing. The incompetent man gets only frustration.

"The field of medicine is now so great that no man can possibly recognize and treat all the infinite varieties of disease. If you wish to practice a specialty, a minimum of three years of postgraduate study is necessary. But you cannot all be specialists. There is a great demand for general practitioners. The people of the state resent the fact that we do not produce more men for general practice. They threaten us with socialized medicine if we don't fulfill this demand.

"General practice is a very satisfying career. If you expect to do general practice, which we hope most of you will undertake, a two-year internship with emphasis on internal medicine is excellent preparation. If you practice alone you will probably need consultation on at least 10 per cent of your more serious cases. Practice is much more satisfactory if you can take your serious cases to a hospital where x-ray and laboratory facilities are available.

"A satisfactory way to practice is to work in a congenial group or clinic and subdivide the work. In this way one need not cover so many fields.

"The old horse-and-buggy doctor didn't do much for his serious cases, but the people loved him. You can learn from him a very important lesson, that is, that personal interest and sympathy play a very important role in satisfying your patients. Be a human doctor, not an ivory tower man or a machine. You and I would go to the most competent man regardless of how he treated us, but the patients want sympathy and understanding.

"The practice of fee-splitting, or kickbacks, is a disgrace to our profession, and the few men who do this, discredit the rest of us. The doctor who wants a split-fee usually sends his patient to the surgeon who gives a kickback, not to the best surgeon. As his conscience weakens, he sends patients who do not need an operation. This practice is a violation of basic human rights. If your patient is seriously ill and you do not understand the nature of his illness, get the best consultation available. Remember that the patient's life may be at stake, and that a specialist might save him.

"One of the greatest evils in the medical profession today is unnecessary surgery. This has even attracted national attention. Thousands of normal organs are removed every year in Minnesota alone. The sinners are chiefly men who have had no formal training in surgical diagnosis. They have forgotten the Hippocratic oath and operate for a fee.

"Do not undertake any operation that you can't perform, and do not recommend surgery unless you think the patient needs it. Men who are thoroughly trained in diagnosis are seldom dishonest. They simply can't do things they know are wrong.

"In summary, my advice to you is to be competent and honest. Be human. Justify the confidence and trust of your patients. Follow the golden rule. You won't be happy unless you enjoy your work, and you won't enjoy it unless you are competent. We are proud of you as our boys and girls, and we have every confidence that you will reflect honor on the University of Minnesota. God bless you!"

HAROLD S. DIEHL, M.D.

Dean of the Medical Sciences
University of Minnesota

Minnesota Academy of Medicine

Meeting of October 14, 1953

The regular monthly meeting of the Minnesota Academy of Medicine was held at the Town and Country Club on Wednesday evening, October 14, 1953. Dinner was served at 7 o'clock, and the meeting called to order at 8:10 p.m. by the President, Dr. E. A. Regnier.

There were forty-two members and one guest present.

Minutes of the May meeting were read and approved.

The President announced the death of Dr. A. E. Benjamin, for many years a member of the Academy.

The scientific program followed.

Dr. Ejvind Fenger, Assistant Medical Director of Glen Lake Sanatorium, read his Inaugural Thesis.

THE NECESSITY FOR TEAMWORK IN THE MODERN TREATMENT OF TUBERCULOSIS

EJVIND FENGER, M.D.

Oak Terrace, Minnesota

Of all the many statements made by Sir William Osler on the subject of tuberculosis, perhaps the most often quoted is this: "It is just as important to know what is in a man's head as what is in his chest, if you want to predict the outcome of his pulmonary tuberculosis." Long before the advent of the term psychosomatic medicine, it was known that not only physiological characteristics, but environmental factors and psychic reactions contribute to the tuberculous patient's progress. These social and psychic factors were considered a phenomenon of the disease.

As understanding grew, the physician realized that his clinical treatment had to be supplemented by other than purely medical services. It was the whole man who had to be cured.

This idea is developed in an interesting way in a recent book by Dr. Rollo May, MAN'S SEARCH FOR HIMSELF, in which he says, "It is not the attitude of 'My body feels' but 'I feel.' The old expression 'I sicken' is accurate. Fortunately in at least one disease the active verb is still used for the process of getting well—tuberculosis patients say 'I cured' at such and such a sanatorium. We propose that illness, whether physical or psychological, be taken not as periodic accidents which occur to the body, but as nature's means of re-educating the whole person." He goes on to say, "We may add that it is an actual clinical fact that some persons, viewing their illnesses as an opportunity for re-education, become more healthy both psychologically and physically, more fulfilled as persons after a serious illness than before."

Perhaps this is more true of tuberculosis than most diseases. It is the basic idea on which all the services of the modern sanatorium are built, and it is also the basis for the subject which I wish to discuss with you—The necessity for teamwork in the modern treatment of tuberculosis.

The sanatorium movement began with the idea of isolating the open case after the private physician had made the diagnosis. Once isolated, the patient could be conveniently ignored; treatment was bed rest, fresh air, food, and finally gradual ambulation. Most sanatoria were small, poorly staffed, and in many respects just rest homes.

Experience and medical advances have painted a very different picture today. Diagnostic surveys ferret out asymptomatic cases. Treatment is varied, with drugs and surgery playing a prominent part. The modern sanatorium is a well-equipped hospital. Realizing that personal, social and vocational adjustments are essential to arrest the disease and avoid relapses, administrators augment the non-medical staff with social workers, occupational therapists, vocational counsellors, teachers and librarians.

This paper is devoted to an analysis of the teamwork at Glen Lake Sanatorium, Minnesota, presentation of an illustrative case, and discussion of needs for greater integrated effort in some aspects of tuberculosis care.

Glen Lake Sanatorium staff may be divided, for convenience, into three general categories, medical, social service, and vocational rehabilitation, with considerable inter-departmental co-operation. The maintenance staff is, of course, just as essential to the team, but it will not be discussed in this paper. There are 626 employes in all for 619 beds, with operational expenses per patient of $13.00 per day.

Medical

The medical department is a vastly more complex organization than in the days when tuberculosis was isolated and ignored. Diagnosis has spread its fingers into the "normal" community, with tuberculin testing in the public schools and by the private practitioner and roving x-ray survey units. Public clinics maintained by the Health Department, sanatoria, and charity hospitals, and the practice of taking admission chest films at all hospitals have aided in earlier detection. Glen Lake Sanatorium's 1951 admissions came from: the Public Health Center's daily chest clinics (41 per cent), private practitioners (30 per cent) and other hospitals (22 per cent). Out of 444 admissions in 1952, seventeen were discovered by survey methods.

On admission, the case is assigned to one of five teams of three doctors each, which is responsible for the patient's care throughout his stay, but major decisions are made by the entire staff at weekly conferences. Each of the teams is also responsible for some special field of tuberculosis care, such as orthopedics or bronchoscopy, and there is free use of outside specialists. Consultation totals for 1952 include orthopedist 510, urologist 356, cardiologist 211, and frequent referrals to dermatologist, psychiatrist, ophthalmologist, and gynecologist. Two dentists give full-time service.

Laboratory and pathology facilities are well developed at Glen Lake (smaller institutions obtain some services from state laboratories). X-ray employs three full-time technicians and additional aides, with a radiologist reading each film. Equipment is available for stratigrams, stereoscopic views, and fluoroscopy as well as the usual chest films. Audiograms regularly check streptomycin's toxic effects.

Since a large proportion of patients now undergo pulmonary resection (resections in 1952 totaled 168), the surgery team compares with that found in general hospitals. We have two chest surgeons who operate several cases each week. Work-up includes development of some exercise tolerance, evaluation and correction of fluid-electrolyte and nutritional disturbances, the usual blood studies, crossmatch for transfusion, vital capacity and electrocardiograms for all patients and bronchoscopy for most. In 1952, 195 bronchoscopies and forty-three bronchospirometries were completed. A skilled anesthesiologist is available for all major surgery. Postoperative care essentials are: modern recovery room techniques, early emphasis on arm exercises preventing stiffness and pain, and close attention to the patient until suction drainage tubes are removed. Complete microscopic and bacteriological studies are performed on all surgical specimens. A state law passed in 1941, provides for transfer of surgical patients to sanatoria with these facilities when home institutions lack them.

A preventive program of B.C.G. vaccine inoculation is conducted at the sanatorium. All employes must have positive Mantoux reactions; non-reactors are given B.C.G. Selected children of positive sputum patients, who have had or will have heavy exposure, are tested and vaccinated at the institution.

The medical staff is active in clinical research, now they are participating in a United States Public Health study of chemotherapy, and completing a survey of bone tuberculosis cases from 1928-47. Frequent panel discussions and lectures by distinguished visitors provide new ideas and test old ones.

Over-all policies are reviewed at monthly staff meetings. The medical staff also sits with the education committee, approves rehabilitation programs, prescribes occupational therapy, helps with nursing education, gives radio talks, and contributes articles for the monthly newspaper published by the patients.

The nursing staff is composed of registered nurses on all floors in the daytime, with practical nurses on noncritical floors at night, supplemented by practical nurses, aides and janitor-orderlies. The practical nurses and aides are carefully trained in isolation technique and the details of tuberculosis care. By affiliation, student nurses from several teaching hospitals receive instruction at Glen Lake.

While all facilities in the state are not equally well developed, there is increasing effort to offer modern care everywhere. The Minnesota Tuberculosis and Health Association conducts clinics, helps with public education, and provides diagnostic surveys in unorganized counties. The Minnesota Trudeau Consultation Committee visits sanatoria on invitation, to offer help in the diagnosis and treatment. Transfer of patients needing special diagnostic studies or surgery is freely arranged, with the method of financial adjustment set by law.

If the patient is referred to Glen Lake Sanatorium by a private physician, the physician is supplied with a case summary soon after the patient's admission, and urged to visit and consult with the staff. When the patient is discharged, a summary of his stay at the sanatorium is sent to the physician together with suggestions for frequent x-rays, rigid control of activities, to avoid exceeding exercise tolerance, and regularity in work, play and rest. If the patient's tuberculosis was service-connected, on discharge he is referred to the Veterans' Administration Out-Patient Department for follow-up. Charity hospital, Public Health Clinic, and non-service connected veteran referrals are followed at the Glen Lake Out-Patient Department where daily chest clinics are offered, pneumothorax refills done, occasionally drug therapy is continued, and physical examinations, x-rays, and laboratory work ordered regularly.

Social Service

A full case work staff is employed to handle the many problems resulting from the lengthy hospitalization and uncertain future of the tuberculosis patient. Glen Lake Sanatorium proper hires a director, four case workers, clerical workers and patient-employes to assist with office work and special services. The case load per worker is about 135. The out-patient clinic includes a director and a financial and residential investigator, three case workers, and a large clerical staff.

Social Service performs six major functions: (1) assistance in personal adjustment, (2) interpretation of the disease to patient, family, and community, (3) utilization of available social agencies and other community resources to help the patient and his family, (4) interpretation of other sanatorium services to the patient, (5) dissemination of pertinent social information to medical, nursing, education, vocational and occupational therapy departments, and (6) maintenance of religious services, entertainments and other morale-building features.

When the diagnosis of tuberculosis is made, the Financial and Residential Investigator makes a home call to establish eligibility, discuss financial plans, and explore resources for care of the family after the patient is hospitalized. Additional interviews with insurance company or employer may be necessary. Immediate referral to pertinent social agencies is then made, the information is reported to the Finance Board and the case worker at the sanatorium, and formal application is filed.

A public health nurse visits the patient's home to discuss the limitations tuberculosis imposes upon the family and to relay information on obvious social needs to the social service department.

Soon after the patient's admission, the case worker takes a personal and family history, explains sanatorium services, and discusses problems resulting from hospitalization. Contact continues throughout the patient's stay as problems arise.

A large part of the case worker's day is spent interpreting the disease to patient and family and interpreting the patient's social needs to the rest of the team. The case is discussed with the doctor, arrangements are made for conferences between the doctor and the patient's family, and conferences are held with other departments. In co-operation with the nursing staff, a co-ordinated effort is made to teach the patient about tuberculosis using interviews, booklets, questionnaires, nurse teaching, special articles in the sanatorium paper, lectures over the sanatorium radio station, and conferences to clarify medical problems as they arise.

Another large segment of the case worker's day is spent integrating the patient's care with other agencies. The most frequent participants are the Hennepin County Welfare Board for Aid to Dependent Children and Old Age Assistance, city relief department for family and individual help, private agencies such as Catholic Welfare Association, Family and Children's Service, and Jewish Family and Children's Service, and various health agencies and hospitals. Other agencies contacted frequently in the last six months include: Minnesota Society for Crippled Children and Adults, Minnesota Society for the Blind, Minneapolis Society for the Hard of Hearing, Legal Aid Society, American Red Cross, Minneapolis Church Federation, Hennepin County Court Commissioner, Division of Social Welfare, Hennepin County Probation Office, Immigration Department, State Board of Parole, Police Department, County Sheriff's Office, State Department of Veterans' Affairs, State and Federal income tax agencies, insurance agencies, Railroad Retirement Board, county welfare boards, and the Division of Vocational Rehabilitation of the State Department of Education.

The social work staff is directly concerned with providing weekly church services, weekly movies, special entertainment features such as the annual Shriner's Christmas party, shopping service, and supervision of visiting children. The department also advises the Trudeau Club, an organization of patients which publishes a monthly paper, arranges radio programs, sounds patient opinion on entertainments, and arranges picnics, parties and table decorations. Volunteer groups are supervised and provided with visitors' lists, and gifts are acknowledged and allocated.

Many months prior to discharge, plans are made for transfer of homeless patients to Sarahurst Boarding Club while they train or search for a job, investigation of housing facilities by a public health nurse with attempts to make necessary improvements, and referral to various social agencies for continued aid until the patient and his family are self-supporting. Post-discharge medical care is discussed with the patient and a social summary is sent to the Out-Patient Department and Veterans' Administration.

A staff of case workers at the Out-Patient Department continues social work started at the sanatorium, providing new services as the need arises. The services of the out-patient social workers are available to private patients on request, as well as to all patients followed at the out-patient clinics.

Vocational Rehabilitation

Therapeutic exercise, library service, public school education, occupational therapy, vocational guidance and vocational training are all part of the rehabilitation program.

Therapeutic exercise assignments are clerical work, elevator operation, mail service, switchboard operation, and transportation of litter and wheelchair patients to improve exercise tolerance and re-establish work habits.

Books and magazines are brought to the bedside as soon as the patient is well enough to read or study. There are 8,000 volumes in the library including occupational literature on fifty major work fields and ninety-four circulating magazines.

Occupational therapy begins six weeks after admission with opportunity to do light arts and crafts such as fly-tying, chip carving, needlework and knitting. Later leatherwork, sewing, ceramics and woodworking with hand or power tools is offered. The therapist visits every patient twice a week to promote hospital adjustment, aid in the development of work tolerance, restore lost skills or develop new ones useful in a vocation, and evaluate interests, attitudes and habits.

Bedside and classroom public school instruction is offered from elementary to high school level with special correspondence courses or vocational training arranged as needed. Homemaking and cultural courses are also available.

Vocational rehabilitation proper begins when the patient fills out a Vocational Rehabilitation Survey questionnaire which will reveal his education, vocational training, former occupation, prospects for future employment and job preferences. The patient's doctor then decides the amount of time that may be safely spent in study. Counselling is provided by a Rehabilitation Co-ordinator, a worker from the Minnesota Division of Vocational Rehabilitation assigned full time to tuberculosis, and a Veterans' Administration advisor providing counsel for service-connected cases. Psychological testing of learning ability, occupational interests, vocational aptitudes and personality help the patient select his vocational goal.

Representatives of the above services, medical and social service departments discuss a co-ordinated rehabilitation program for each patient at bi-weekly meetings. Special schooling and training is provided as soon as the patient's condition supports it. At discharge, the patient may enter a special technical, trade or professional school sponsored by the Veterans' Administration or the Division of Vocational Rehabilitation. A discharged patient without a home during this training period may live without charge at Sarahurst Boarding Club, operated by the Hennepin County Tuberculosis Association and financed by Christmas Seal funds.

Case History

J. E., a twenty-four-year-old white, married, truck driver was admitted to Glen Lake Sanatorium June 20, 1951, with pulmonary tuberculosis, moderately advanced.

When he was in the fourth grade a routine tuberculin test was reported positive and six annual x-rays showed stable childhood tuberculosis. At seventeen, he was rejected by the Navy and at eighteen by the Army because of x-ray findings. At nineteen, he began work in Minneapolis as a truck driver. At twenty-one, he was again called by the Army, rejected, and referred for check-up to a clinic in Saint Paul, to which he did not return for follow-up. Information on his status at that time is not available.

At twenty-four he was again rejected by the Army, and a private physician's examination revealed a spread of the lesion, though the patient had no symptoms except a cough productive of one tablespoon sputum daily for one year. He reported one sister with tuberculosis at Sunnyrest Sanatorium, Crookston, Minnesota. He was referred to the Public Health Center where admission to Glen Lake Sanatorium was recommended when it was established that he had been a continuous resident of Hennepin County more than one year. The importance of institutional care in the treatment of tuberculosis was discussed with him and a pamphlet clarified preparations needed before admission. The patient was assured that financial help would be arranged for his wife and child when his insurance benefits were exhausted, and his own care would be free since his savings were small. The financial worker then completed application arrangements and checked with the patient's employer to verify information and collect any benefits due.

Previous to his admission, a public health nurse called and reported that the family was living in a very small apartment with no yard, one child was thirteen months old and another on the way, thus financial aid would be necessary. She discussed tuberculosis with the couple, emphasizing home precautions such as isolation of dishes and use of tissues. Regular checkups for the wife and child at the Public Health Center, with consideration of B.C.G. for the child, were urged.

On admission, J. E. was interviewed briefly at the office, then given a bed in one of the admitting wards. No chest abnormalities were noted on physical examination. Admitting x-ray showed slight scattered infiltration in both upper lobes with no apparent cavities. Stratigrams showed two small cavities in the posterior segment of the right upper lobe, and scattered small densities anteriorly in the left upper lobe suggesting an older process. Sputum smears were negative, but cultures proved positive. Strict bed rest was advised.

The social worker assigned to him took family and personal history, discussed with him the available sanatorium services, and reported that Aid to Dependent Children benefits would go to his wife as soon as insurance ceased. Through the social worker's efforts, the family moved to a new housing project where they had more space and a yard.

Meanwhile, J. E. was learning about tuberculosis and its treatment. He filled out a survey questionnaire and looked up and discussed the questions he missed. Nurses demonstrated techniques for covering cough and sputum disposal, discussed services available, and showed him the limits imposed upon him by the treatment "strict bed rest." He read several booklets and articles in the sanatorium paper on the disease and listened to talks about tuberculosis on the sanatorium station. Thanks to movies, careful room placement, store and library service, he began to feel a little at home. He completed several occupational therapy projects with industry and skill. It was reported he was adjusting to routine well, understanding his disease, taking necessary precautions, and remaining at bed rest.

He filled out a Vocational Rehabilitation Survey form indicating he realized he would need to give up truck driving and had been promised an office job in the trucking company. The Education Committee reviewed his case, recommending business mathematics, business English and bookkeeping courses while at bed rest, later typing. His doctor predicted probable ultimate full-time work capacity in a sheltered occupation.

In November, 1951, the patient reported to his social worker that his wife was unable to get along on the money allowed her by Aid to Dependent Children, a reduction in income of nearly 75 per cent. This was discussed with the ADC worker who said the patient's wife budgeted money very poorly though advice on realistic budgeting was being given. A conference was arranged with the patient, ADC worker, and sanatorium case worker in which the allowances and limitations of the ADC program were explained. After participating in planning for his family, the patient was less resentful and promised to encourage his wife's budgeting efforts. At that time a special allowance for a baby-sitter was arranged so the wife would be able to visit two to three times a week.

A medical conference called to review J. E.'s progress after six months of bed rest noted some improvement of extent of infiltration and cavity size and decided he should have bilateral resection after four to six months of drug preparation. He was placed on antimicrobial drugs on January 26, 1952. His last positive sputum was found on March 28, 1952. The social worker contacted officials of the truckers' union who agreed to replace all blood used at the time of his surgery.

His total vital capacity just before surgery was 4,000 c.c. with 3,500 c.c. for the first second. On June 18, 1952, he underwent right apical segmental resection. The remaining right lung was grossly free of the tuberculous process. Some apical adhesions required suture to control bleeding after they were divided. The next day hemothorax dictated a thoracotomy to control the bleeding and evacuate clots. No lung bleeding points were found but many clots were removed, an intercostal bleeder was secured and a catheter left in place. The bleeding did not recur, and the patient's condition remained good with generous blood replacement. On June 24, 1952, an intrapleural pocket in the anterolateral right chest was located by x-ray and aspirated, yielding 200 c.c. of light brown fluid. An intercostal catheter was inserted into the space at the right anterior axillary line until drainage ceased. By July 1, 1952, all tubes were out, the lung re-expanded and x-ray showed clearing.

On October 30, 1952, a left thoracotomy was done with the preoperative intention of wedge resection, but extent of nodules on exploration dictated segmental resection of apical posterior and anterior segments of the left upper lobe. The postoperative course was uncomplicated.

The right lung specimen showed one small cavity remaining with scattered fibrous infiltration and nodules, all appearing chronic and inactive. The left lung tissue showed several nodules with no caseation. No acid fast bacilli were seen on sections, and smears and cultures were negative. Strict bed rest was resumed for six months, to be followed by gradual ambulation over an eight- to ten-month period. Total vital capacity eight months after surgery was 3,100 c.c. with 3,000 c.c. for the first second.

After his first resection, J. E. was referred to the Rehabilitation Co-ordinator for more specific vocational planning. Eligibility for re-instatement with the trucking company was verified, the employer recommending bookkeeping instruction and promised opportunity for advancement. The patient was assured that pre- and post-discharge training to qualify him for advancement would be provided. Tests revealed clerical and bookkeeping aptitude. After the second resection, his doctor approved one and one-half hours daily study. He was enrolled in bookkeeping, typing and social studies courses (he had completed his other studies successfully).

In May, 1953, when ambulation was started, J. E. found he had outgrown all his clothes. Replacements from the Sanatorium Clothing Fund were arranged by Social Service.

At present the patient is on four hours' exercise daily, continuing his studies (he plans to start business correspondence when time permits) and is looking forward to traffic school enrollment on discharge. He is asymptomatic, with negative sputum and clear x-rays.

Weak Links in Teamwork

While teamwork in the modern sanatorium has progressed to an astonishing extent in the last twenty-five years, there is, of course, room for improvement.

The team may overlook personal problems of patients more reticent than J. E. The patient may not understand the social worker's interest in his adjustment, thinking only economic problems are referred to her. Doctors and nurses are available, but may seem too interested in his disease process to discuss emotions. All personnel must be more aware of the devastating personal impact of the disease on many patients, that the unco-operative patient may be unhappy, not "bad," and that only by maintaining a close rapport with patients will emotional problems be brought to light as they arise and thus reduce the number of irregular discharges.

The education of the patient about tuberculosis always seems incomplete, in spite of a well developed interdepartmental program. Nurse teachers must realize many patients do not learn by reading or from one hearing; persistent daily emphasis is needed with repeated survey tests to check for weak spots. One wonders about the advisability of using classroom teaching for patients and perhaps their families, as is done for diabetics in many hospitals. Or perhaps, ambulatory patients might be utilized to help a busy nursing staff in teaching. Review with special reference to patients' own habits in such matters as personal sanitation and strict bed rest should be undertaken at regular intervals. Certainly, the facts needed to live with tuberculosis are few, but these few must be clearly understood and practiced.

Nearly ignored in the vocational rehabilitation program are mothers of young children. Homemaking courses are offered by the Education Department, but little additional individual counselling has been attempted, although the work of a new baby is the villain in many breakdowns. Rehabilitation funds should be available to provide help in especially busy households until discharged patients build up sufficient work tolerance to carry on alone. Case work before and after discharge should attempt to recruit available help to the home so that the mother is assured adequate rest and freedom from strenuous work for permanent recovery.

Certainly the alcoholic, supplied with his bottle by bootlegging friends or businessmen and leaving against advice on this artificial optimism, is not being rehabilitated with the present team facilities. It is to be hoped that funds will be appropriated for detention of these patients (a fifty-bed addition to Anoka Tuberculosis Unit has been suggested), and that when this is available, treatment of alcoholism as well as tuberculosis will be attempted.

Another weak link in rehabilitation teamwork is medical follow-up and supervision of private patients. With routine examination, x-ray and laboratory procedures at chest clinics, it is easier to remember all facets of discharged patient control. The private patient, however, is prone to economize on follow-up visits, and the physician in the press of other responsibilities may fail to look him up. Both clinics and private physicians all too often fail to impress ex-patients with the necessity of a regulated life free from excesses of work or play.

Summary

The modern sanatorium is an up-to-date hospital, not a rest home, because of the necessity of teamwork in tuberculosis care. The medical staff provides well-integrated general diagnostic and treatment measures, wide consultation service, radiology, dentistry, surgical teams trained in chest work, complete laboratory, technical and pathology facilities, skilled nursing, and active research programs. The social service staff provides assistance to the patient in personal and sanatorium adjustment, interprets the disease to patient, family, and community, utilizes community resources to help the patient and his family, and assists with religious services, entertainments, volunteer services, and other morale-building activities. The rehabilitation team provides counselling, aptitude testing, education and training in specific skills needed to hold and advance in a vocation suited to the individual's exercise tolerance on discharge, as well as occupational therapy, public school education, and library service. All these services, in turn, must be carefully integrated by frequent interdepartmental conferences on each patient's special needs and continued by private physician or out-patient departments after discharge.

A case report is presented illustrating the many-sided teamwork needed in the care of a "routine" patient.

Pitfalls in the rehabilitation of the patient with emotional problems, the practical education of patients about tuberculosis, adjustment of young mothers to tolerable work loads, the care of alcoholics, and supervision of discharged patients are briefly discussed.

Discussion

Dr. S. Marx White, Minneapolis: It happens to be my personal privilege to be actively interested in the affairs of Glen Lake Sanatorium for many years—since 1919, in fact. I have seen a remarkable evolution in the care and treatment of tuberculosis. Dr. Fenger has traced this evolution and has made clear to us how many forces play a part in the care of the tuberculous patient and in his restoration to an active life in the community, when this cure has been established.

One problem that plagued us in former years, was that of the patient who left the Sanatorium too soon—thus often only to relapse. The physician who sends the patient to the Sanatorium can be the largest factor in preventing this.

Before our own institution became the effective instrument it is now, I often sent patients to River Pines Sanatorium at Stevens Point, Wisconsin. Before doing this, however, I carefully gave this instruction: The sanatorium is not going to keep you one day longer than necessary. They can fill the bed the day you leave as they already will have an applicant for it. No living person knows the exact day on which your cure is so complete that you will not relapse. It is better to stay a few days too long than to make the mistake of leaving too soon. A few extra days in the Sanatorium is much less expensive than a relapse. Therefore, when the Superintendent comes to you and says: "You may communicate with your family and tell them you will be ready to leave on a certain date"—I want you to look him squarely in the eye and say—"Doctor, won't you let me stay another month?" Dr. Coons, then the superintendent at River Pines, told me at a national meeting in Washington that he did not have the usual trouble with my patients in regard to wanting to leave too early, and asked me why. I told him the instructions I gave.

I would like to have Dr. Fenger tell us whether there is as much trouble these days in that regard as there was then. Also, where does the post-sanatorium follow-up by organized health forces belong: in the hands of a sanatorium, out-patient department, or in the hands of a health department concerned with other and multiple health problems?

Dr. Thomas Lowry, Minneapolis: I would like to make two comments. One is that in my experience as a practitioner of internal medicine who has referred a number of patients to Glen Lake Sanatorium, the teamwork and development of peace of mind of the patients at that institution is exceedingly effective. Most patients approach the sanatorium with hesitation, not to say fear, and a large majority of them get over these emotions in the manner which Dr. Fenger has so ably described. My second point is derived from the fact that, like Dr. Fenger and Dr. White, I still belong to the school of thought which wishes to remain ultraconservative in the treatment of tuberculosis. Such an approach has been very well summed up by Dr. White in his remarks about instructing patients to request permission to stay in the sanatorium an extra month.

The only complaint which I have ever had about the management of any patient of mine at Glen Lake Sanatorium or at any other sanatorium, is that I have thought some of these people were discharged too soon. I wonder whether in our present concept of the treatment of tuberculosis, including the use of drugs, surgery, and all the other present day accessory measures, some of us and possibly some of the members of the sanatorium team are yielding to the temptation of trying to get patients out of the institution before they are entirely ready for discharge.

Dr. F. F. Callahan, Saint Paul: I think Dr. Fenger has pointed out very successfully that the modern sanatorium is not only a treatment center but an educational institution. It is there they begin to restore the patient as a useful member of society. There is no use in taking a truck driver and sending him back to that occupation after he has lost a lung. Patients of this type must be trained in a lighter occupation if they are to remain well.

Dr. Ejvind Fenger, in closing: We have always had trouble with the number of irregular discharges that occur at Glen Lake. That difficulty, however, is shifting. We have a different type of population. When I first came to Glen Lake, I had one floor of forty-two patients consisting of high school and university age groups. Today, with the same service, I have only three patients who fit into that age group. Now we have many, many patients sixty and seventy years of age and over. We likewise have many more federal transients and a higher percentage of our patients come from the lower economic strata of our city and county. In the survey, in eight months' time, 58,000 x-rays revealed thirty-two cases having active tuberculosis. Twenty-two of these cases were found in the city jail or the city workhouse. We have a bigger problem with alcoholics today. A higher percentage of our people, because of their older ages, are on pensions. Perhaps that has something to do with their ability to buy liquor.

As to Dr. Lowry's comments about not discharging the patient too soon, I see eye to eye with Dr. Lowry on these problems. I think I can consider myself a disciplinarian. Some of our staff feel that because of chemotherapy, the patient's stay can be shortened. I do want to emphasize that I am still a strong believer in conservative bed rest treatment. I am sure if Dr. Evans were here he would agree that I am very conservative in my over-all treatment.

Dr. J. Arthur Myers, Minneapolis (by invitation) gave the following address.

PRESENT STATUS OF IMMUNITY IN TUBERCULOSIS

J. ARTHUR MYERS, M.D.
Minneapolis, Minnesota

No sooner was the discovery of the tubercle bacillus announced than scientists, including Koch himself, began working on methods to immunize artificially against this disease. Jenner had produced an efficacious vaccine for smallpox and Pasteur satisfactorily prevented fowl cholera with a vaccine. Several French scientists in the 1880's reduced virulence of living tubercle bacilli by various methods and used them as vaccine. After introducing these organisms into animals they waited a time for immunity to develop, then injected virulent tubercle bacilli into vaccinated animals and controls. In due time the controls began to fall ill and when all were dead, the vaccinated animals, for the most part, appeared well. Enthusiasm ran high, for on the surface it appeared that the vaccine was effective. However, it was not long until the vaccinated animals began to fall ill and all died from tuberculosis. This dampened enthusiasm and repeated experiments using vaccine consisting of living tubercle bacilli, and in some instances, that composed of dead tubercle bacilli, resulted in the same failure. These scientists concluded that tuberculosis differs from such diseases as smallpox, typhoid fever and diphtheria in that an attack does not result in immunity.

Marfan established a law which stated that people who had healed scars from tuberculosis of the skin and those who had recovered from tuberculous lymphadenitis would never have pulmonary tuberculosis. This seemed like good evidence that immunity must be present in humans and therefore further interest was stimulated in attempts to produce it artificially. A large number of workers in various parts of the world entered the field. Simultaneously Koch and Trudeau worked with a substance later designated tuberculin. Trudeau published negative results and Koch positive. Koch was soon found to be in error, for no one since his time has been able to make an animal or a person more resistant than normal

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Presented before the Minnesota Academy of Medicine, St. Paul, October 14, 1953.

to tubercle bacilli by tuberculin. In 1891, Koch's phenomenon was thought to be a manifestation of immunity. Time proved that it was only a demonstration of allergy.

Workers in this country and elsewhere resumed the use of living tubercle bacilli with diminished virulence in the late 1890's and early 1900's. One of the most colorful demonstrations was that of von Behring, who maintained a culture of tubercle bacilli of human type over a period of six and one half years. When he thought its virulence was so reduced that it could safely be used as vaccine, he practically promised the world freedom from tuberculosis in a brief period. The German government constructed a large plant where this vaccine was made and from which it could be distributed to the world. Von Behring's conclusions could not be confirmed and the organisms in his vaccine not only produced disease among employes in his laboratory but also were transmissible from "vaccinated" animals to people.

Webb and co-workers administered living, virulent, human type of tubercle bacilli to animals and children. They believed that immunity could be established by giving a child one tubercle bacillus and gradually increasing the number. The results were such that this work was concluded following severe condemnation by Calmette of France.

In 1906, Calmette and Guérin obtained a highly virulent bovine type of tubercle bacillus which Nocard had isolated from a tuberculous cow in 1902. This grew luxuriantly on ox bile-potato medium and at the same time its virulence was reduced. These authors became enthusiastic about having reduced the virulence of this organism to such a low level that it would not produce tubercles in animal tissues. Moreover their experiments on cattle made them believe it was efficacious in producing resistance to tubercle bacilli.

Calmette and Guérin were so convinced that they had found a solution for a large part of the tuberculosis problem that they strongly recommended that this organism be administered to infants as a protection against clinical tuberculosis. Their integrity cannot be questioned for they were willing to risk their great names in 1921, when they designated this organism Bacillus Calmette Guérin. It has since been known as BCG. Three years later they declared it a virus fixé.

On entering the tuberculosis field professionally in 1920, two tasks carrying immense responsibility were assigned to me. One was to aid in the control of tuberculosis among students on the campus of the University of Minnesota and the other among the children of the city of Minneapolis. At that time there were many clinical cases in both groups. Among the various methods considered for solving the problem was immunization. The entire literature from 1882 was studied.

Pressure was brought from several sources to employ the bacillus which Calmette was recommending so highly. This and dozens of other so-called vaccines which had been in the limelight the previous thirty-eight years were held in abeyance because none of the others had proved satisfactory and Calmette's had not even been tried on humans. In the meantime we employed fundamental measures which consisted of dealing with tuberculosis as a contagious disease.

In 1926, the opportunity came for a personal interview with Dr. Calmette. By this time the first experiments on infants had been reported. His enthusiasm was running high which his data did not seem to support.

For example, Weill-Halle and Turpin had given BCG to 120 infants from July, 1921, to June, 1922. At the beginning of 1926, only eighty of these children could be located. A second group of 356 infants received BCG between June, 1924, and May, 1926, but only 145 had remained under supervision of the experimentors. Moreover these studies were not well controlled and infants died who had received BCG. This did not seem good enough and also appeared to carry some hazard for our students. Therefore, it was still held in abeyance despite the increased pressure that was brought upon us for its use.

After Petroff, Branch and Steenken, Saranac Lake, dissociated BCG into two "R" and "S" colonies with the "S" colony invariably producing progressive tuberculosis in guinea pigs and occasionally in rabbits, and a number of other workers produced disease in animals with it, BCG fell into disrepute in most of the world. However, cultures were kept growing in a few places and administered to small groups of individuals by such persons as Heimbeck of Oslo, Kareszturi and Park of New York, Holm of Copenhagen and later, Rosenthal of Chicago and Aronson of Philadelphia.

BCG received another severe blow when the United States Bureau of Animal Industry conducted intensive and thoroughly controlled studies on cattle. About the same time W. P. Larson, Chief of the Department of Bacteriology, University of Minnesota, and W. A. Evans, Chicago, conducted a large experiment with BCG on cattle. They, and the United States Bureau of Animal Industry, not only found BCG of no avail, but also lamented that it destroyed the value of the tuberculin test which was regarded as the most valuable weapon in eradicating tuberculosis.

A little later, Watson of Ottawa reported that from an extensive study he observed that when animals which had received BCG and controls were subjected to the same exposure, as much tuberculosis developed in one group as the other. Thus his results were the same as those in the United States.

A considerable number of published reports, in which sweeping statements are made concerning efficacy of BCG, have been written by bacteriologists who are participating in production of BCG and dispensing it. Cursory reading of articles could lead one to believe that the studies had been well controlled, diagnoses bacteriologically confirmed and post-mortem findings well documented. On close analysis, however, one finds satisfactory compliance with none of these factors. To date, there has been no well controlled study among humans and it seems doubtful if such a study could be conducted anyplace in the world with people living in their homes. The Oxford University investigators of vole tubercle bacillus vaccine said, "To plan a controlled experiment in tuberculosis vaccination in man is a matter of so much difficulty as to be virtually impossible." Medlar said, "The difficulties of assessing the value of vaccination of BCG in experimental animals under controlled conditions, emphasizes the greater difficulty encountered in the experiments in man because of the existence of uncontrolled and uncontrollable conditions."

In the BCG experiments in people, for the most part, diagnoses have been made from x-ray shadows alone. Inasmuch as shadows are never pathognomonic for any disease, it is not likely that any considerable degree of accuracy in diagnosis obtained in such studies. Moreover, there has been a tendency to consider a shadow-casting lesion in the person who has received BCG, as representing a non-tuberculous condition, whereas a similar lesion in a person who has not been vaccinated is regarded as a tuberculous one. For example, Dalstrom and Difs who reviewed the records of men conscripted for military service in Sweden between 1941 and 1944 compared 36,235 who, on conscription were non-reactors to tuberculin and were given BCG on a voluntary basis and 25,230 who did not react to tuberculin and who rejected BCG. They said, "Generally speaking the diagnosis was somewhat easier to establish in the (originally) tuberculin negative than in the vaccinated group, being helped by the conversion from the non-reactive to the reactive state." Apparently this means that a lesion in a person becoming a tuberculin reactor without BCG was presumed tuberculous unless it was proved otherwise, but in a person who had received BCG it was presumed to be non-tuberculous unless it was proved to the contrary. In most cases proof was lacking. Obviously, accurate diagnoses are not possible without bacteriological determinations.

Among those in BCG experiments who have died in both the vaccinated and the non-vaccinated groups, there has been such a sparsity of dependable necropsies as to significantly invalidate reports. Inasmuch as other diseases closely simulate tuberculosis in gross and microscopical appearance, differentiation is possible only by finding the etiological agent. Relatively few bacteriological determinations have been reported at post-mortem in BCG experiments.

Opponents of BCG are often asked if they know of a single person who has fallen ill or who has died from BCG, whereupon the proponents are asked whether they know that BCG has not been responsible for many persons who have fallen ill and a considerable number who have died subsequent to its administration.

Disasters have occurred recently which were promptly attributed to growing BCG on other than glycerol-bile-potato medium. It is stated that if this medium is used exclusively the bacillus remains at a fixed virulence. Even if this were an established fact, ". . . we must bear in mind that the human body is not to be compared to glycerol-bile-potato as a medium for its continued viability and development. No one can predict what may happen in the course of time if BCG is implanted in the human body and if accidentally carried from human being to human being, or what transformations may occur after such a train of events." (Petroff et al 1929)

Erroneous conclusions have been drawn by comparing unlike groups and conditions. For example, in the first experiment on infants by Weill-Halle and Turpin, those who received BCG were carefully guarded against contacts with contagious cases of tuberculosis whereas the controls were left in their homes with adults who had contagious disease. BCG was given credit for the fact that less tuberculosis developed among the vaccinated infants even though Hess of Chicago, Bernard, Debres and Grancher of France had previously conducted almost identical experiments, except that BCG was not administered to the group protected from exposure and reported as good results as Weill-Halle and Turpin did when BCG was added.

Concerning the BCG experiment on Indians of North America, Aronson says, "Principally the information for the analysis consists of annual observations on the tuberculin reaction and annual x-ray films of the chest with only limited clinical and laboratory data." There has been such an inadequate supply of nurses and physicians in the Indian Bureau that it would be physically impossible for the few professional workers to carry out well controlled experiments, since it is admittedly impossible to carry out such experiments in well organized societies of people. The difficulty is multiplied manyfold when working with Indians, even if personnel were available. Moreover, better results have been reported in tuberculosis control among Indians without BCG than in areas where it has been employed.

Such unlike conditions as allergic manifestations, including erythema nodosum, small primary parenchemal infiltrates, enlarged hilum structures, et cetera, which have occurred in those who have not received BCG, have been considered cases of clinical tuberculosis and compared with the number of cases of actual clinical disease that developed among persons previously sensitized to tuberculin. The two conditions are not comparable in any respect.

One is often asked why the World Health Organization decided to adopt a world-wide BCG campaign and why it went about soliciting governments to participate. This was largely the result of observations which had been made in Denmark. There in 1927, it was decided to administer BCG, especially to children living in areas of considerable exposure and to students of nursing and medicine. Over the next few years it was observed that the mortality rate was decreasing. This resulted in so much enthusiasm that it was decided (1940-1942) that practically all school children between the ages of ten and fourteen years would ultimately receive BCG. Following this there was a continued decrease in mortality but not as great as previously. About 1947 and thereafter, a sharper decrease in mortality occurred and at first it was presumed that this was due to BCG. Therefore, the Danish Red Cross, having been convinced that BCG is extremely efficacious, along with other organizations entered into an extensive BCG campaign which became world-wide in scope.

On analysis, the evidence of effectiveness of BCG in Denmark is nil. Its proponents had apparently neglected to observe that the mortality rate in Denmark had been decreasing sharply for a long time prior to the first administrations of BCG in 1927, and that the rate of decrease was not changed following 1927 nor was it changed in a favorable manner following the extensive administration in the early 1940's. Nor was it known when the world-wide campaign was started that the sharp decrease beginning about 1947 and continuing thereafter in Denmark was due largely to chemotherapy, particularly streptomycin. Moreover, this sharp decrease beginning about 1947, occurred practically everyplace in the world where new chemotherapy was employed, regardless of whether BCG was also in use.

Another oversight on the part of those who sparked the world-wide BCG campaign was that in areas comparable to Denmark where BCG was not in use a decline in mortality rate was as great and in some places greater than in Denmark. For example, the states of Wisconsin and Minnesota each with a population approximately the same as that of Denmark, but where BCG has never been used, except in minor experiments, has long decreased at approximately the same rate as that of Denmark, both before and since the Denmark BCG era. There is not the slightest evidence that BCG affected in any way the mortality curve in Denmark or that its absence changed the curves in an unfavorable way in Wisconsin and Minnesota. Therefore, the world-wide BCG campaign was started, and has continued with no evidence whatsoever that it was of value in Denmark or anyplace in the world.

The credit given to BCG belonged to other factors and it was these which the World Health Organization would have done well to offer the world more abundantly, rather than BCG. Factors common to Denmark, other Scandinavian countries, Wisconsin, Minnesota, the greater part of the United States and other parts of the world where good tuberculosis control work has been demonstrated have been, and still are, sanatoriums, good diagnostic and therapeutic work and control of the disease in animals. Everyplace where these three factors have been operating to a reasonable degree, there has been marked decrease in tuberculosis among humans.

As we pursued our tuberculosis control program among children and University students, allergy to tuberculoprotein became an ill omen. It was observed that only those who were allergic developed clinical tuberculosis. Experimental work had revealed that allergy is pre-requisite for the development of serious and progressive tuberculosis. In 1952 Krause said, "Accordingly, the allergic reaction is seen to be responsible for all of the acute manifestations of tuberculosis and, when so considered, it must be viewed as a potent contributor to pathogenesis. Human beings pass as perfectly well as long as they hold their tuberculous infections asymptomatic, inactive. But they are allergic and any discharge of sufficient focal material to a new place will render them immediately ill because of the allergic reaction that ensues promptly." Therefore, one of our chief endeavors was to prevent allergy to tuberculoprotein by avoiding exposure to tubercle bacilli.

To allergize human tissues willfully by introducing living tubercle bacilli, BCG or others into the individual's body was entirely contrary to our method of attack. We are trying to avoid allergy rather than produce it. When it did appear through natural infection we were using allergy to locate the source of the exposure. In fact it had long been our best epidemiological agent. Proponents of BCG, vole tubercle bacillus and the like, maintain that the creation of allergy by the introduction of these living organisms indicates successful vaccination despite the fact that evidence is entirely lacking that allergy is anything more than a dangerous factor in the evolution of tuberculosis in the human body. If we had allergized all of our children and students on first seeing them we would have lost our best epidemiological agent, all possibility of early diagnosis in the strict sense of the word and in all likelihood the tuberculosis situation among them would have remained unchanged or even become worse.

Our observations on children and students, together with those of others, convinced us that the conclusions drawn by the French scientists of the 1880's from their attempts to vaccinate against tuberculosis were correct, namely, that no dependable immunity develops in the human body as the result of presence of tubercle bacilli of any degree of virulence. Tuberculosis is notoriously a relapsing disease and we could find no evidence of immunity being produced as the result of a mild or severe attack.

Scientists and physicians were long deceived by interpreting the presence of calcification in the chest as a manifestation of immunity. It was later learned that deposition of calcium is not a specific process for any disease and that in some areas of the world more calcium deposits are due to other causes than tuberculosis.

Presence of fibrosis in and about the tuberculous lesion also deceived. It is now known that this only indicates response to an irritant and not evidence of immunity. Other irritants than tubercle bacilli result in more fibrosis, notably silicon dioxide.

Although in the 1920's there was no evidence of efficacy of BCG or any other of the dozens of agents that had been employed, nor have we since seen such evidence, the danger element alone never would have permitted us to introduce live tubercle bacilli of any degree of virulence or total absence of virulence into the bodies of humans. Recent developments have justified this fear. Although no one doubts that Calmette and Guérin had reduced the virulence of BCG to such low levels that it would not produce tubercles in animal tissues and they declared it a virus fixé they had no assurance that it would actually remain of fixed virulence.

As time passed it was observed that when BCG was introduced subcutaneously sometimes tuberculous ulcers and abscesses formed at the sites of administration and the regional lymph nodes became markedly involved as manifested by enlargement, breaking down and discharge of pus for many months. This occurrence was frequent and annoying and should have been frightening. Rosenthal of Chicago, and others sought to solve this problem by using the puncture method of administration similar to that now used in smallpox vaccination or by applying it to a superficial scarification of the skin. This decreased somewhat the incidence of clinical lesions developing at the site of administration and in the regional lymph nodes but some still occurred.

On several occasions we have called attention to numerous reports of clinical tuberculosis which develop in the skin and subcutaneous tissues as well as regional lymph nodes. Only a few of the more recent ones are now presented. Olga Rojas of Equador (1953), observed results of BCG vaccination in 14,500 infants and children up to the age of fifteen years. Lymphadenitis occurred in twenty-two. The lesions varied from small simple infarction to those that were acute and progressed to caseation. This complication occurred in two or three months after administration of BCG. In the majority of cases there was spontaneous regression. In others antibiotics had to be administered and in still others surgical treatment was necessary. J. I. de Bruyne et al (1952), reported on 809 infants who received BCG. Of the 592 who were later examined 165 had moderate swelling of the lymph nodes of which seventy-eight broke down. They later reduced the size of the dose of BCG and found such complications less frequent. In five infants and in one twenty-one-year-old medical student skin lesions developed in the area of intracutaneous vaccination which resembled lupus vulgaris lesions. Exanthems resembling tuberculids were also seen in some infants. They state that since lymphadenitis, tuberculids and even mastoiditis have also been observed after administration of BCG they feel that the frequency of complications is the result of the size of the dose rather than the mode of application.

It is curious that apparently no one sensed that something had happened to the cultures of BCG that had been maintained in different parts of the world because the original Bacillus Calmette Guérin would not produce tubercles in living tissues. In fact, Calmette had scorned the idea of introducing as a vaccine any live tubercle bacillus that would produce tubercles. Therefore the so-called BCG that was resulting in the clinical lesions in the arms and axillary lymph nodes in so-called vaccinated individuals did not meet Calmette's requirements.

Particularly after these clinical lesions were being produced by the so-called BCG cultures in use, it is difficult to understand why no one investigated these cultures in an attempt to determine what had occurred that was responsible for the development of these clinical lesions. This is especially remarkable in view of the fact that Petroff, Branch and Steenken as well as others had produced tuberculosis with BCG in animals in 1929 and earlier. Apparently the few bacteriologists in the world who were maintaining cultures believed that Calmette could not have been wrong when he declared BCG a virus fixé.

The necessity for investigating cultures of BCG now extant was again emphasized when Dubos of Rockefeller Institute for Medical Research, produced progressive and sometimes fatal pulmonary tuberculosis in mice on deficient diets and Vorwald et al produced progressive and killing disease with BCG in silicotic guinea pigs. Hauduroy and Rosset of France, and also Puntigam of Vienna, have produced fatal tuberculosis with BCG in normal golden hamsters, and Hauduroy has found that it results in disease in ground squirrels.

It is also disturbing to know that the statement so frequently made to the effect that BCG has only a fleeting existence after introduction may now be seriously questioned. Vorwald et al found so-called BCG alive and virulent in silicotic guinea pigs eighteen months after administration. At this moment there is no proof that this organism may not survive in human tissues indefinitely just as bacilli acquired through natural infection do.

The teaching that BCG remains at the site of the initial administration and in the regional lymph nodes has not been supported by recent animal experiments. After intracutaneous administration Strom found them three minutes later in the nearest lymph node 18 centimeters distant and half had arrived at this point in 10 minutes. Gernex-Rieux found organisms in the spleen on the eleventh day after they had been administered by the skin scarification method. It is difficult to understand why it should have been expected that these tubercle bacilli would remain in the area of administration since it is well known that this does not happen with other tubercle bacilli or even inanimate material such as lampblack.

Although Calmette declared BCG a virus fixé in 1924, it was not until recently that serious effort was made to determine what changes had taken place in so-called BCG cultures which have been maintained in different parts of the world. Dubos and co-workers assembled cultures from various laboratories and found no two alike even in their gross cultural appearances. Apparently different changes had occurred in the various cultures. On further investigation Dubos found that no culture contains a single bacterial form, such as Calmette and Guérin designated BCG, but each one contains multiple bacterial forms. Presumably these originated through mutation. Moreover, these bacterial forms varied markedly in their ability to multiply *in vivo*. It is possible these forms with such invasiveness in animal tissues which have been producing progressive and killing disease in animals and causing clinical lesions at sites of inoculation and regional lymph nodes in people, may result in much destruction over the next few decades in the bodies of persons who have been willfully infected with so-called BCG.

Animal experiments continue to be performed, but no evidence has accrued to change the conclusions of the French scientists of the 1880's. For example, Helmert, 1952, found that guinea pigs which had received BCG and six weeks later were inoculated with virulent tubercle bacilli, lived a few days longer than controls, but all died from tuberculosis. Other animals received dead tubercle bacilli instead of BCG and the prolongation of life after they were given virulent organisms was essentially the same as those given BCG.

Although sweeping claims regarding efficacy of BCG in humans continue to appear in the literature, none has been supported by adequately controlled experiments. Bogen, 1953, said, "Despite the great enthusiasm expressed by proponents of BCG vaccination, both with the 'concurrent' oral regemen of De Assis and with the intradermal or percutaneous method sponsored by the UNICEF, these measures which have been applied to more than a million infants and other young negative tuberculin reactors in South America, have not yet been given to enough of the older adults, who contribute the greater part of the tuberculosis mortality, to be credited with playing any part in the striking reduction in the tuberculosis deaths among adults."

Palmer reported on the four experiments with BCG by the Division of Chronic Disease and Tuberculosis of the United States Public Health Service. During the first few years of the experiment a very large portion of the clinical tuberculosis which appeared was in the group who reacted to tuberculin when the experiment began and therefore were not vaccinated. Second, so little tuberculosis occurred among those eligible for vaccination, whether or not they were vaccinated, that the number of cases and deaths is still too small to provide any definite evidence. "It is possible that in the Indian and Puerto Rican mortality data there may be early signs that BCG in certain circumstances could be useful in tuberculosis control. On the other hand it is also true that in none of the morbidity comparisons is there any evidence of the beneficial effect of BCG." Third, these studies to date do not indicate that tuberculosis in the United States would be more effectively controlled by adding mass vaccination programs.

D. Hoogendoorn, 1953, wrote on what can be expected from vaccination with BCG in the Netherlands. He pointed out that in 1951 a total of 130 children less than fourteen years of age of whom seventy-nine were less than five years died from tuberculosis. He stated that if vaccination with BCG should be 100 per cent effective only a comparatively small number of children's lives should be saved and to accomplish this 220,000 would have to be vaccinated annually. Furthermore, he said that it is estimated that about one of 3,000 vaccinated would die from tuberculosis. Thus seventy-four of the vaccinated would die each year.

Obviously, no one now knows the actual composition of any of the cultures of tubercle bacilli being dispensed as BCG. We do know that marked changes have occurred in the various cultures since Calmette produced them. We have no assurance that even more invasive forms will not appear in the future nor do we know that this will not occur among those which have already been introduced into human bodies.

References

1. Bogen, E.: Tuberculosis in South America. Am. Rev. Tuberc., 67:676, 1953.
2. De Bruyne, J. I., van Creveld, S., Prakken, J. R., and Stoppelman, M. R. H.: Complications of intracutaneous BCG vaccination of newborn infants. Nederl. Tiyschr. v. geneesk., 96:2675, 1952.
3. Helmert, E.: Experimental studies on the effectiveness of inoculation against tuberculosis. Abl. Bakt., 1. Abt. Orig., 158:3-5, 193-199, 1952.
4. Hoogendoom, D.: What can we expect from BCG vaccination? Maandschr. v. kindergeneesk., 20:269, 1952.
5. Palmer, C. E.: Present status of BCG studies. Am. Rev. Tuberc., 68:462, 1953.
6. Petroff, S. A.: A new analysis of the value and safety of protective immunization with BCG. Am. Rev. Tuberc., 20:275, 1929.
7. Petroff, S. A.; Branch, A., and Steenken, W., Jr.: A study of bacillus Calmette Guerin (BCG). Am. Rev. Tuberc., 20:275, 1929.

The meeting was adjourned.

Robert E. Priest, M.D., *Secretary*

AMERICAN FOUNDATION FOR ALLERGIC DISEASES

Formation of a Scientific and Educational Council of nationally known scientists and educators to promote more research and wider professional education in the field of the allergic diseases has been announced by the American Foundation for Allergic Diseases from national headquarters, 525 Lexington Avenue, New York City.

The members are as follows: Robert A. Cooke, M.D., director The Institute of Allergy, Roosevelt Hospital, New York; Frank J. Dixon, M.D., professor of Pathology, University of Pittsburgh Medical School, Pittsburgh, Pa.; Alfred Gilman, Ph.D., professor of Pharmacology, College of Physicians and Surgeons, Columbia University, New York; Elvin A. Kabat, Ph.D., professor of Microbiology, College of Physicians and Surgeons, Columbia University, New York; Richard A. Kern, M.D., professor of Medicine, Temple University Medical School, Philadelphia, Pa.; Giles A. Koelsche, M.D., assistant professor of Medicine, Mayo Foundation Graduate School, University of Minnesota, Rochester, Minn.; John H. Mitchell, M.D., professor of Clinical Medicine, Ohio State University, Columbus, Ohio; Henry Stevens, Ph.D., head of Section of Allergens, Agricultural Research Service, U.S. Department of Agriculture, Washington, D. C.; Cyrus C. Sturgis, M.D., director Simpson Memorial Institute for Medical Research, professor of Medicine, University of Michigan, Ann Arbor, Mich.; Marion B. Sulzberger, M.D., chairman Department of Dermatology and Syphilology, Post-Graduate Medical School, New York University-Bellevue Medical Center, New York.

The American Foundation for Allergic Diseases was established a year ago under the sponsorship of the American Academy of Allergy and the American College of Allergists as a non-profit organization of physicians and laymen to promote allergy research, improve professional education, increase treatment facilities for allergy sufferers, and spread public understanding of the allergic diseases so as to bring about early diagnosis and treatment.

Minneapolis Surgical Society

Meeting of February 3, 1954

The President, Robert Tenner, M.D., in the Chair

POLYPS OF THE COLON AND RECTUM

RUPERT B. TURNBULL, JR., M.D.
Cleveland, Ohio

POLYPS of the large intestine and rectum have been the subject of intense and varied studies during the past ten years. With sigmoidoscopy performed more frequently as a routine procedure, more polypoid lesions are discovered. This is of increasing significance in cancer control since the adenomatous polyp of the colon and rectum is the seat of cancer, or closely associated with it. One-third of our rectal cancers have had so-called "satellite polyps" and it is not unusual to see a polyp protruding from the colostomy following abdominoperineal resection of the rectum. In addition, rectal polyps are frequently found in patients with a cancer in some other parts of the colon. We would agree with other workers that a polyp in the rectum is a danger signal, and possibly a "sentinel lesion" for a cancer above.

The interpretation of polyp biopsy specimens is not uniform among pathologists, and rather critical problems arise as to the best method of treatment, when a diagnosis of cancer arising in polyp is made or inferred. When submitted to the pathologist, the innocent-appearing polyp may be reported as any one of the following; "adenocarcinoma Grade I in adenomatous polyp", adenoma malignum", "severe dysplasia, no evidence of invasion.", "adenomatous polyp with atypism", "adenocarcinoma", "adenocarcinoma *in situ* in papillomatous polyp", or "microscopic focus of adenocarcinoma arising in polyp". Is radical surgery indicated when this polyp has been removed from the rectum? Is simple fulguration adequate? Should a segment of colon be removed at a second operation (following transcolonic polypectomy) on the basis of one of these reports? There is no question that radical removal of the rectum is being carried out for rectal lesions of doubtful malignancy, as well as for frank cancer.

Our main concern is the treatment of polyps of the rectum in which cancer is said to be present. It has been our belief that such polyps can be successfully treated by local removal, rather than by radical surgery, providing the carcinoma is not invasive in respect to the muscularis mucosa, and providing the biopsy specimen is representative of the lesion.

Any soft or pedunculated polyp must be viewed as potentially benign rather than malignant. Ulcerating, hard tumors are practically always malignant and several biopsies from the firm, rolled-over border are sufficient. Soft lesions, however, must be critically analyzed before definitive treatment is begun. The whole lesion should be pulled away from the mucosa with grasping forceps, and all of the tissue sectioned carefully. In this manner, the patient would receive full benefit of teamwork between the surgeon and the pathologist. Unfortunately, such consideration is not always given. The reasons for this are that all too often only a small part of the lesion is removed for section and may not be representative. Moreover, there is a great variation between pathologists and their interpretations of cancer in polyps. Dysplastic epithelium may be labeled as adenocarcinoma, and the surgeon may use this nomenclature as a basis for advising and performing radical surgery. It is probably more important to save a normal rectum, than it is to remove one for a well-established cancer. In any event, we still see a number of such patients in consultation with their own physicians, who are not satisfied with the pathologic diagnosis or advice of the surgeon. In most of the cases, repeat proctoscopy reveals no evidence of a lesion (previously coagulated or excised), and the sections are those of a polyp with dysplastic epithelium. Others bring slides with obvious adenocarcinoma or adenocarcinoma in polyp. In those with no residual lesion, no operation is indicated regardless of the biopsy finding of cancer or cancer in polyp. We cannot bring ourselves to remove a rectum where no lesion is visible. Where polyp still remained, following biopsy elsewhere, it was totally removed for study before making a final decision regarding treatment.

Pathologic Considerations

The decision to carry out radical or conservative surgery depends on an understanding of the histologic characteristics of polyps of the colon and rectum. These epithelial tumors are true neoplasms arising from the glandular epithelium. They may be on pedicles, or they may be on a wide base or growing diffusely over the surface of the rectum. Some form of adenocarcinoma is found in about ten per cent of the patients. The epithelium of the polyp is separated from the submucosa of the colon by the muscularis mucosa, and since the submucosa contains the bulk of blood vessels and lymphatics, it would appear that the muscularis mucosa is a true dividing line between superficial and invasive carcinoma arising in polyp. In 1952, Dr. Edwin Fisher[1] of the Department of Pathology made a detailed histologic study of 350 rectal polyps that were removed and treated by coagulation and implantation of radon seeds. Of the thirty-five (10 per cent) classified as containing adenocarcinoma arising in polyp, eleven were found to be invasive in respect to the muscularis mucosa, while twenty were found to be superficial. Since our two previous reports[1,4] thirty-one of these thirty-five were followed and examined, leaving four unaccounted for. This group of patients was followed for a period of two to ten years. In each instance, the operator thought that the lesion was benign and that local treatment would be sufficient. This demonstrates the fallacy of attempting to determine the state of malignancy of a given tumor by observation or palpation. It is significant that none of these patients have died of recurrence or metastasis from the original tumor. Two have died from undetected cancers in the colon above the lesion, while two others have had resections of the colon for other primary lesions located above the rectum. The suggestion that the rectal polyp may be a "sentinel polyp" for lesions above is well demonstrated in this series. In all soft, non-ulcerated polyps of the rectum, we are continuing to consider them benign until proven otherwise. The lesion is either completely excised or if not readily accessible, it is "plucked" with biopsy forceps and submitted for multiple section.

We are in complete agreement with Judd's[2] statement that "a colon in which polyps are prone to form serves warning that it is also a colon in which carcinoma is prone to form". This statement is even more significant if one will consider the visible rectum and lower sigmoid colon to be the commonest site of polyps, and conduct a proper investigation to find the lesion that may be present above.

A plea is made for careful consideration of the histologic picture of the benign-appearing rectal lesion before decision is made for treatment. Whether or not invasive cancer is present is determined only by adequate biopsy rather than solely by the operator's opinion. One must be quite familiar with the pathologists' nomenclature in relation to whether or not it actually represents true cancer arising in polyp. An individualized plan of therapy can, therefore, be established by excision biopsy and surgeon-pathologist teamwork. Our own experience would indicate that rectal polyp with noninvasive adenocarcinoma can be treated conservatively, while invasive carcinoma may be considered to be suitable for radical surgery.

Although our series is relatively small, it is of value when included with those of others.[3]

References

1. Fisher, E. F. and Turnbull, R. B., Jr.: Malignant polyps of rectum and sigmoid, therapy based on pathologic considerations. Surg., Gynec. & Obstet., 94:619-625, (May) 1952.
2. Judd, E. S. and Carlisle, J. C.: Polyps of the colon. A.M.A. Arch. Surg., 67:3, (Sept.) 1953.
3. McLanahan, S., Grove, G. D. and Kiefer, R. F.: Conservative management for certain rectal adenomas showing malignant change. J.A.M.A. 141:822 (Nov.) 1944.
4. Turnbull, Rupert B., Jr. and Fisher, Edwin R.: Carcinoma in rectal polyp; follow-up observations on 27 patients. Postgraduate Medicine, 12:303-307, (Oct.) 1952.

2020 E. 93rd Street

The meeting adjourned.

N. C. Plimpton, M. D., *Secretary.*

HIGH BLOOD PRESSURE

High blood pressure appears to be much less dangerous among women than among men, according to a pilot study of her company's employes made by Miss Annie Mary Lyle, fellow of the Society of Actuaries and underwriting research analyst of the Prudential Insurance Company. Results of the study were presented to the Western Spring Meeting of the Society at the Edgewater Beach Hotel in June.

The mortality experience among 1,227 employes with high blood pressures ranging from slight to very marked was carried through a period ranging from six months to twenty years with periodic examinations. Statistics were kept for the group as a whole and for men and women separately.

The women, comprising 40 per cent of the total number examined, showed a death rate much lower than that for men and also much lower than that which had been anticipated for the women on the basis of normal underwriting procedure.

"The group available for this study is much too small for the mortality ratios to be relied on for rating purposes and the female mortality is incredibly low for reasons that are not apparent," Miss Lyle said. "The difference between the sexes is so great, however, as to indicate that in future investigations, males and females should be studied separately. If a substantial difference is confirmed by an experience sufficiently large to be dependable, more lenient treatment of females with high blood pressure would be in order."

The study also indicated that electrocardiograms are of considerable value in judging high blood pressure cases, but that the x-ray does not appear to contribute anything.

"One is forced to the conclusion that women withstand the stresses of high blood pressure better than men do," Miss Lyle said, commenting on the trend over the twenty years. Although the women in the group studied had more hypertension of moderate and marked degrees than did the men, on entering the experience, they showed a much lower death rate than did the men.

✦ Reports and Announcements ✦

MEDICAL MEETINGS

National

American Heart Association, Washington, D. C., September 12-17, 1954.

Annual Assembly in Otolaryngology, University of Illinois, Chicago, Illinois, September 6-11, 1954.

Annual Convention of the National Society for Crippled Children and Adults, Hotel Statler, Boston, Massachusetts, November 3-5, 1954.

Neurosurgical Society of America, Grand Hotel, Mackinac Island, Michigan, July 21-24.

International

Conference of International Union Against Tuberculosis, Madrid, Spain, September 26-October 2, 1954.

Congress of International Association for the Prevention of Blindness, New York, New York, September 12-17, 1954.

International Anesthesia Research Society, Los Angeles, California, October 10-14, 1954.

International Congress of Clinical Pathology, Washington, D. C., September 6-10, 1954.

International Congress of Diseases of the Chest, Barcelona, Spain, October 4-8, 1954.

International Congress of Gynecology and Obstetrics, Geneva, Switzerland, July 26-31, 1954.

International Congress of Group Psychotherapy, Toronto, Ontario, Canada, August 12-14, 1954.

International Congress of Mental Health, University of Toronto, Toronto, Ontario, Canada, August 14-21, 1954.

International Poliomyelitis Congress, University of Rome, Orthopedic Clinic, Rome, Italy, September 6-10, 1954.

International Congress of Hematology, Paris, September 6-11, 1954.

International Society of Blood Transfusion, Paris, France, September 12-19, 1954.

International Congress of Industrial Medicine, Naples, Italy, September 13-19, 1954.

International Congress of Internal Medicine, September 15-18, 1954.

International Congress of Ophthalmology, University of Montreal and McGill University, Montreal, Canada, September 9-11, 1954.

International Congress of Orthopedic Surgery and Traumatology, Berne, Switzerland, August 30-September 3, 1954.

International Society of Geographical Pathology, Washington, D. C., September 6-10, 1954.

Pan American Congress of Gastroenterology, Sao Paulo, Brazil, July 19-24, 1954.

World Congress of Cardiology, Washington, D. C., September 12-18, 1954.

World Federation of Occupational Therapists, Edinburgh, Scotland, August 17, 1954.

World Medical Association, Rome, Italy, September 26-October 2, 1954.

MINNESOTA ACADEMY OF OCCUPATIONAL MEDICINE AND SURGERY

The Minnesota Academy of Occupational Medicine and Sugery, which now has fifty-six members, held its first annual meeting on May 26, 1954, at the Minneapolis Athletic Club.

At a business session after the 7:00 o'clock dinner, Dr. John Shronts, Medical Director, General Mills, Inc., Minneapolis, was named president of the organization to succeed retiring president, Dr. Wilford E. Park, Industrial Physician, Minneapolis Health Department. The other 1954-55 officers will be: Vice President—Leslie W. Foker, M.D., Minneapolis; Secretary—James Fox, M.D., Minneapolis; Treasurer—Tracy Barber, M.D., Austin; Recorder—Gordon MacRae, M.D., Duluth; Executive Committee member for two years is H. E. Drill, M.D., Hopkins. Continuing in the second year of his two-year term as Executive Committee member is Edwin G. Benjamin, M.D., Minneapolis.

A scientific paper, "General Problems of the Heart and Pre-employment Examinations," was presented by Dr. A. C. Kerkhof, cardiologist, Minneapolis.

CONTINUATION COURSE

The University of Minnesota announces a continuation course in *Psychiatric Principles in General Practice* from September 8 to 10, 1954. The course will be held at Douglas Lodge, located deep in the woods of northern Minnesota on the shores of beautiful Lake Itasca. The program will concern those psychiatric and psychosomatic problems which are most frequently seen in general practice, and practical management will be emphasized throughout. The guest faculty will include Dr. Bernard C. Glueck, Jr., Ossining, New York, and the course will be presented under the direction of Dr. Donald W. Hastings, Professor and Head, Department of Psychiatry and Neurology, and Director, Division of Psychiatry. The remainder of the faculty will be drawn from the Univesity of Minnesota Medical School and the Mayo Foundation.

Sessions will end each day in mid-afternoon to permit time for fishing, boating, and other recreational activities. The fee will be $130.00 which will include registration, tuition, meals and lodging, instructional material, transportation *from Minneapolis* to Douglas Lodge and return, and fishing license. Further information may be obtained from the Director of Continuation Medical Education, University of Minnesota Hospitals, Minneapolis 14, Minnesota.

Letters to the Editor

TEACHING THE NURSES AID

Dear Dr. Wells:

The Minnesota Legislature of the 1953 session passed an enabling act providing for the establishment of a program of nursing education at the Mankato State Teachers College, Mankato, Minnesota. This legislation was requested and supported by a group of physicians and citizens from southern and southwestern Minnesota among which two of the active leaders were Dr. L. L. Sogge of Windom, and Dr. H. J. Nilson of North Mankato. The actual enabling act passed by the 1953 legislature reads in part—

> "An act appropriating money to the Mankato State Teachers College . . . to be used by the college to teach certain subjects in the preliminary training of students who take courses in nurses training so that they may qualify to become registered nurses as defined by Minnesota Statutes . . ."

Because the college officials did not receive official word that the governor had signed the bill until about the first of May, no advertising or publicity went out to high school graduates of surrounding areas concerning the new nurses training program to be offered. Even with the lack of advance notice, however, twenty-six girls enrolled for the course in September, 1953, when the fall term of the Mankato State Teachers College started. There have been a few losses in students during the year due to marriage, inability to maintain satisfactory academic work, or to transfer to the teaching program. For this coming year, when it was possible to notify area high school graduates of the program, more than 220 prospects listed their names with the college. This indicates that there is no apparent shortage of prospects for nurses training in the area served by the college.

Under present plans, students may enter the course once each year, in the Fall. During the first year, classes will be in general education and pre-professional courses, such as English, sociology, psychology, chemistry, biology, anatomy, physiology, microbiology, nutrition, and nursing arts. Should the student desire to change her vocational objective after the first year, she may do so without loss of credit.

The remainder of the four-year program includes approximately one additional year of broad general education and two years of courses in nursing: classroom and clinical instruction, and supervised practice in the various types of nursing. On the completion of this curriculum, the student receives the degree of Bachelor of Science in nursing. The program is so arranged that the student who has completed the first three years of work should be eligible to write the state examination for registration (R.N.).

The local hospitals at Mankato—St. Joseph's with its new $4,000,000 plant, and the Immanuel with its new 60-bed addition—are co-operating with the college, and will serve as training centers for much of the program. The auxiliaries from both hospitals were active in the support of the program. Other affiliations with hospitals are arranged to provide specialized clinical experience.

The instruction and the entire curriculum are under the direct administration of the College. The student nurse is a regular college student, eligible to participate in the campus activities. She attends many of the classes with students specializing in other Divisions of the College and she receives the advantages of the full resources of the college's program, faculty, and educational facilities. The curriculum is planned to meet the standards of the profession.

Miss Clara May Miller will direct the program. She has a background of teaching and nursing. Miss Miller comes to Mankato from the Ball Memorial Hospital of Muncie, Indiana, where she has been Superintendent of Nurses and Director of the School of Nursing. The nursing school there is affiliated with Ball State Teachers College, an institution quite similar to Mankato State Teachers College in which the new program has been initiated.

C. L. CRAWFORD
President, Mankato Teachers College

Mankato, Minnesota
April 30, 1954

MEDICAL MALPRACTICE INSURANCE

Dear Dr. Wells:

I have read with great interest the editorial, "Medical Insurance and Its Costs," in the May, 1954, MINNESOTA MEDICINE. The series of statements of fact are gleaned from malpractice lawsuits. The medical profession, too, is aware of and has accepted these facts. The insurance companies have lectured our profession concerning these abuses that oftentimes precipitate a malpractice suit. Our profession, even as the insurance companies, has likewise brought forth these facts before our organization concerning these ever-worsening ugly clouds, but again it has not been of much avail. At least the insurance premium rates are not coming down.

Possibly, many of us feel that since we carry this insurance in companies out of the State, even as we do on our automobiles, we need not be perturbed when we get into trouble. We simply drop it into their laps and then let them take over from there. Personally, I have been fortunate in that respect. But it does not follow that one may not encounter difficulties. And, since the ever-increasing premium is a growing problem, and since it is running rife, and the insurance companies are compelled to continue raising the rates, one

(Continued on Page 544)

◆ Of General Interest ◆

Dr. Gilbert S. Campbell, Department of Surgery, University Hospitals, who was recently awarded one of the distinguished Markle Scholarships in the amount of $30,000.00, has now been awarded the John Horsely Memorial Prize by the University of Virginia in the amount of $600.00, for his paper entitled "Effect of pH Changes on Vagal Inhibition of the Heart."

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Dr. Ivan D. Baronofsky, associate professor of surgery at the University hospitals, was guest consultant at the last of three clinical sessions held for the McLeod County Medical Society in Glencoe.

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Dr. R. P. Pearsall, Virginia, was presented with a gold pin and certificate in recognition of his fiftieth year in the practice of medicine, at the State Medical Convention in Duluth.

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Dr. Charles A. Neumeister, Minneapolis, attended the annual meeting of the American Proctologic Society in Los Angeles, California, June 2-5. He presented a joint paper with Dr. Walter A. Fansler entitled "The Treatment of Incontinence and Other Abnormalities Following Infection of the Anal and Rectal Regions."

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Dr. Walter A. Fansler, Minneapolis, was one of three guest speakers at the Schoitz Memorial Hospital Annual clinic, Waterloo, Iowa, May 27. He presented two papers, one on "The Diagnosis and Treatment of Polyps of the Colon and Rectum," and the other on "Common Errors in the Diagnosis and Treatment of Anorectal Diseases," and participated in a general discussion panel composed of the three guest speakers.

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At the meeting of the Society for Clinical Investigation in Atlantic City the following doctors from the Mayo Clinic attended: **Drs. A. Albert, C. M. Blackburn, H. B. Burchell, J. C. Cain, C. F. Code, W. S. Fowler, Jr., E. A. Hines, D. R. Nichols, H. F. Polley, R. M. Salassa, R. G. Sprague, W. E. Wellman** and **E. H. Good.**

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The offices of Doctors **Clarence E. Arlander** and **Carl J. Olson** were moved in May from 2300 Central Avenue, to their new building at 901 Twenty-third Avenue, N.W., Minneapolis.

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Drs. H. A. Andersen, O. T. Clagett, John Kirklin, J. R. McDonald and **H. W. Schmidt,** Mayo Clinic, attended the meeting of the American Association for Thoracic Surgery in Montreal, Quebec.

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Dr. F. R. Kotchever, President of the East Range Clinic of Eveleth and Virginia, is one of the many doctors who were in the group of the American College of Surgeons who were the guests of the Association of Surgeons of Great Britain and Ireland and the Royal College of Surgeons of Edinburgh, Scotland, at sectional meetings. Dr. Kotchever was gone for seven weeks. Mrs. Kotchever and their two daughters accompanied him on the trip.

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Dr. and Mrs. Thomas Lowe, South Saint Paul, spent three weeks in Italy where Dr. Lowe attended the sessions of the International College of Surgeons.

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Dr. R. H. Puumala lectured to the interns of St. Luke's hospital in Cloquet in May. He spoke to them on the office equipment needed for a medical practice in a smaller community.

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Dr. J. E. Verby, Litchfield, attended a two-day refresher course at the University of Minnesota Center for Continuation Study.

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Dr. Walter J. Bo, of the department of anatomy of the University of Minnesota, has been given a $3,135 research grant to study the relationship between vitamin A and estrogen in producing uterine cell changes in rats.

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Dr. Ann Whelan Arnold, Minneapolis, visited her son and attended her daughter's graduation from Cornell University's college of medicine en route to the American College of Surgeons' convention in London.

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Dr. Roy G. Holly, associate professor of the department of Obstetrics and Gynecology at the University of Minnesota, was the guest consultant at the first of two clinical conferences held in Breckenridge on May 6. Dr. Holly presented some case histories on cancer and discussed cervical cancer. He supplemented his discussion with slides and other clinical materials.

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Dr. R. L. Faucett, of the section of psychiatry of the Mayo Clinic, spoke at the international forum on alcoholism and highway safety held May 12 and 13. He discussed the effects of problem drinking within the home. The gathering was called "Operation All-Out" and was an attempt to alert business and industry on alcoholism and prevent employe job loss.

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Dr. J. A. Bargen, Rochester, conducted clinics and gave lectures at the University of Louisville school of medicine in Louisville, Kentucky, in May. He also addressed a meeting of the staff of the Veterans Administration there.

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Dr. J. C. Ivins, Mayo Clinic, participated in a military medical symposium conducted by the army medical service at Fitzsimons army hospital in Denver, Colorado, in May.

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As a member of the medical committee, **Dr. Jan Tillisch,** Mayo Clinic, attended the meeting of the International Air Transport association in Bermuda in May.

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Dr. C. W. Rucker, Mayo Clinic, was a member of the program committee of the American Academy of Ophthalmology and Otolaryngology when it met in New York City the first week in May.

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Dr. Gordon Lee, Glenwood, has been appointed medical advisor by the American Cancer Society for Pope County. On May 18, he explained the motion picture, "Breast Self-Examination" at the American Legion Memorial Building.

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Dr. Hiram E. Essex, of the Mayo Clinic, delivered this year's Joseph Graham Mayo lecture in the State University of Iowa's medical amphitheater at Iowa City, May 10. His speech, one in the current series of college of medicine lectures by outstanding scientists from the United States and abroad, was entitled "Observations on Vascular Spasm Produced by Various Agents."

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Dr. Tague C. Chisholm, Minneapolis, spoke on pediatric surgery at the annual meeting of the Upper Midwest Conference of Nurse Anesthetists.

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Dr. George Logan, Rochester, was guest speaker at the Woman's auxiliary of the Four-County Medical Society meeting, May 5, in the Mayo Foundation House. Dr. Logan presented the pros and cons for the Salk polio vaccinations being given to children throughout the United States.

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Dr. and Mrs. E. C. Wilkins, Rochester, sailed for Europe aboard the Queen Mary. They toured England, Scotland, Sweden, Denmark, The Netherlands, Belgium, France, Switzerland and Italy before returning to Rochester on July 11. Dr. Elkins is secretary of the American Board of Physical Medicine; he attended the meeting of the International Federation of Physical Medicine in Ostend, Belgium.

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Dr. K. A. Storsteen, Duluth, spoke at a meeting of the St. Louis County Medical Society, May 13, at St. Mary's Hospital. His subject was "Surgical Emergencies in Infancy and Childhood."

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C. F. Cervenka, New Prague, gave a talk on Health Management at the Rotary club meeting in May. Srtessing the complexity of present-day living, he pointed out how slowing down in all our daily tasks would relieve the inherent tension in many nervous systems.

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Dr. V. S. Counseller, Mayo Clinic surgeon, addressed a meeting of the Mexican Urologic Association in Mexico City, May 5.

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The American Cancer Society said that scientists, including two from the University of Minnesota, have found human polio viruses multiply fast and completely destroy at least two types of human cancer tissue while feeding on it in test tubes. The work was done jointly by a group at the Johns Hopkins medical institutions in Baltimore headed by Dr. George O. Gey and anoher group at the University of Minnesota headed by **Dr. Jerome T. Syverton,** head of the department of bacteriology and immunology, and **Dr. William F. Scherer,** assistant professor of bacteriology and immunology.

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Resignation of **Dr. Peter Ward** as medical director of the Charles T. Miller Hospital and the Amherst H. Wilder dispensary was announced by the chairman of the board of directors of the hospital and foundation. Dr. Ward has been medical director of Miller hospital and Wilder dispensary since 1930. William N. Wallace, present assistant, has been named new administrator.

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Tobacco and lung cancer was the topic of an address given by **Dr. Harod N. Wright** at the Duluth branch of the University of Minnesota. Dr. Wright is professor of toxicology and pharmacology at the University.

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Three professors will retire in June from the faculty of the Mayo Foundation. They are: **Dr. Stuart W. Harrington,** professor in surgery, **Dr. Frederick A. Willius,** professor of medicine, and **Dr. Byrl R. Kirklin,** professor of radiology.

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Dr. P. J. Pankratz, Mountain Lake, attended the Southwestern Medical meeting in Worthington on May 10.

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Dr. L. W. Johnsrud, Hibbing, was elected president of the Minnesota Surgical Society at its semiannual meeting in Rochester on May 14. Dr. Johnsrud is affiliated with the Mesaba Clinic in Hibbing.

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Dr. Donald W. Hastings, head of the department of psychiatry and neurology at the University of Minnesota, was named chairman of the new medical policy committee of the state department of public welfare. Other members of the policy committee are **Dr. L. R. Critchfield,** Saint Paul pediatritian; **Dr. F. J. Hirschboeck,** Duluth internist; **Dr. A. B. Baker,** professor and director of the university's division of neurology, and **Dr. J. L. Bollman,** head of the section of clinical pathology and biochemistry, Mayo Clinic, Rochester. The committee of the state department of public welfare was created by the 1953 State legislature.

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Dr. Giles A. Koelsche, assistant professor of medicine at the Mayo Foundation, has been appointed a member of the scientific and educational council of the American Foundation for Allergic Diseases, according to an announcement from foundation headquarters in New York City. Composed of ten nationally known research scientists and physicians, the council has the responsibility of directing the medical program of the foundation, which was established a year ago by the American Academy of Allergy and the American College of Allergists as a non-profit organization of physicians and laymen to work in the field of the allergic diseases.

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Dr. Mellvin E. Lenander, St. Peter, was reappointed by Governor Anderson to serve a full seven-year term on the state board of medical examiners.

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Dr. M. W. Hanson of the Carleton College health service attended a conference on health, in New York City, in May.

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Dr. J. Arthur Myers, University of Minnesota medical school, spoke to the 3,000 delegates of the convention of the National Tuberculosis Association. **Dr. Walter J. Marcley,** Minneapolis, tuberculosis consultant to the Minnesota health department, was honored as a founder of the organization. Dr. Marcley established Minnesota's state tuberculosis sanatorium at Walker, and was the first medical director of Glen Lake sanatorium.

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Dr. E. C. Elkins, Mayo Clinic, conducted a polio clinic for area physicians in April at the Mankato Rehabilitation Center. Dr. Elkins is head of the polio ward at St. Mary's hospital in Rochester. He is also a member of the physical medicine staff of the Mayo clinic.

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Dr. B. A. Flesche, Lake City, attended a symposium of army medicine of the 5th Army Reserve Corps in May. Sessions were held at Fitzsimons Army Hospital.

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Dr. F. J. McCaffrey, Minneapolis, gave a talk on "Operative Obstetric Problems" and gave a forceps mannequin demonstration for the Tri-County Medical Group, Camp Release District Medical Society, Chippewa, Lac qui Parle, and Yellow Medicine counties at Montevideo on May 13.

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At the American Psychiatric Association meeting in Saint Louis in May, **Dr. Henry Hutchinson,** Superintendent of the Moose Lake State Hospital, **Dr. Ralph Rossen,** Superintendent of the Hastings State Hospital, and **Dr. Burton P. Grimes,** Superintendent of the St. Peter State Hospital, were elected as Fellows of the American Psychiatric Association. All three of these doctors had been members of the American Psychiatric Association for some years.

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At the American Psychiatric Association meeting in Saint Louis, in May, the committee on certification of mental hospital administrators of the American Psychiatric Association certified **Dr. Henry Hutchinson,** Superintedent of the Moose Lake State Hospital, as a competent mental hospital administrator.

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The monthly staff meeting of St. Joseph's Hospital, Brainerd, was addressed by **Drs. Erling S. Platou** and **A. J. Schroeder,** Minneapolis, who spoke on the subject of "Pediatric and Obstetric Aspects of Hemolytic Disease of the Newborn."

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Dr. William J. Martin, consultant in medicine at the Mayo Clinic, was the speaker at the last of four Spring meetings of the Southwestern Minnesota Medical Society. Dr. Martin discussed "General Principles in the Use of Antibiotics."

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Dr. Ralph G. Ghormley and **Dr. John J. Morton,** members of the Robert Jones club, national organization of orthopedic surgeons, attended the meeting which was held in Washington University school of medicine for the annual clinic program in May.

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Dr. D. S. Childs, Jr., of the Mayo Clinic, attended the first annual meeting of the Society of Nuclear Medicine in Seattle, May 29 and 30. He presented a paper entitled, "Training of Residents in Radiosotope Technics."

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Dr. C. H. Slocumb, Mayo Clinic, attended a meeting in Minneapolis, May 29, regarding formation of a Minnesota chapter of the Arthritis and Rheumatism Foundation.

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Surgical chiefs of thirty-four veterans hospitals in ten states attended a two-day surgical institute June 3-4. The surgeons discussed latest techniques and developments in thoracic, abdominal and vascular surgery.

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Dr. Silas W. Giere, Benson, left May 23 for New York where he sailed for Norway. Dr. Giere will spend the summer months touring Norway, Sweden, Denmark and England.

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Robert P. Jeub, Hennepin County Medical Society member, now a Captain in the Air Force stationed at the Lackland Base, San Antonio, Texas, presented a paper on "Location and Treatment of Head Injuries" at the Air Force World Wide Sports Conference, held at the University of Oklahoma on May 21.

CHANGES IN LOCATION

Dr. Robert Kallsen has joined Dr. C. M. Marshall at the Cuyuna Range Clinic in Crosby. Previously he practiced in La Jolla, California.

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Dr. R. J. Camp has become associated with the Madison Clinic. For the past four years he has been on the staff of the University of Oklahoma School of Medicine.

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Dr. R. M. Lundblad is leaving Clara City to return to the University of Minnesota for further study.

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Dr. Roger Parsons and **Dr. Kenneth Moulton** are opening a medical practice in St. James. Both men graduated from the University of Minnesota last year and have been interning at St. Joseph's Hospital in Saint Paul.

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Dr. Paul Brabec has joined Dr. Herman Just in the general practice of medicine and surgery in Hastings. He previously had been in practice in Bagley.

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Dr. D. W. Selzer, formerly of Manhattan, Kansas, is assisting Dr. F. C. Closuit in Aitkin until the middle of September, when he plans to go to Rochester on a fellowship.

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Dr. William E. Walter is opening an office for the practice of medicine in Wanamingo the first part of August. He is at present completing his internship training at General Hospital in Minneapolis.

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Dr. Charles E. Turbak is leaving Canby to take a postgraduate course in surgery as a resident doctor at Ancker Hospital in Saint Paul.

* * *

Dr. Royden A. Belcher and **Dr. C. J. Swendseen** have joined the staff of the Oliver Clinic in Graceville. Dr. Belcher recently completed his internship at Ancker Hospital in Saint Paul, and Dr. Swendseen was associated in practice in Minneapolis for the past year.

* * *

Dr. Vincent Ryding joined the Surgical and Medical Clinic of Albert Lea as its surgeon. He has been the senior resident in surgery at the Northwestern Hospital in Minneapolis for the past year.

* * *

Dr. Roy G. Holly has taken over a new position as head of the obstetrics department for the University of Omaha. Formerly he was an associate professor of obstetrics and gynecology at the University of Minnesota.

* * *

Dr. Frank A. Rudolph, formerly of Rapid City, South Dakota, has opened his practice of medicine in Sandstone.

BLUE SHIELD-BLUE CROSS NEWS

In the Blue Shield financial and statistical report for the first four months of 1954 which has just been released, a marked increase is shown in the number of claims paid and the total amount paid for claims. In the first four months of 1954, Blue Shield has paid out more than in any other identical period in its history and has returned to the subscribers a greater portion of the cash income from subscribers than before. Though the financial data available for the first four-month period reflect a greater increase in claims payments dollar-wise, only two months of the four-month period reflect the increase in benefits which became effective January 1, 1954, since payments in January and February were, to a great extent, for services rendered to patients during 1953.

Records in the Blue Shield Claims Department show the following numbers of claims processed for payment during each of the first four months of 1954: January, 9,506; February, 9,480; March, 10,990; April, 11,195. These figures show that the marked increase occurred in March and April. This fact is of further significance in that most of the claims paid in January and February were for services rendered prior to January first, whereas those paid in March and April involved services rendered after January first, the date when a large number of benefit increases became effective. The total number of claims paid during the first four months of 1954 was 41,171, whereas during the same period in 1953, 40,376 claims were paid.

The financial report for April shows a comparable increase in the cost of claims for the first four months of 1954. For instance, while in January $366,994 was paid for claims and $375,340 was paid for claims during February, in March claims costs increased to $449,983 and in April claims costs amounted to $449,463. These figures show a total of $1,641,781 paid for claims during the first four months of 1954, or an average of $410,445 a month. This is to be compared with $1,465,119 paid for claims during the first four months of 1953, or an average of $366,279 a month for this period in 1953. The total increased cost of claims for the first four months of 1954, as compared with the same period in 1953, amounted to $176,662 or $44,165 per month.

Translated into the percentage of the cash earned income from subscribers, these figures show 85.1 per cent was spent for claims in the first four months of 1954 as compared with 81.6 per cent during the same period in 1953.

In considering the data presented, two factors are of importance. Customarily the first four months of the year show greater utilization or a larger number of claims than similar periods in the balance of the year. Also, part of the increased cost is to be attributed to the increase of benefits which became effective January 1, 1954.

Hospitalization expense incurred by Blue Cross subscribers during the month of April, 1954, amounted to $1,503,406.98. This expense is 2.9 per cent greater than anticipated. For the first four months of 1954, $5,854,094.92 has been incurred for the care of Blue Cross sub-

(Continued on Page 544)

BOOK REVIEWS

Books listed here become the property of the Ramsey, Hennepin and St. Louis County Medical Libraries when reviewed. Members, however, are urged to write reviews of any or every recent book which may be of interest to physicians.

THE PAINFUL PHANTOM. Psychology, Physiology and Treatment. Lawrence C. Kolb, M.D., Section of Psychiatry, Mayo Clinic, Rochester, Minnesota. 50 pages. Price $1.50, paper cover. Springfield, Illinois: Charles C Thomas, 1954.

GERIATRIC MEDICINE. Medical Care of Later Maturity. Third Edition. Edward J. Stieglitz, M.S., M.D., F.A.C.P. Consulting Internist, Suburban Hospital, Bethesda, Maryland and Washington Home for Incurables; Chairman of Staff, 1945-47, Suburban Hospital, Bethesda; Consultant in Geriatrics, Chestnut Lodge, Rockville, Maryland, St. Elizabeth's Hospital and Veterans Administration; Associate, Washington School of Psychiatry; Lecturer in Industrial Medicine, New York University—Bellevue Post-Graduate Medical School; Chairman, Advisory Council on Professional Education, Commission on Chronic Illness; Consulting Editor, *Geriatrics:* formerly Associate Clinical Professor of Medicine, Rush Medical College, University of Chicago; Washington, D. C. 718 pages. Illus. Price $15.00, cloth. Philadelphia: J. B. Lippincott Co., 1954.

CARDIOVASCULAR SURGERY. Gerald H. Pratt, M.D., F.A.C.S. Associate Clinical Professor of Surgery, New York University College of Medicine; Attending Surgeon and Chief of Vascular Clinic, St. Vincent's Hospital, City of New York; Attending Surgeon, St. Clare's Hospital, City of New York; diplomate of American Board of Surgery; Consultant to the U. S. Naval Hospitals, Meadow Brook Hospital, Long Island; All Souls Hospital, Morristown, New Jersey. 843 pages. Illus. Price $15.00, cloth. Philadelphia: Lea & Febiger, 1954.

LET'S EAT RIGHT TO KEEP FIT. Adella Davis, A.B., M.S. Consulting nutritionist. 322 pages. Price $3.00, cloth. New York: Harcourt, Brace & Co., 1954.

BEYOND THE GERM THEORY. The Roles of Deprivation and Stress in Health and Disease. A New York Academy of Medicine Book. Iago Galdson, M.D., Editor, 182 pages. Price $4.00, cloth. New York: Health Education Council, 1954.

PERIPHERAL CIRCULATION IN MAN. Edited by G. E. W. Wolstenholme, O.B.E., M.A., M.B., B.Ch., and Jessie S. Freeman, M.B., B.S., D.P.H.; assisted by Joan Etherington. For the Ciba Foundation. 219 pages. Illus. Price $6.00, cloth. Boston: Little, Brown & Co., 1954.

75 YEARS OF MEDICAL PROGRESS. Louis H. Bauer, M.D., Editor. 279 pages. Illus. Price $4.00, cloth. Philadelphia: Lea & Febiger, 1954.

SYNOPSIS OF OBSTETRICS. By Jennings C. Litzenberg, B.Sc., M.D., F.A.C.S. Late Professor Emeritus of Obstetrics and Gynecology, University of Minnesota Medical School, Minneapolis. 4th ed. rev. by Chas. E. McLennan, M.D., Professor of Obstetrics and Gynecology, Stanford University School of Medicine, San Francisco. St. Louis: C. V. Mosby Co., 1952. 378 p. Illus. Cost $5.50.

This treatise, originally written by a well-known and beloved authority on obstetrics and now revised and edited by a former associate of his, will always be well received and certainly well worth reading and reviewing. It definitely adheres to the well-known conservative tendencies of Dr. Litzenberg, but has kept up with the times. When one is lost and needs guidance, without any lengthy reading, he is put back on the right track.

The book serves as a guidepost and keeps one from dangerous detours. It mentions old methods and, if they are obsolete, depreciates them. It contains a clear, concise explanation of the Rh factor and its problems. This is about as clear as one can ever get on this subject in so short a space.

All in all, this is a very useful book, both for the general man and the specialist.

J. F. Bicek, M.D.

FUNDAMENTALS OF PSYCHOANALYTIC TECHNIQUE. By Trygve Braatøy, M.D., formerly Head of Psychiatric Department, Ullevol General Hospital, Oslo, Norway. 404 pages. New York: John Wiley & Sons, Inc., 1954. Price $6.00.

Known for his attention to descriptive material and the biological and physiological aspects of psychoanalysis, the late Trygve Braatøy concentrated on these points in his final work, "Fundamentals of Psychoanalytic Technique," published in May by John Wiley & Sons.

The new book departs from the "logical" procedure that parallels the development of psychoanalytic treatment. A volume for analysts in training, it gives the student a greater freedom in approach by beginning with the emotions of the analyst himself. Braatøy then goes into the questions of diagnosis and indications for psychoanalytic treatment, including risks, evaluates the therapeutic potentialities and dangers, and concludes with general and special aspects of interpretation.

In a letter to his publisher, Dr. Braatøy stated his belief and practice in these words: "The unconscious, the infantile experiences of a patient are important in explaining his present behavior and words. But this indirect explanation must fit what the therapist can see and hear and feel directly.

Although he did not underestimate the value of theory, Dr. Braatøy believed in the importance of relating psychoanalyis to its biological and physiological roots. He classified his own work as belonging to the psychosomatic approach in contemporary psychiatry. "The risks connected with explanations derived from physiology and experimental biology," Dr. Braatøy wrote, "are best countered by always returning to clinical material (which) decides whether our approach, including our theory, is right."

Minnesota-born and of Norwegian ancestry, Dr. Braatøy completed his medical studies in Norway, spent a period of neurological work in the Clinique Charcot in Paris, and later at the Psychoanalytical Institute of Berlin under the late Dr. Otto Fenichel. He was the author of many books and papers, the latest volume partly written during his two-year stay at The Menninger Foundation as a training analyst. In 1953 he resigned as head of the psychiatric department of Ullevål General Hospital in Oslo in order to devote more time to therapeutic work.

THE BILLROTH I GASTRIC RESECTION, with Especial Reference to the Surgery of Peptic Ulcer. By Horace G. Moore, Jr., and Henry N. Harkins. 175 pages. Illus. Price $7.50. Boston: Little, Brown and Company, 1954.

The reviewer found this book a particular stimulus and pleasure. From the standpoint of make-up, typography, type and quality of illustrations, and other purely mechanical matters, it is well done, a pleasure to handle, a pleasure to read. The subject matter concerns not only the Billroth I operation, but includes a great deal of valuable, informative and interesting material on the larger general subject of surgery for peptic ulcer. To illustrate—there are eight main chapters: of these, only two deal primarily with the Billroth I procedure. These are Chapter V on Surgical Technique, comprising an extensive, detailed, step-by-step elucidation that is clear and graphic, with excellent illustrations, and in itself well worth the price of admission, and Chapter VII on Criticisms and Advantages, which appealed to the reviewer as a fair and honest recital of the pros and cons without the weighting and bias occasionally seen in the dissertations of those who are enthusiastic proponents of something that is not generally or entirely accepted. The other chapters deal with the history and rationale of gastric resection for peptic ulcer, selection of patients, pre- and postoperative care, and postgastrectomy syndromes. These chapters deal well and fully with the points enumerated in clear and concise fashion. The book ends with a series of interesting biographical sketches of some of the outstanding gastric surgeons of the past.

This book fills a real need. In any informal discussion among surgeons, many of the objections to the Billroth I procedure are grounded on a species of handed-down folklore. The disuse into which the operation has fallen is a good illustration of how a procedure can, because of poor results or unhappy experiences in past years, suffer unmerited neglect. Newer techniques and concepts, here collected in one readily available compendium for the first time, have vitiated many of the arguments which have, starting a long time ago, been carried into the present as proofs of the operation's undesirability, without much in the way of critical re-evaluation. While the reviewer does not feel convinced that all gastric resections should be of the Billroth I variety, it should be more true than it now is, as Dr. J. M. Waugh points out in his preface, that every gastric surgeon should be able to carry out expertly a Billroth I and a Billroth II operation, just as the skilled gynecologist can carry out both vaginal and abdominal hysterectomy.

The book has a rather limited field of interest, namely, surgeons doing gastric surgery, but within that field the reviewer feels that the book can be unhesitatingly recommended. Many surgeons would like to use the Billroth I more, but have been reluctant to do so because of unfamiliarity with it. This book has the answers and the help that are needed to banish such unfamiliarity. The reader (the reviewer, too) may not agree with everything that is said in the book, but he will find very rewarding a diligent and thorough perusal of it, as did the reviewer. The material is presented sanely, clearly and in fulsome detail in a matter where the previously available information was scarce and hard to come by. The Billroth I operation can and should be done more than it is, and this book is both a stimulus in that direction, and an outstanding aid to those wishing to travel in that direction.

BLUE SHIELD-BLUE CROSS NEWS

(Continued from Page 542)

scribers, or 2.0 per cent greater than anticipated for the year to date.

For the first two months of 1954, 186 cases each amounting to over $1,000.00 were allowed by Blue Cross. The total amount of benefit for these 186 cases was $248,078.99 or an average of approximately $1,334.00 per case. The average length of stay on these more than $1,000.00 cases was 53.6 days.

MEDICAL MALPRACTICE INSURANCE

(Continued from Page 538)

naturally muses on what can be done to ameliorate the situation.

Our profession has been confronted in the past with equally as challenging problems, and they have been corrected to the degree whereby we are the envy of our neighbors. Here is a challenge our state association should consider very seriously. Personally, I have complete confidence that a small group headed by our nationally recognized attorney, Manley Brist, can build an insurance structure of which our State Medical Association would be proud. I am confident that there would be no difficulty in obtaining financial aid from the individual members as an operating fund to get it launched, even as we did in the building of the Blue Shield Insurance company. If we, as a state medical family, operated such a mutual insurance policy to protect us, we then would certainly play a most cautious hand among ourselves, as well as toward the patient, when this ugly malpractice cloud appears on the scene. It would cement our family into a finer appreciation of our mutual problems toward one another. Thoughtful consideration would be a strong guide and a helpful brake in these moments. Human nature being as it is, we would not be so liable to act maliciously in matters pertaining to our own insurance child.

Again, our medical family is confronted with a challenge. I believe, with united effort we can lick it to the envy of our neighbors.

J. J. Heimark, M.D.

Fairmont, Minnesota
June 1, 1954

Minnesota Medicine

Journal of the Minnesota State Medical Association, Southern Minnesota Medical Association, Northern Minnesota Medical Association, Minnesota Academy of Medicine and Minneapolis Surgical Society

Volume 37 August, 1954 Number 8

Symposium on Fluid and Electrolyte Balance

One hundred and thirty-six years have passed since Claude Bernard referred to the composition of fluids surrounding cells as the "milieux interieur." Since that time there has been a progressive accumulation of knowledge of the physiology of fluid and electrolyte balance in man and experimental animals.

The past ten years have witnessed a remarkable growth in the clinical usefulness of basic fluid and electrolyte physiology, so that a working knowledge of new terms and techniques of water and electrolyte balance have become essential in the care of many medical and surgical diseases. The "milli-equivalent doctor" and the "get-em-well doctor" have become one and the same.

It is the purpose of this "Symposium" to present in a useful way the fundamental physiology of body fluid and electrolytes with an approach to the application of recent knowledge in the everyday care of patients. This series of articles, including the basic physiology, pediatric, medical and surgical facets of fluid and electrolyte balance, has been prepared through invitation by Dr. Edward B. Flink, associate professor of medicine, Dr. Richard Raile, instructor in pediatrics, Dr. Howard Worthen, research fellow in pediatrics, and Dr. Bernard Zimmermann, assistant professor of surgery, University of Minnesota.*

S. W. A.

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*Dr. Flink is also Chief of the Medical Service, Veterans Hospital, Minneapolis, Minnesota.

FLUID AND ELECTROLYTE BALANCE

Part I: Basic Considerations

EDMUND B. FLINK, M.D., and BERNARD ZIMMERMANN, M.D.
Minneapolis, Minnesota

SINCE it has become apparent that a symposium on electrolyte and fluid problems might be of value at this time, the authors have planned to review the most important general principles and the most pressing problems involved in clinical fluid and electrolyte disturbances. It is our aim to make this series of articles useful practical guides to diagnosis and therapy.

Balance

To start with, it is important to define some terms and to explain the arithmetic of this subject. The idea of balance or equilibrium in its basic principles is as simple as a bank balance. Any element or substance for which adequate analyses are available and which enter into metabolism can be measured in terms of balance. A negative balance means a deficit of the substance and a positive balance means storage of an excess. When intake equals output, then balance or equilibrium exists. One of the most difficult problems, however, is to ascertain that a patient has a deficit or an excess when he is first seen and intake and output data are not available.

Dehydration

Dehydration is strictly defined as a deficiency in water. Simple water deprivation, however, is infrequent, it being much more commonly accompanied by a concomitant loss of electrolytes.

Acidosis and Alkalosis

Acidosis and alkalosis are terms used to indicate deviations of the acid-base equilibrium from normal. PH is a convenient logarithmic representation of the reciprocal of the hydrogen ion

TABLE I. COMMON IONS IN BODY FLUIDS
(Atomic and molecular weights are recorded to the nearest gram).

	Atomic Weight	Equivalent Weights
Simple Ions		
Sodium ($Na+$)	23 Gm	23 Gm
Potassium ($K+$)	39 Gm	39 Gm
Calcium ($Ca++$)	40 Gm	20 Gm
Magnesium ($Mg++$)	24 Gm	12 Gm
Chloride (Cl)	35 Gm	35 Gm
Complex Ions		
*Phosphorus ($HPO_4=$)	31 Gm	31/1.8†
*Sulfur ($SO_4=$)	32 Gm	16
**Carbon Dioxide (CO_2+HCO_3)	22.26 liters	22.26 liters

*Determined in terms of mg. of sulfur or phosphorus usually.
**Note that carbon dioxide is measured as a gas.
†Factor takes into account small amount of monovalent (H_2PO_4-) at pH of serum.

TABLE II. MOLECULAR WEIGHT OF SALTS COMMONLY USED IN THERAPY

Sodium chloride	58
Potassium chloride	74
Ammonium chloride	53
Sodium bicarbonate	84
Sodium lactate	112

TABLE III. SAMPLE CALCULATIONS OF ELECTROLYTE CONCENTRATIONS

	Mg/100 cc serum	Mg/liter	mEq/liter
Sodium ($Na+$)	325	x 10 = 3250	3250/23 = 141
Chloride (as $NaCl$*)	580	x 10 = 5800	5800/58 = 100
Protein	Gm.% 7.0		7 x 2.43** = 17
CO_2	Vol.% 55 cc	x 10 = 550 cc	550/22.2 = 24.8

TABLE IV. NORMAL VALUES OF ELECTROLYTES IN SERUM (mEq/1)

Basic ions (cations)		Acid ions (anions)		Sum equals 130
$Na+$	138—145	$Cl-$	100—106	sum of these equals about 25
$K+$	4—5.6	CO_2	23—28	
$Ca++$	4.5—5.5	PO_4	1.7—2.3	
$Mg++$	1.5—2.1	Protein	14—17	
		$SO_4=$	.5—1.0	
		Organic Acids	5—7	
Average	155 mEq/1.		155 1 mEq/1.	

*Most laboratories report chlorides in terms of milligrams of molecular sodium chloride per 100 cc rather than as mg. of chloride alone.
**2.43 is a derived factor for this conversion.

concentration. The body tries to maintain pH in a very narrow range (7.35-7.42). In order to elucidate acid-base problems, pH determinations have been invaluable and are very helpful clinically if available, but can be usually dispensed with in most ordinary clinical cases. The details of acid-base regulation will be discussed below.

Milliequivalents

Electrolytes (particularly sodium, chloride, carbon dioxide and potassium) are best reported and thought of in terms of milliequivalents per liter rather than by any other units. This is as reasonable as counting assets in terms of dollars and cents instead of pounds of tin, copper, salt and bread. The sum of ions with a positive charge (cations) must equal the sum of ions with a negative charge (anions). Since equivalents are an expression of the number of ions, the same equality holds when values are expressed in terms of equivalents or milliequivalents. It does not, of course, apply to other units such as milligrams.

An equivalent weight of an element is the atomic weight divided by the valence of the element. A milliequivalent weight is 1/1000 of an equivalent weight. A milligram being 1/1000 of a gram, milligrams per liter can be converted to milliequivalents per liter by merely dividing the value in milligrams by the equivalent weight. Since a molecular or equivalent weight of carbon dioxide (CO_2) occupies 22.26 liters, the factor 2,23 is used to convert volumes per cent to mEq./1.

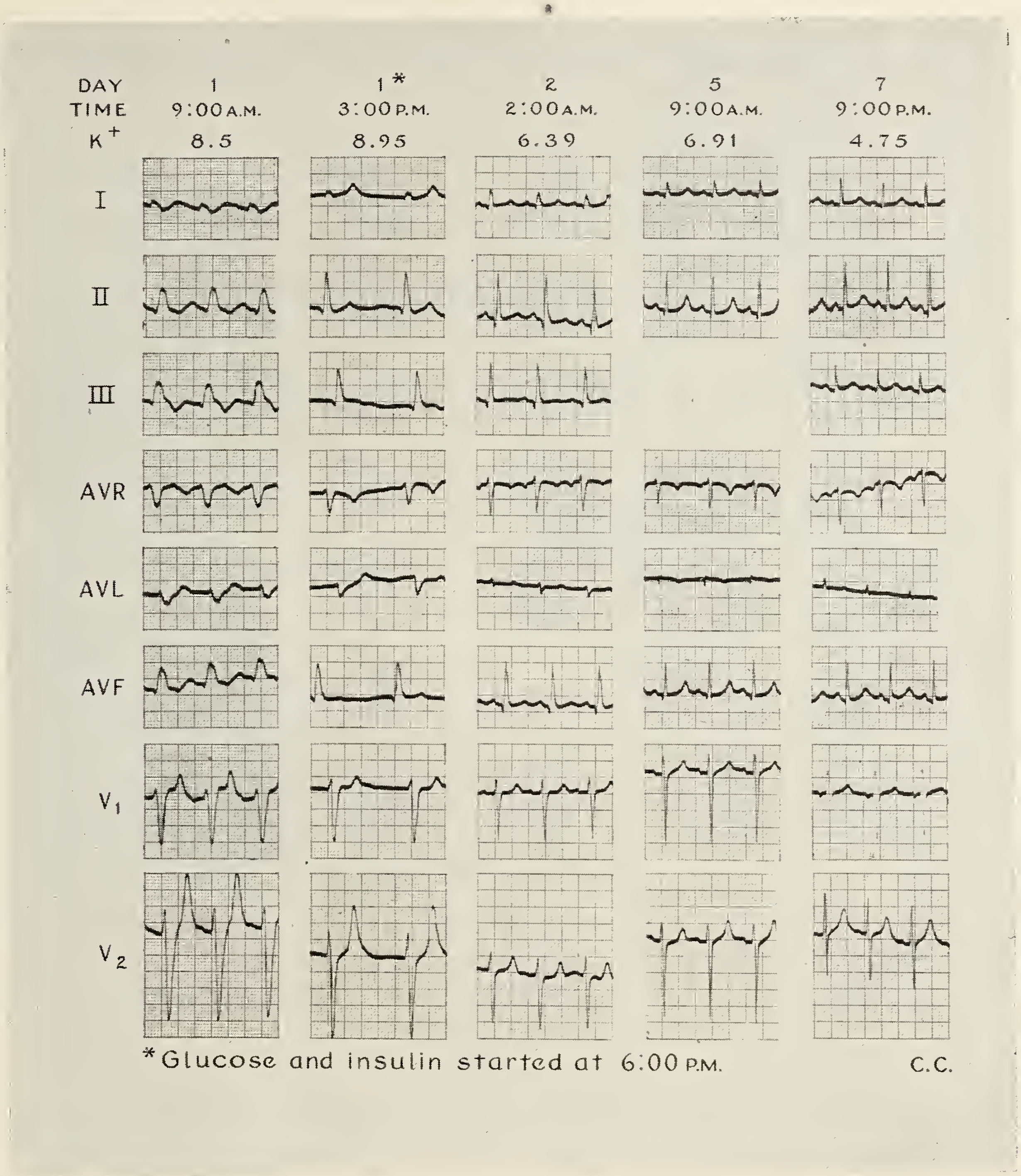
Osmotic pressure means the pressure which a dissolved substance (solute) exerts when confined by a membrane which it cannot penetrate. An osmolar solution contains one molecular weight of a solute. Ionized solutes exert an osmotic effect equivalent to the number of ions in a molecule. A milliosmolar solution refers to 1/1000 of an osmolar solution.

Tables I and II record the atomic weight, equivalent weights and molecular weights of the elements and compounds which are of clinical importance.

In order to illustrate methods of conversion, sample calculations for hypothetical values of Na,$^+$ Cl, Protein and CO_2 are given in Table III.

Table IV gives the normal range of values for serum electrolytes.

Ions other than Na,$^+$ K,$^+$ Cl^- and HCO^-_3 which are present in small concentration are frequently recorded in terms of milligrams or grams. For the sake of uniformity, however, it is better to report all of them as milliequivalents. As is implied in Table IV, the sum of the two main anions Cl^- and CO^-_2 is a clinically useful figure. Sodium is the main cation and magnesium and calcium vary within such a small range that their contribution to osmolarity is relatively fixed. Similarly the small range within which potassium varies is of little consequence from the osmolar standpoint, though, as will be shown later, it has highly significant clinical implications. The sum of sodium and potassium is 142-150 mEq/1. The last four items in the anion column are often not determined, are frequently as "R" and can be calculated by subtraction from the total cations. This process of calculating undetermined quantities by addition and subtraction is possible only when milliequivalents are used.



*Glucose and insulin started at 6:00 P.M.

Fig. 1: This series of tracings records the progress of a patient who had anuria and hyperpotassemia. Note the wide QRS complexes in the first 2 tracings and the high and peaked T waves in the first 2 tracings in V_2 especially. The K concentrations are recorded at the top. Note the progress of the electrocardiograms to normal as the serum potassium returned to normal.

The advantages of recording chemical values in terms of milliequivalents are further illustrated by the following samples:

(1)

Chloride (Cl^-)	105 mEq/1	= 608* mg %
Bicarbonate (CO_2)	25 mEq/1	= 56 volumes %
Sum	130 mEq/1	?

One cannot add milligrams per cent and volumes per cent. There is no doubt that one can see that the figures are in the normal range with either method of notation and the sodium concentration cannot be far from normal under these circumstances. However, the following data derived from a case of metabolic alkalosis accentuates the difficulties involved with the older units.

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*Reported as milligrams of NaCl per 100 cc.

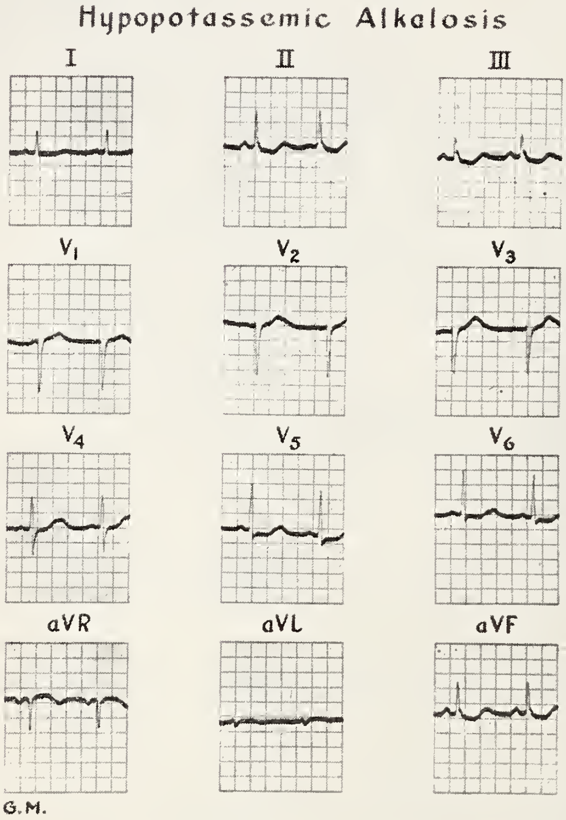


Fig. 2: This electrocardiogram records some of the abnormalities found in hypopotassemic states (not just hypopotassemic alkalosis). The QT interval is prolonged, the ST interval is depressed and the T wave is inverted diphasic in leads 2 and 3, there is often a prominent "U" wave (after the T wave) in V_2 especially. Various degrees of heart block may also occur. The most significant findings are the prolonged QT and ST segment depression. (The apparent prolongation of the QT interval is actually due to blending of "U" wave with the "T" wave.)

(2)

Chloride (Cl)	90 mEq/1	= 522 mg %
Bicarbonate (CO_2)	40 mEq/1	= 89 volumes %
Sum	130 mEq/1	?

In this example of some of the chemical findings in alkalosis it can be seen quite readily that the sum of major anions is normal and this gives the additional information that the major cation concentration (sodium) must also be approximately normal. There is no way of deducing any such quantitative information from the concentrations recorded in milligrams per cent and volumes per cent. Any number of such examples could be cited.

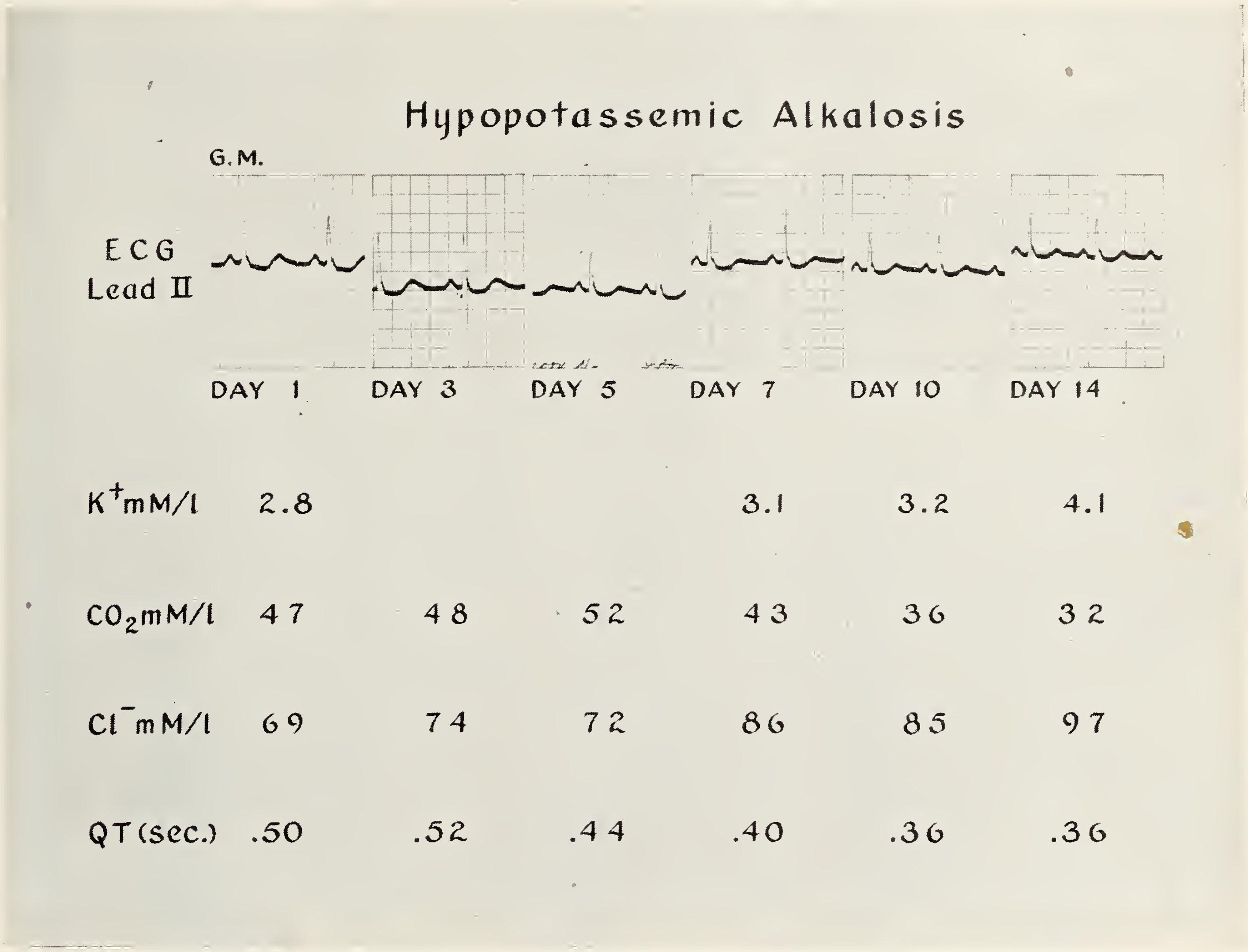
The bicarbonate and chloride concentrations cannot be used as a guide to the concentration of sodium in situations where either uremia or ketosis exists, for in ketosis there is an accumulation of organic acids which displace some of the bicarbonate so that if the sum of carbon dioxide and chloride concentrations were to be used as in the example above it would give an erroneously low estimate of cations. In uremia, retention of inorganic acid radicals (HPO^4 and SO_4) cause a similar displacement of CO_2. Uremia, incidentally, is one condition where expression of phosphorus concentration in terms of mEq/1 is useful to indicate the significant contribution it makes to the anion column in this condition.

Most clinical laboratories can perform chloride and carbon dioxide determinations. Though the availability of sodium and potassium determinations is much more limited, the electrocardiogram furnishes a means of recognizing hyperpotassemia and hypopotassemia. Electrocardiographs are available in every hospital and are very valuable as guides to therapy with potassium and as a means of detecting both deficiencies and intoxication. Patterns of each extreme are distinctive as illustrated by Figures 1 and 2. The first of these shows the characteristic tracing in a patient with high potassium which is subsequently reduced by administration of glucose and insulin. The second gives the electrocardiographic features of potassium depletion. Figure 3 illustrates gradual transition toward normal.

The Fluid Spaces: Their Boundaries and Constituents

The relative ease with which a sample can be obtained from the circulating blood inevitably results in the tendency of the clinician to describe abnormalities of fluid and electrolyte physiology in terms of the blood or plasma values with which they are associated. Although the value of such determination cannot be discounted, any rational consideration of the fundamentals of fluid and electrolyte physiology must take into account a consideration of all the major fluid spaces and the boundaries which separate them.

Conventionally, the fluid volume in which the soluble constituents of the body are distributed is divided into the intracellular fluid and the extracellular fluid, the latter being in turn composed of the blood plasma and interstitial fluid. The extracellular volume is usually given as 20 per cent of the body weight although this value varies quite widely depending on the methods of measurement which are used. The thiocyanate method gives a somewhat larger figure (23 per cent), whereas more recent methods such as inulin and thiosulfate which are believed to determine the extracellular space with greater accuracy give values of 17 per cent to 18 per cent. The intracellular fluid has been considered to comprise about 50 per cent of the body weight, so that the total body water for the adult human amounts to approximately 70 per cent of body weight. Here again more recent methods such as the use of heavy water for the direct measurement of total body water suggest that this value should be considerably lower, probably in the neighborhood of 61 per cent for men and 52 per cent for women[5]. The difference is the result of the somewhat larger proportion of fat in the latter.



	DAY 1	DAY 3	DAY 5	DAY 7	DAY 10	DAY 14
K^+mM/l	2.8			3.1	3.2	4.1
CO_2mM/l	47	48	52	43	36	32
Cl^-mM/l	69	74	72	86	85	97
QT (sec.)	.50	.52	.44	.40	.36	.36

Fig. 3: A series of chemical determinations on serum and lead II of the electrocardiogram on the corresponding day are recorded. Note the gradual transition toward normal of the chemical findings and a return to normal of the electrocardiogram.

The remarkable differences in the ionic composition of the two major fluid compartments are shown in Figure 4 which is reproduced from Gamble's Lane lecture.[6]

The blood plasma and interstitial fluid are essentially the same in composition except for the difference in concentration of protein which is much higher in the plasma. The composition of the cell fluid, on the other hand, is radically different from that of the extracellular fluid. The most striking difference is that sodium is the major extracellular cation and potassium occupies this position inside the cell. Although it was previously believed that sodium did not enter the cell, it has been definitely shown that small amounts of sodium, such as are represented on the diagram, are actually present. Chloride is a major extracellular anion and phosphate the predominant intracellular one. The question of whether there is ever any significant amount of chloride inside the cell under either normal or abnormal circumstances remains to be settled. Most calculations relating to the distribution of electrolytes in the body even under circumstances of disease are based on the assumption of the exclusively extracellular position of chloride. The mechanism by which this inequality of distribution of sodium and potassium is maintained represents a problem as yet largely unsolved, but one of intense interest to those concerned with the basic properties of living membranes. In the past, it was simply assumed that the cell membrane was impermeable to sodium ions. The application of tracer techniques, however, has shown that this is in no way the case, for the cell membrane is apparently freely permeable to sodium ion and the phenomenon cannot, therefore, be explained on the basis of membrane permeability alone. The fact is, that active processes within the cell are constantly working to extrude sodium and thereby maintain the extreme gradient across the cell membrane which normally exists. This process requires the expenditure of energy which is produced by cellular metabolism and, although numerous theories for the mechanism by which energy is utilized to maintain this gradient have

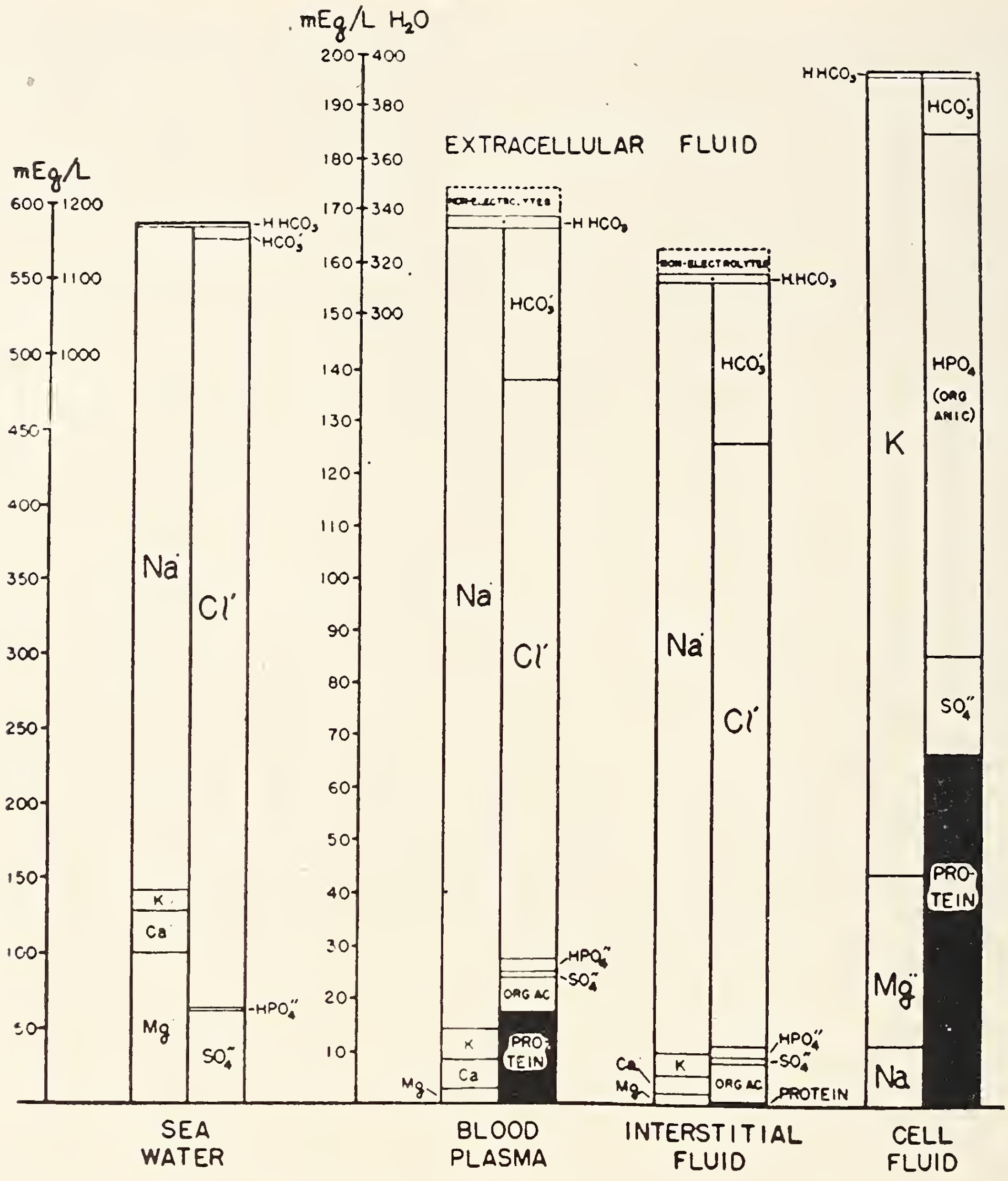


Fig. 4: Concentration of major constituents in blood plasma, interstitial fluid, intracellular fluid and sea water. Gamble, J. L., Lane Medical Lectures: Companionship of water and electrolytes in the organization of body fluids. Reproduced by consent of the author and the Stanford University Press.

been proposed, the question is still an obscure one. Likewise the nature of the high intracellular potassium concentration with respect to the level in the extracellular fluid remains to be elucidated. The maintenance of these two extreme gradients across the cell membrane is surely one of the most fundamental processes of life and is as important to the normal function of living cells as the assimilation of oxygen and the transport of carbon dioxide. As will be discussed in the succeeding sections, recent studies of electrolyte changes during illness have shown that the great alterations from a normal intracellular-extracellular gradient of sodium and potassium can occur, the characteristic response, in most instances, being one of loss of potassium from the cell with entrance of sodium from the extracellular fluid to replace it. Thus, as might be anticipated, the characteristic of "sick cells" is the loss of ability to expend energy in the maintenance of these unequal concentrations and the production of an intracellular ionic pattern more closely resembling that of the medium by which the cells are surrounded.

The first column in Figure 4, which describes the composition of sea water, gives a clue to the background of the astounding relationship which exists between chemical components of the cell and those of the fluid which bathe it. It will be observed that the concentrations of extracellular fluid and sea water are remarkably similar except for few significant differences, most important of these being that the overall concentrations of salt is over three times as high in the sea as in mammalian extracellular fluid. The interpretation of this is that the internal environment of land creatures today is that which existed in the sea at the time when the prototypes of these animals emerged from the ocean millions of years ago. The progenitors of the vertebrates acquired a closed circulation and thereby segregated themselves from their oceanic environment in the early Cambrian era when the sea was much more dilute than it is now. Though the total salinity of the sea has greatly increased, the relationship of concentrations of the ions to each other has remained the same. The outstanding exception to this is the case of magnesium whose concentration has mounted much more rapidly. This principle was demonstrated by Macallum whose extensive studies of the extracellular fluid in many species established the relationship between ionic concentrations in the organisms as they exist today and the calculated concentrations in sea water at the time their original prototypes acquired a closed circulation.[1,8] The principle is of more than amusing historical interest, for the characteristic of the ocean with its vast volume and complete immutability except for the changes wrought by millions of years is one of extraordinary constancy of both composition and physical qualities. Consequently, the basic requirement for cellular life which began in the sea is one of a constant fluid environment. This necessity for constancy is summarized in Claude Bernard's famous dictum which stated that constancy of the internal environment is the necessary condition for independent life.[2] The physiologic and pathologic principles with which these papers are to be concerned all revolve around the efforts of the body to maintain these conditions.

The Regulation of Water and Fluid Spaces

Although the cell in a rigid sense is permeable to sodium ion, the fact that sodium is predominantly maintained in an extracellular position endows it with osmotic properties identical with those of a substance to which the membrane is actually impermeable. Thus sodium, being the major extracellular component, governs the major osmotic force which stabilizes the distribution of water between the cells and the extracellular compartments. This fact was first stated by Darrow and Yannet, who, in 1935, postulated that the volume of the cell was not sensitive to the overall extracellular volume but only to the total osmolarity (and therefore the concentration of sodium) in the extracellular fluid.[3] An increase in sodium concentration results in the movement of water from the cell and a consequent shrinking of cells until the osmolar concentrations of the two media are again in equilibrium. Conversely, a loss of sodium ions from the extracellular fluid results in an inward motion of water until a similar equilibrium is attained. Furthermore, addition of water with dilution of electrolytes has the same effect as removal of salt. The phenomenon is illustrated in Figure 5 by a diagrammatic representation of the changes in volume consequent to adding or subtracting 500 milliosmols of sodium chloride from the extracellular fluid compartment. As will be seen, these shifts of water may be the basis for clinical aberrations of the severest nature.

The Regulation of Salt Balance

In view of the fundamental relationship between extracellular sodium and intracellular volume it is important to consider the factors which maintain the concentration of this ion in the extracellular fluid. Most human beings ingest considerable excesses of salt in the course of a day, intakes of three to six grams being common. For some reason our taste leads us to take far larger quantities of sodium chloride than are actually required. One can, on the other hand, get along with much less and herein lies one of the most important homeostatic mechanisms of the kidney. Diets such as are given in the treatment of cardiac disease which contain as little as 200 mg of sodium may be tolerated for long periods of time. This results from the ability of the kidney to reabsorb nearly all of this ion from the glomerular filtrate and replace it in the blood stream for the maintenance of the proper extracellular concentration. This power for conservation of sodium does not obtain with respect to potassium which is put out in the urine in considerable amounts even when eliminated from the diet or when extracellular levels become seriously depressed. Though this difference of economy between sodium and potassium ions seems superficially unsound, we are again reminded of the circumstance that we are basically marine organisms highly adapted to the function of carrying our sea water environment around with us. The vertebrate land animal is endowed with the ability to maintain his sodium-salt environment in a world where potassium is abundant and sodium is precious and the herbiverous animal in particular can ingest sodium-salt only by actual visits to his watery ancestral home or by seeking the dried up remnants of the ocean which exist in the form of salt deposits.

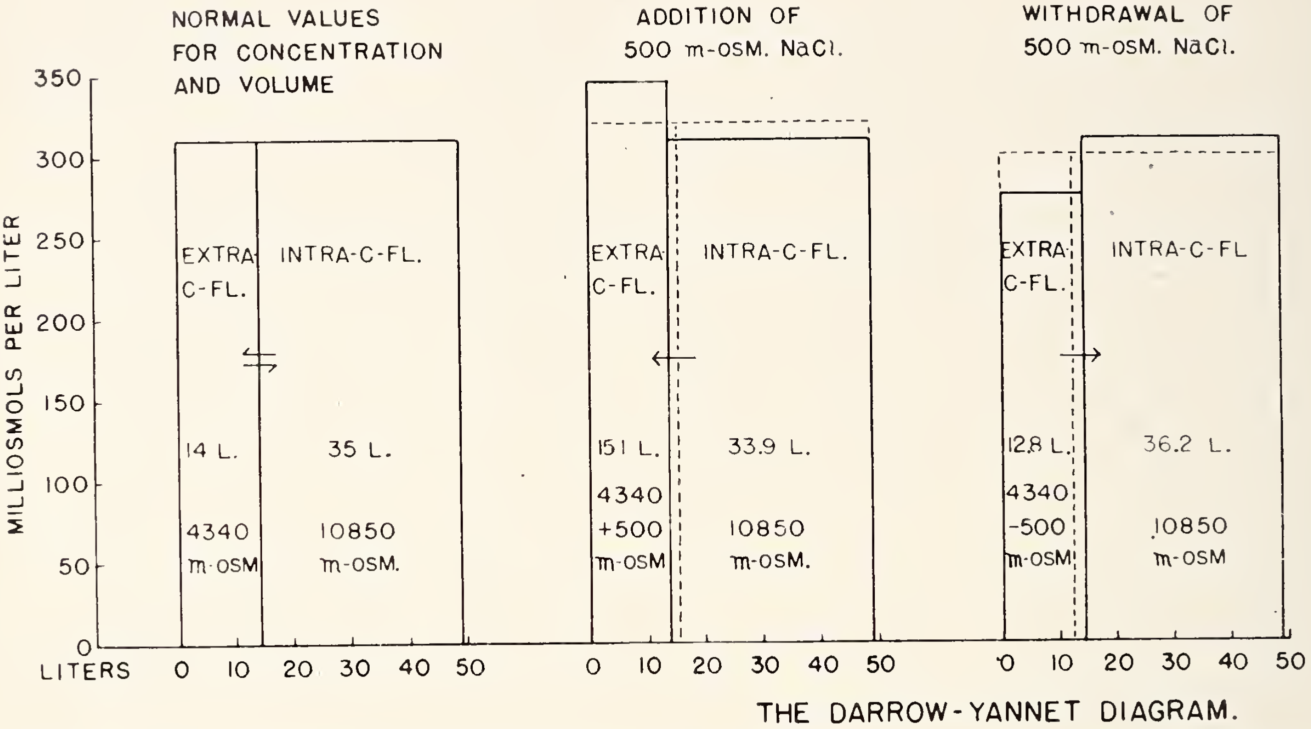


Fig. 5: The Darrow-Yannet diagram illustrating the effects on intracellular volume of adding or removing sodium from the extracellular fluid compartment. Reproduced from Gamble, J. L., Extracellular fluid, with kind permission of the author and the Harvard University Press.

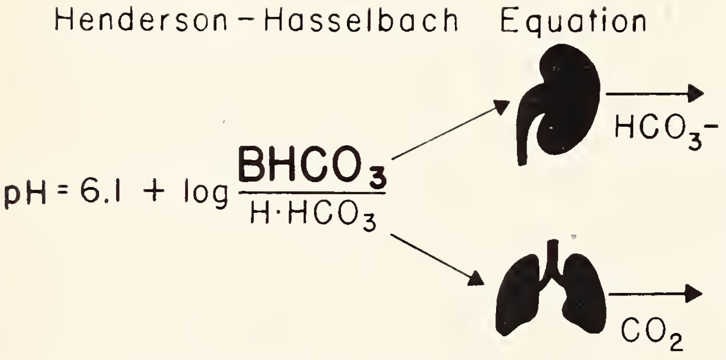

Henderson - Hasselbach Equation
pH = 6.1 + log BHCO3 / H·HCO3
HCO3-
CO2

Fig. 6: The Henderson-Hasselbalch equation for the relationship between pH and the concentrations of carbonic acid and bicarbonate. This emphasizes that the normal concentration of bicarbonate is much greater than that of carbonic acid (ratio 20:1) and that the former is controlled mainly by the kidney, the latter being under control of the lungs.

The Regulation of Acid-Base Balance—Acidosis and Alkalosis

Most critical of conditions for which constancy must be maintained in a tolerable environment for living cells is that of the concentration of hydrogen ions. The pH is maintained within narrow limits in the normal plasma of from 7.35 to 7.42. The first line of defense in this regulation is the buffering power of the weak acid carbonic which exists in equilibrium with bicarbonate ions as shown below.

$$H_2CO_3 \rightleftarrows H^+ + HCO_3^-$$

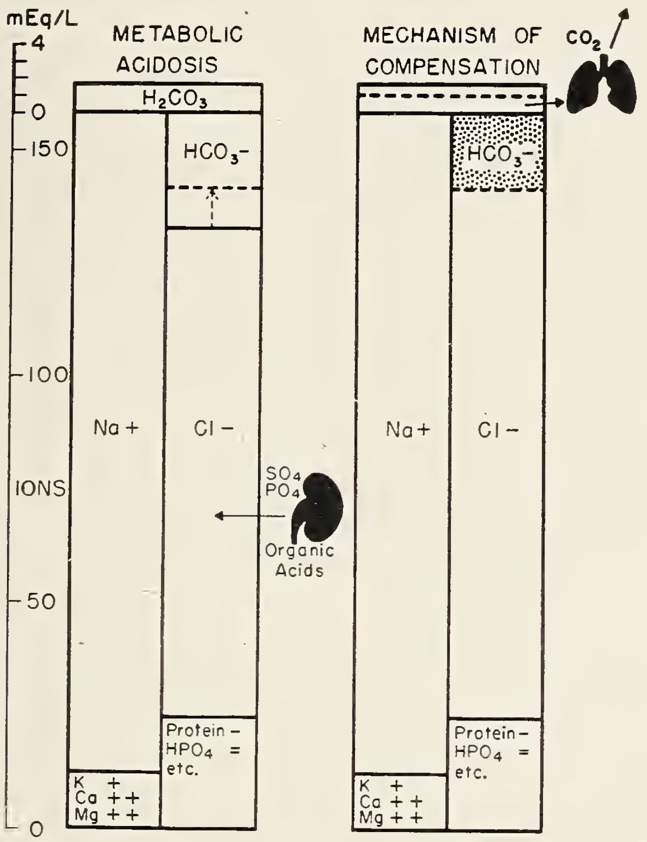


Fig. 7: Metabolic acidosis. Left: Primary disturbance caused by retention of acids by the kidney. Right: Compensation is achieved by hyperventilation and consequent decrease in carbonic acid.

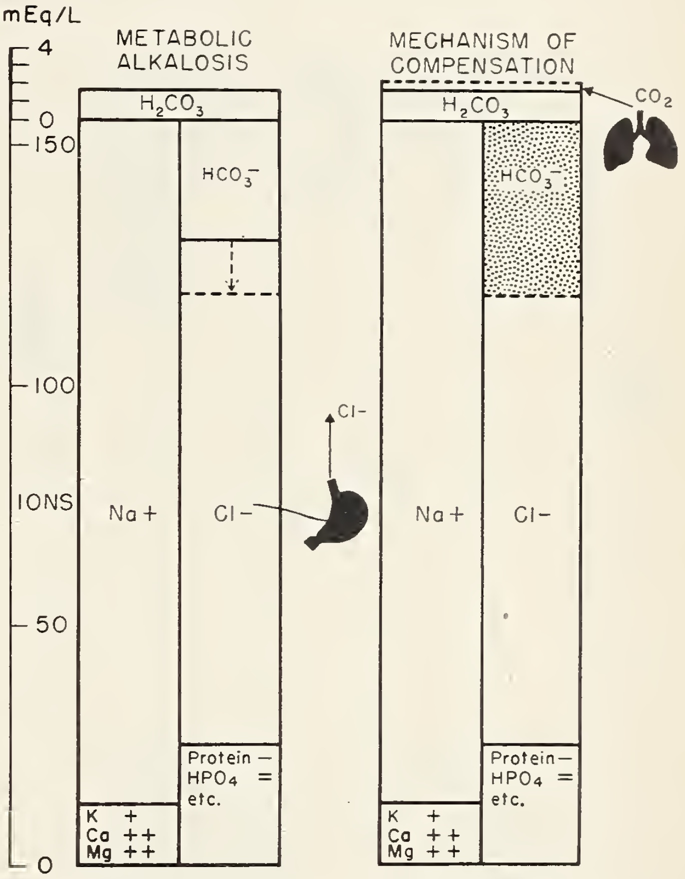


Fig. 8: Metabolic alkalosis. Left: Primary defect caused by loss of chloride through vomiting and consequent increase in bicarbonate. Right: Compensation is achieved by retention of carbon dioxide by the lungs and consequent increase in carbonic acid of the plasma.

The combination is a buffer because when more hydrogen ions are added to the system, the equilibrium is shifted to the left, more H_2CO_3 is formed and hydrogen ions thereby "absorbed." The relative amounts of carbonic acid and bicarbonate determine the pH and the situation is further stabilized since the concentrations of these two substances are controlled respectively by the lungs and the kidneys. The formula which relates the pH to the dissociation constant of carbonic acid and the existing ratio of carbonic acid to bicarbonate is simply derived from the mass action law which describes the dissociation of an acid into its component ions. This formula which is known as the Henderson-Hasselbalch equation is shown in Figure 6 in such a way as to demonstrate that the bicarbonate is present in much greater quantities than carbonic acid (the normal ratio is 20 to 1). It indicates, furthermore, that excesses of bicarbonate are handled by excretion through the kidney and carbonic acid is removed from the body by conversion to carbon dioxide and excretion through the lung.

Disturbances of acid-base balance are of two types: *Respiratory disturbances* in which the primary effect is on the carbonic acid or dissolved carbon dioxide of the blood with secondary or compensatory changes occurring in the bicarbonate ions, and *metabolic disturbances* in which the primary effect is on the bicarbonate ions with compensatory adjustments occurring in the carbonic acid. Since the regulation of carbonic acid is managed entirely by the lung it is apparent why respiratory disturbances should be manifested in its concentration. Bicarbonate, on the other hand, is altered by metabolic disturbances because it is labile and its concentration is completely limited by the concentration cations which have not already been covered by other anions. The movement of bicarbonate in response to changes in other anions is therefore illustrated in Figure 7 in which the acidosis resulting from failure of excretion of anions in uremia (sulfate, phosphate, and acids) results in a decrease in bicarbonate concentration.

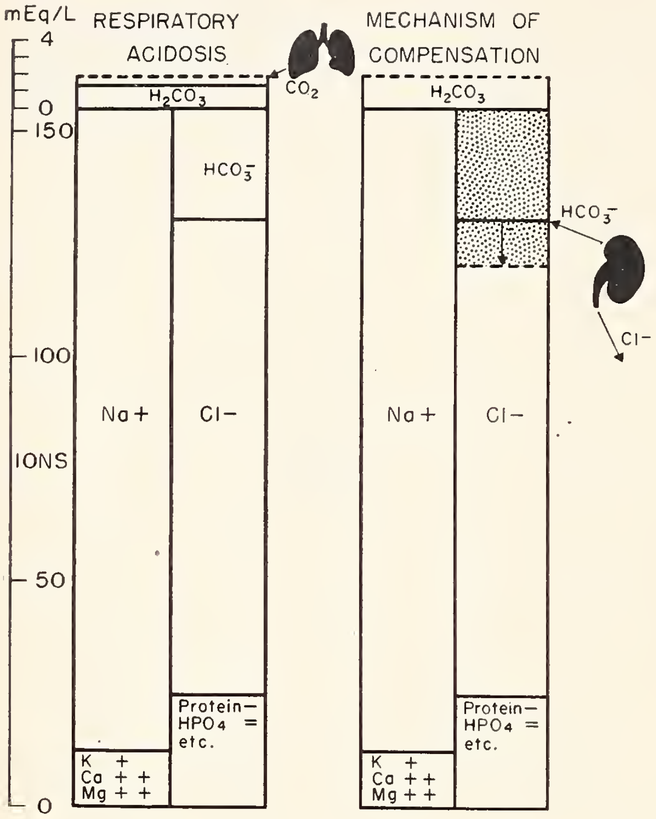


Fig. 9: Respiratory acidosis. Left: Primary defect caused by retention of carbon dioxide due to respiratory impairment. Right: Compensation is achieved by retention of bicarbonate and excretion of chloride by the kidney.

Figure 8 shows the converse situation: an alkalosis is produced by a loss of chlorides through vomiting and is reflected in an increase in bicarbonate concentration.

Consider now the respiratory compensation to these two situations. The effort of the body is, insofar as possible, to return to the normal 20 to 1 fraction of bicarbonate to carbonic acid. If, therefore, bicarbonate decreases as in the situation described for metabolic acidosis, carbonic acid must be decreased as well and this is accomplished by increasing ventilation in order to allow an escape of carbon dioxide by decreasing the concentration of this gas in the alveolae. The characteristic breathing of acidosis results. Conversely, when the bicarbonate is increased through the mechanism of chloride loss, a decreased depth and rate of respiration must take place in order that an accumulation of carbonic acid can balance the accumulation of bicarbonate. Since the final value of the bicarbonate is the value which is actually measured clinically, this value is shown as a stippled area in each of the diagrams.

The majority of disturbances of acid-base equilibrium which are clinically seen are of the metabolic type. Nevertheless, respiratory disturbances are not uncommon and are receiving increasing interest, for example, in connection with thoracic surgery, and inhalation anesthesia. Figure 9 illustrates the situation in disturbances of primary respiratory origin. A decreased ventilation results in accumulation of carbonic acid and the result is respiratory acidosis.

In response to this situation the kidney stops excreting bicarbonate and along with this, in order that the sodium concentration may be maintained, begins to excrete excessive amounts of chloride. The situation is therefore partially compensated by altering the numerator of the fraction in the same direction that the denominator has been changed. The result is a high bicarbonate. The point is that in the compensated situation the bicarbonate is high in both metabolic alkalosis and respiratory acidosis. A measurement of the bicarbonate alone, therefore, or of the carbon dioxide combining power which is usually done in hospitals as a measurement of the bicarbonate, will not distinguish between these two conditions and in order to make this distinction one must have an actual measurement of the pH or at least must have clearcut clinical information as to whether the patient is suffering primarily from a metabolic or a respiratory derangement. Fortunately, the latter information is usually available and it is only in the unusual situation that the physician does not know on clinical grounds which type of disorder is present. Consider then the situation when through nervousness or other mechanisms the patient begins to hyperventilate (Figure 10).

Carbon dioxide is removed and the carbonic acid drops to a low value. Respiratory alkalosis results and the compensation consists in the excretion of large amounts of bicarbonate by the kidneys with a corresponding retention of chloride. Here again a measurement of the bicarbonate or the CO_2 combining power is not a basis for distinction between the depressed values which are characteristic of both respiratory alkalosis and metabolic acidosis. Fortunately again, in most clinical situations the necessary facts are known. Occasionally, however, a determination of pH must be done.

Whereas, the buffering capacity of the blood is responsible for the fine adjustment of the hydrogen ion concentration, the main work of relieving the body of great excesses of acid or alkali is achieved by the kidney. The most continual problem is the removal from the body of large amounts of acid which are discharged from the cells as a result of the oxidation processes of metabolism. The removal of acid is made possible by the fact that urine can be produced at a pH far lower than can be tolerated in the blood. The excretion of such an acid with the conservation of base (cations) is made possible by two mechanisms:

1. Whereas, phosphate ion exists in the blood ordinarily in the form of its dibasic ion and covers two cationic equivalents, it is excreted by the kidney in the monobasic form with corresponding conservation of positively-charged ions.

2. The kidney produces ammonium ions (NH_4^+) for excretion with acid radicals with resultant conservation of important cations such as sodium. As will be seen later the acidosis which results from renal damage involves both the failure to excrete acid radicals and a breakdown in the mechanism for conserving sodium.

The Regulatory Effect of Hormones

Increasing attention in recent years has been directed towards the regulatory effect exerted by hormones on the balance of salt and water. This is a most important subject about which a great deal remains to be learned. Hormones are never entirely responsible for any single physiological phenomenon. All available evidence appears to indicate that in every vital process, which endocrine substances influence, the basic enzymatic pattern exists in the cells themselves, the hormones only acting as catalysts whose function is to regulate the rate of the reactions concerned. This concept does not contradict the obvious fact that such regulation is frequently necessary to life. An example of this is the re-absorption of sodium by the renal tubules which is not far from being completely absent following adrenalectomy but is vastly intensified in the presence of these glands.

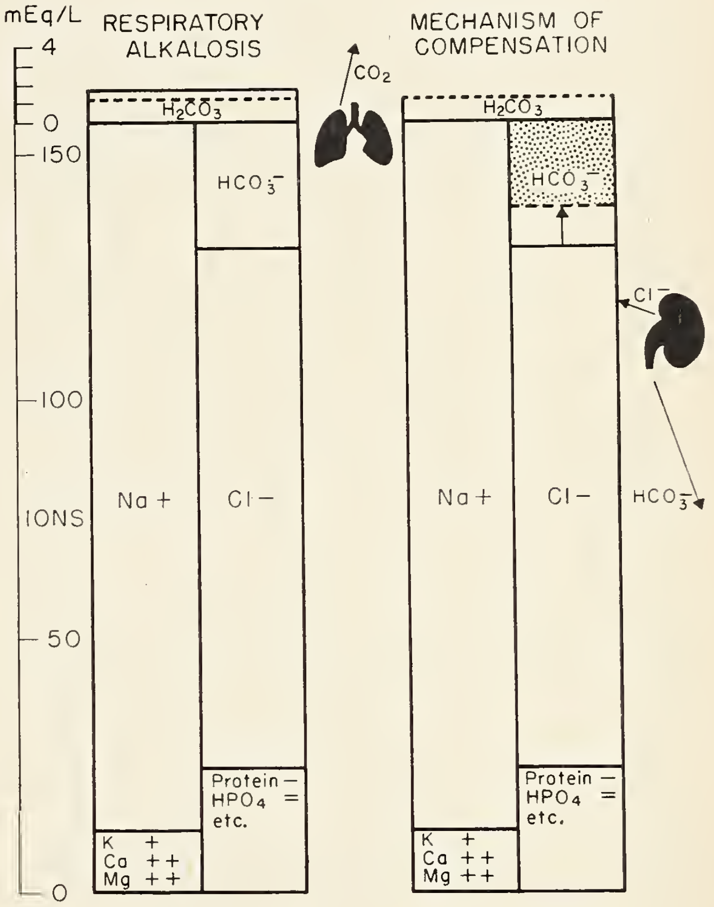
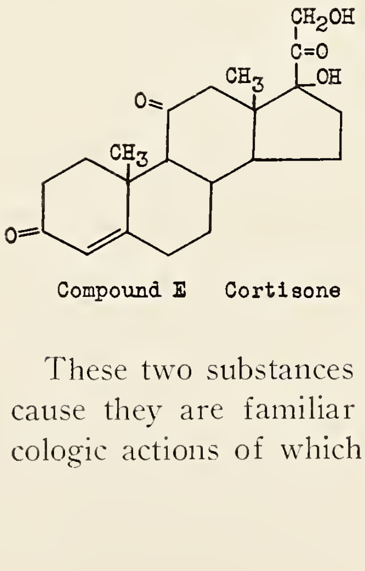
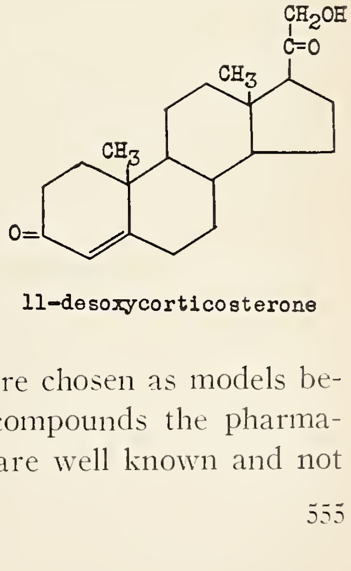


Fig. 10: Respiratory alkalosis. Left: Primary defect resulting from increased ventilation and loss of carbon dioxide resulting in decreased carbonic acid. Right: Compensation is achieved by excretion of bicarbonate and retention of chloride by the kidney.

The adrenal cortex: The adrenals produce a variety of hormones of which two groups, the glycocorticoids and the mineralocorticoids are illustrated below by their prototypes, cortisone and 11-desoxycorticosterone.



Compound E Cortisone



11-desoxycorticosterone

These two substances are chosen as models because they are familiar compounds the pharmacologic actions of which are well known and not because they are produced in the greatest amounts by the adrenal gland. As a matter of fact, desoxycorticosterone has been obtained in only very minute quantities from adrenals and this precise compound by itself may have little or no significance normally as an adrenal hormone. In general, the first group of substances are identified mainly with a metabolism of protein and carbohydrate, the second group being concerned with the handling of electrolytes. Nevertheless, the distinctness of these actions is by no means complete, and there is a good deal of overlap of functions. The typical effect, for example, of the adrenal hormones on sodium and potassium metabolism is to cause retention of sodium ion and increase excretion of potassium. This general phenomenon varies very widely between the substances and is much more pronounced in relationship to compounds like desoxycorticosterone. Nevertheless, the pronounced tendency toward sodium retention and potassium loss is also a familiar clinical phenomenon during treatment with cortisone and the frequent necessity for employing a low sodium diet with addition of excess of potassium during such treatment is now well recognized. The effects of these substances on the metabolism of water is complex. The accumulation of edema fluid during chronic administration of adrenal materials is significant and is frequently demonstrable clinically. Nevertheless, if one considers the effect of these hormones on the balance of water alone it is primarily one of diuresis.[7] Thus, the Robinson-Power-Kepler water test is used to diagnose adrenal insufficiency by demonstrating the inability of the individual to respond to a load of water.[11] These effects on both electrolyte metabolism and water balance are important not only because of the frequent use of these substances in present-day therapy but also because of the increase in adrenal activity which is known to accompany injury and disease. These specific problems with which they are believed to be associated will be discussed later particularly in connection with electrolyte problems of surgery. It is important to recognize also that the effects of adrenal hormones on salt and water are not exclusively related to the activity of the kidneys. Extrarenal influences on the distribution of sodium and potassium across cell membranes have been demonstrated in nephrectomized animals and are surely of importance in the shifts of electrolytes known to occur in disease. Thus, the effect of either adrenal treatment or adrenal stimulation is to cause a loss of potassium from the cell with a concomitant gain in intracellular sodium. The overall effect of this, which is to produce a metabolic alkalosis, will be discussed more fully in the sections dealing with potassium deficiencies.

What mobilizes these materials from the intact adrenal in the physiological adjustment of the healthy individual? The glycocorticoids are released from the adrenal in response to the pituitary corticotrophic hormone (ACTH). The pituitary, in turn, is stimulated by the hypothalamus and there is some evidence that the pituitary is also sensitive to the circulating levels of adrenal steroids and can respond by secreting more ACTH when there is a drop in plasma-steroid concentrations. Almost nothing is known, however, as to the mechanisms which control the production and release of substances specifically concerned with salt retention. The pituitary corticotrophic mechanism is apparently not involved in this as shown by the fact that ingestion of a diet designed to call forth the production of "salt-retaining adrenal hormones" does not result in evidence of increased corticotrophic production.[13] Many investigators feel that the adrenal in this respect is directly sensitive to the sodium-potassium ratio of the blood. It must be appreciated, furthermore, that we really do not know the chemical nature of the most important salt-retaining adrenal hormones. It was mentioned above that desoxycorticosterone was probably not a significant naturally occurring substance. Recent chemical extraction of adrenal glands by Reichstein in Switzerland and Mason in this country have resulted in the discovery of substances as yet chemically unidentified which are far more active than desoxycorticosterone on sodium retention in the rat.[9,10] The occurrence of such substances in human subjects are currently being investigated, and it would appear, therefore, that within a few years we should know much more about the true nature of the endocrine regulation of sodium and potassium metabolism.

Posterior pituitary: Finally, in this connection, consideration should be given to the posterior pituitary, a special adaptation in the vertebrate which, through control over the absorption of water in the distal tubule, is able to cause the excretion of the urine which is hypertonic with respect to the blood. The regulation of the secretion of this hormone is of particular interest since, as has been shown by Verney, there are in the ramifications of the circulation to the brain, receptors which are sensitive to tonicity of the blood.[12] The stimulation of these by hypertonic concentrations apparently stimulates the release of antidiuretic hormone which in turn affects retention of water by the kidneys. Except in the case of diabetes insipidus wherein the effect of the posterior pituitary is completely removed the significance of this gland in disease remains largely to be explored. Part of the difficulty lies in the fact that only biological methods of assay are available for this substance and there would be no question that the development of chemical methods of analysis would be a great stimulus to the study of this field. Nevertheless, with present methods it would appear that in certain diseases such as cirrhosis, excessive amounts of antidiuretic hormone are circulating. Similar observations have been made regarding postoperative surgical patients.

References

1. Baldwin, E.: Introduction to Comparative Biochemistry. University Press. Cambridge, 1940.
2. Bernard, C.: Leçons sur les phénomènes de la vie communs aux animaux et aux végètaux. Paris: J. B. Ballière et fils, 1878.
3. Darrow, D. C., and Yannet, H.: The changes in the distribution of body water accompanying increase and decrease in extracellular electrolyte. J. Clin. Investigation, 14:226, 1935.
4. Davenport, H. W.: The ABC of Acid-Base Chemistry. 3rd Edition. University of Chicago Press, 1953.
5. Edelman, I. S., Holly, H. B., Schloerb, M. D., Sheldon, D. B., Friis-Hansen, B.J., Stole, B., and Moore, F. D.: Further observations on total body water. I. Normal values throughout the life span. Surg., Gynec. & Obst., 95:1, 1952.
6. Gamble, J. L.: Companionship of water and electrolytes in the organization of body fluids. Lane Medical Lectures, Stanford University Press, 5:1, 1951.
7. Gaunt, R., and Birnie, J. H.: Hormones and Body Water. Springfield, Illinois: Charles C Thomas, 1951.
8. Macallum, A. B.: The paleochemistry of the body fluids and tissues. Physiol. Rev., 6:316, 1926.
9. Mattox, V. R., Mason, H. L., and Albert, A.: Isolation of a sodium-retaining substance from beef adrenal extract. Proc. Staff Meet. Mayo Clin., 28: 569, 1953.
10. Reichstein, T.: Data presented at International Congress of Rheumatic Diseases, Geneva, August, 1953.
11. Robinson, F. J., Power, M. H., and Kepler, E. J.: Two procedures to assist in the recognition and exclusion of Addison's disease. Proc. Staff Meet. Mayo Clin., 16:577, 1941.
12. Verney, E. C.: Absorption and excretion of water. The antidiuretic hormone. Lancet, 2:739, 1946.
13. Zimmermann, B., and Schoenbauer, M. M.: Studies on the Control of Salt-Regulating Adrenal Hormones. Surgical Forum. Philadelphia: W. B. Saunders Company, 1952.

EARLY TREATMENT INCREASES SURVIVAL CHANCES IN HODGKIN'S DISEASE

The importance of early diagnosis and treatment is clearly brought out by research figures based on irradiation therapy of 208 patients with Hodgkin's disease, according to Dr. Charles M. Nice, a radiologist, and K. Wilhelm Stenstrom, Ph.D., a radiation physicist, in a paper published in the May, 1954 issue of *Radiology*.

Their conclusions stem from research done at the University of Minnesota Medical School, department of radiology.

A series of 208 patients with proved Hodgkin's disease showed a five-year overall survival rate of 25 per cent. For 167 patients followed ten years or longer, the ten-year overall survival rate is 11 per cent.

"Clinical staging," the authors report, "is the most accurate aid in prognosis and is necessary in comparing series from various medical centers."

Their research conclusions were based on the clinical classification into three stages devised by M. V. Peters.

For twenty patients in Stage I, the five-year survival rate is 85 per cent; for thirteen of these, followed ten years or longer, the ten-year survival rate is 77 per cent.

In Stage II, there is 90 per cent survival for twenty patients followed five years and a 35 per cent survival rate for seventeen patients followed ten years.

"Thus, the difference between Stages I and II is shown in the ten-year period," the authors write.

In the much larger group comprising Stage III, representing those patients with disseminated disease, the five-year and ten-year survival rates are 10 and 3 per cent respectively.

General conclusions advanced, based on the research:

(1) In Stage I, the treatment of choice is either intensive irradiation or, possibly, surgical excision followed immediately by intensive irradiation.

(2) In Stage II, intensive irradiation is the treatment of choice.

(3) In Stage III, palliative irradiation to reduce tumor masses or relieve symptoms is indicated.

"It is in the latter group that the nitrogen mustards and triethylene melamine may serve as useful adjuncts," the authors conclude.

FLUID AND ELECTROLYTE BALANCE

II. Parenteral Fluid Therapy in Infants and Children

HOWARD G. WORTHEN, M.D., and RICHARD B. RAILE, M.D.
Minneapolis, Minnesota

IN THE preceding paper of this symposium, Flink and Zimmermann[9] discussed in some detail the fundamentals upon which a rational program of fluid and electrolyte therapy must be based. They have outlined and defined the terminology, physiology, and primary disturbances of body water and electrolytes. These basic considerations warrant careful study by the physician who intends to make use of the advances in therapy resulting from recent studies in this field.

The treatment of any patient who is in need of parenteral fluid therapy requires an understanding of the way a particular disease affects fluid balance and electrolyte composition. In addition, the treatment of infants and children necessitates a knowledge of the different requirements for water and electrolytes that result from differences in size, metabolic rate, and kidney function.

It is not possible to translate therapy as used in the adult directly to the child. Because of differences in size, the quantity of fluid that is correct for an adult is obviously too much for a small child; however, because the higher metabolic rate produces a relatively greater water requirement in the child, a quantity of fluid and electrolytes calculated from adult values, using body weight as a guide, will often be inadequate for the child. Therefore, correct therapy must consider not only the size, but also the relatively higher requirements of the infant or child in need of such therapy.

The quantity of fluid required may be calculated on the basis of weight if a different value in milliliters per kilogram or milliliters per pound of body weight is used for various age groups. A simpler method is to use the surface area of the body as a guide (i.e. cc/m^2), since surface area is closely correlated with metabolism and renal function. In this way requirements can be calculated without reference to size or age—the small child having the same requirement in milliliters per square meter of surface area as the adult. Customarily the surface area of the body is calculated by the use of the formula $M^2 = \sqrt[3]{(W)^2} \times 0.10$, in which M^2 = square meters surface area, $(W)^2$ = the square of the value of the weight in kilograms. However, for simplicity the values for surface area, weight and twenty-four-hour fluid requirement may be obtained by use of the convenient graph in Figure 1.

Water comprises a greater proportion of the total body weight in the infancy period than at any other time. In the infant, approximately 75 per cent of the total body weight is water;[12] this percentage decreases to the adult value of 60 to 65 per cent at about two years of age. The extra water of the infant is located in the interstitial compartment (Fig. 2). The large interstitial compartment functions as a buffer, to prevent the relatively large intake and excretion of water from producing drastic changes in plasma volume. Although this large volume would seem to protect the infant against plasma volume depletion, it is more than offset by the higher rate of water loss, and so offers very little protection.

The necessity for increased fluid intake and excretion renders the infant, who is deprived of water—or who loses water as a result of disease, much more liable to dehydration and circulatory collapse than the adult. Therefore, diseases leading to dehydration require more vigorous treatment in the young child than in the adult.

The rate of turnover of body water is relatively greater in the infant because the higher rate of metabolism demands a higher urine volume to excrete the metabolic waste products, and because the relatively larger surface area produces greater losses of water from insensible perspiration and sweat. An understanding of the amount of water and electrolytes necessary to replace the normal losses will serve as a basis for understanding the amounts necessary to treat dehydrated patients.

The quantity of these normal losses is expressed in terms of surface area, so it can be applied to patients of any size.

From the Department of Pediatrics, University of Minnesota, Minneapolis, Minnesota.

Routes of Fluid Loss

(a) *Loss as insensible perspiration*—via the skin and lungs: This loss is dependent upon temperature regulation and body surface area, not on fluid balance. It is essentially continuous, and varies with the patient's temperature, and with environmental temperature and humidity. Under basal conditions, this loss approximates 500 cc/m²/24 hrs.[14] The average patient on bed rest without fever loses approximately 600 cc/m²/24 hrs., as insensible perspiration.

(b) *Loss as sweat:* Sweat loss also depends largely upon temperature regulatory mechanisms and environmental conditions. This loss varies from 0 to 3 Liters/m²/24 hrs.,[1] depending upon environmental temperature and humidity.

(c) *Loss in stools:* Stool water normally averages 100 to 150 cc/m²/24 hrs. In diarrheal

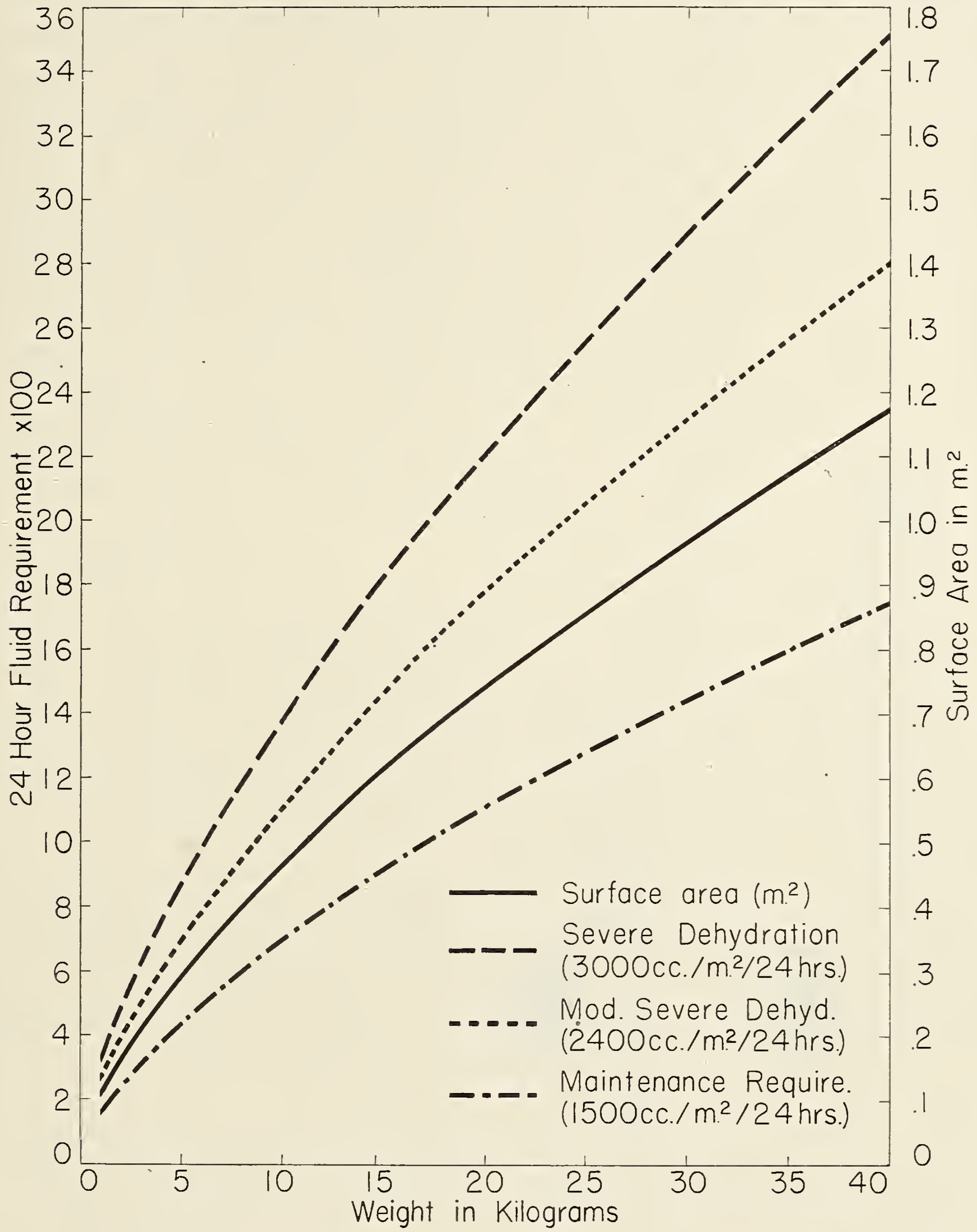


Fig. 1. Twenty-four-hour fluid requirements for three situations. The requirement in cc/24 hrs. is found at the intersection of the patient's weight and the line appropriate for the patient's state of hydration.

states the loss may be more than 1,000 cc/m²/24 hrs. In a patient not receiving oral fluids and without diarrhea the losses are usually minimal and can be ignored.

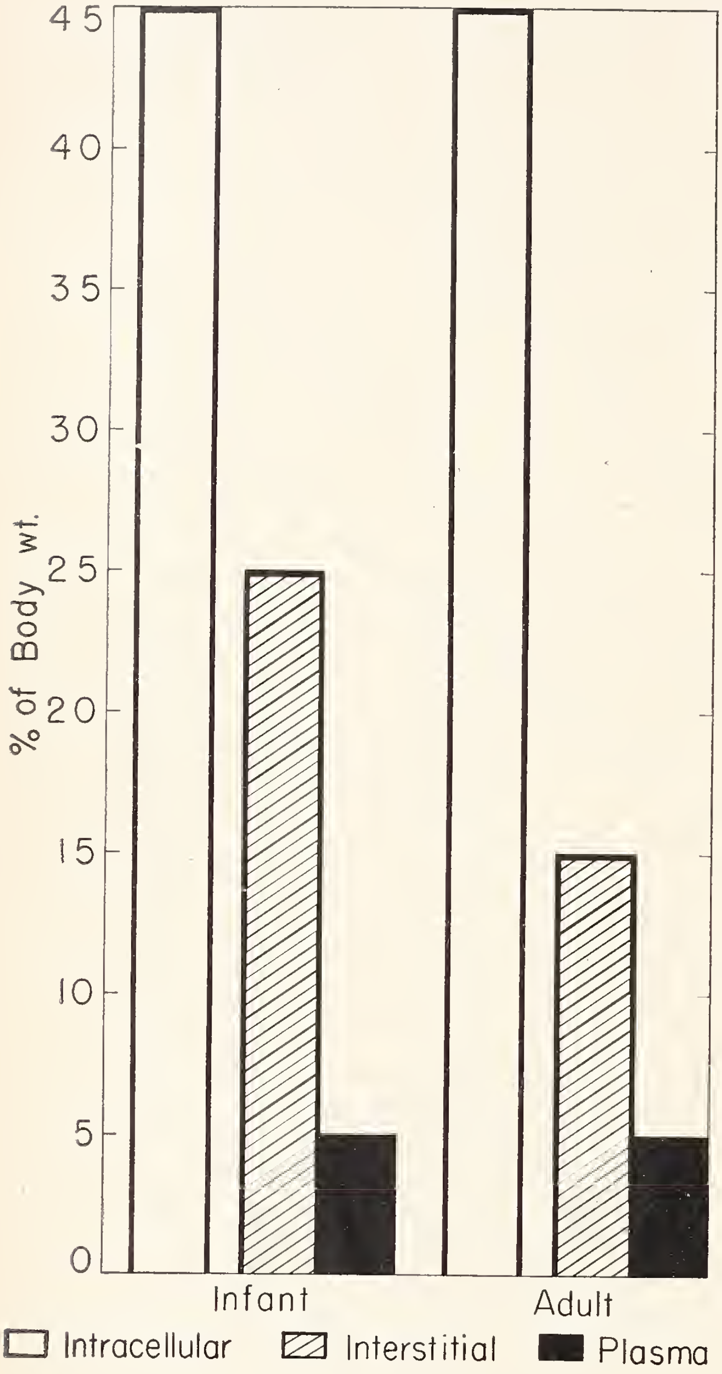


Fig. 2. Comparison of the volume of the fluid compartments in the infant and the adult, expressed as percentage of body weight.

(d) *Loss in urine:* The kidney is the most important regulator of water and electrolyte balance. The amount of urine excreted depends upon the concentrating ability of the kidney and the amount of solute requiring excretion. Minimum urine volume therefore occurs when concentration is maximal and solute load is minimal. In a healthy person, maximum concentration is approximately 1.4 m-osm/cc (Specific Gravity 1.030).[11] However, in a person under the stress of severe illness the maximum concentration may be only 0.4 m-osm/cc (Specific Gravity 1.012).[4]

The amount of solute that must be excreted depends upon the rate of catabolism of body tissues, especially protein, and the amount of exogenous solute furnished by the diet or parenteral fluids. The minimum solute excretion of 200 m-osm/m²/24 hrs. occurs when no food other than carbohydrate is given.

The least amount of glucose which will prevent ketosis and minimize protein breakdown is 75 gms/m²/24 hrs.[5,10] The maximum amount that can be given without causing glycosuria and adverse osmotic effects from hyperglycemia is approximately 300 gm./m²/24 hrs.[2] The minimum urine volume necessary to excrete 200 m-osm is 140 cc, if the patient can concentrate to a specific gravity of 1.040, and 500 cc if his concentrating ability is limited to a specific gravity of 1.012.

Sources of Water

In addition to the water administered, water is derived from catabolism of body tissue and from oxidation of food. When 5 per cent glucose is given as the only nutrient, in amounts of 75 to 300 gms./m²/24 hrs. the average amount of water derived from oxidation and catabolism will approximate 250cc/m²/24 hrs.[5,10]

Maintenance Water Requirement.—In a patient who is not dehydrated and who has no unusual losses, the minimum amount of water necessary to supply maintenance needs is equal to the water normally lost minus the water derived from oxidation and catabolism:

Insensible loss	600 cc/m²/24 hrs.
Stools	100-150
Urine	200-500
Minus the water of oxidation	250
	750-1000 cc/m²/24 hrs.

Most patients who require parenteral fluids have fever and move about in bed, and are therefore not under basal conditions. In such cases the insensible water loss and sweat loss are increased. An approximation of the water requirement for maintenance in a sick person would be 1500 cc/m²/24 hrs.

Limits of Electrolyte Tolerance

Parenteral fluid therapy with glucose solution only, is rarely indicated, since in almost any condition leading to a need for water, there exist electrolyte deficits also. Electrolyte deficits can be repaired more accurately if the physician understands the minimum electrolyte requirements and the maximum amounts that can be tolerated.

(a) *Sodium:* A healthy person is able to maintain sodium balance with as little as 10 meq./m^2/24 hrs., and can tolerate loads to 700 meq./m^2/24 hrs.[15] A person under the stress of acute illness tends to retain sodium, probably as a result of adrenocortical hormone action upon the kidney. Under such circumstances the maximum amount of sodium that can be tolerated may decrease to 250 meq./m^2/24 hrs.

The amount of sodium that is to be given a patient should therefore fall within the limits of 10 to 250 meq./m^2/24 hrs.[16] A reasonable amount for such purposes in the ill patient is from 50 to 150 meq./m^2/24 hrs.

(b) *Potassium:* The limits of tolerance for potassium are from 10 meq./m^2/24 hrs. to 250 meq./m^2/24 hrs.[8] Since "stress" results in increased potassium excretion, as well as decreased sodium excretion, it is readily conceivable that an ill patient, given sodium but not potassium, may rapidly develop potassium depletion. If oliguria is present, however, the excretion of potassium is limited, and even small doses of potassium may produce a toxic concentration in the blood. It is absolutely necessary that the adequacy of circulation and urine formation be established before solutions containing potassium are added to a treatment regime.

(c) *Chloride:* The limits of chloride tolerance are not well defined. The amount of chloride needed will depend on the acid-base balance of the patient. Unless severe alkalosis is present, about two-thirds of the anion should be given as chloride; the rest as lactate.

(d) Other electrolytes such as phosphorus, calcium, and magnesium are usually disregarded in short term maintenance regimes unless there is evidence of specific deficits.

Only in recent years has the need for using potassium as a vital part of parenteral fluid therapy been recognized. Formerly, it was believed that only the extracellular electrolytes were affected, and that losses from the intracellular space involved only water. Darrow,[7] using balance studies in patients with diarrhea, found that potassium was lost as well as sodium and chloride. He demonstrated that the mortality could be decreased by furnishing potassium in the repair solution. The need for potassium has been demonstrated also by Butler,[3] in diabetic acidosis, by Lowe et al[13] in surgical patients, and by Danowski et al[6] in pyloric stenosis.

The depletion of cellular potassium in conditions causing either acidosis or alkalosis is apparently due both to increased urinary excretion of potassium, and to replacement of cellular potassium by sodium. The entrance of sodium into the cell and the loss of potassium seems to be the primary abnormality; the augmented urine potassium excretion reflects the increased potassium available because of its extracellular location.

In infantile diarrhea, for example, acidosis is not due primarily to the sodium lost from the body, since the chloride loss is equal to the sodium loss, but to the shift of a portion of extracellular sodium into the cell, in exchange for potassium. The potassium is excreted, and there remains an excess of extracellular chloride. If solutions containing sodium but not potassium are given, more potassium is forced out of the cell, and a severe or even fatal potassium deficiency may ensue. This explains why sodium lactate and sodium biscarbonate solutions will correct the low serum bicarbonate, but often actually make the patient worse. When potassium is supplied along with sodium, cellular potassium is repleted. The extracellular sodium is restored by the sodium given plus the sodium leaving the cell when potassium enters.

Most of the illnesses leading to dehydration cause a loss of fluid with an electrolyte concentration either isotonic or hypotonic as compared to plasma. At the same time, water is being lost by the insensible routes. Therefore, the usual type of dehydration encountered in practice will be hypertonic dehydration. Because of the hypertonicity of the extracellular space, water is shifted from the intracellular space, leading to intracellular as well as extracellular dehydration. The situation, when treatment is begun, is, therefore, hypertonic dehydration of both the intracellular and the extracellular spaces, lowered intracellular potassium, and increased intracellular sodium. For this situation, a hypotonic solution containing both potassium and sodium is specific. The water given in excess of electrolytes restores hydration and corrects the hypertonicity, and the electrolytes furnished correct deficiencies in both the intracellular and extracellular compartments.

This type of solution is also well suited for furnishing the maintenance requirements of nondehydrated patients who must receive parenteral fluids. The free water supplied will replace that lost by the insensible routes, and the electrolytes replace those lost in the urine and sweat.

Actually, even hypotonic dehydration, if not too marked, will respond very well to a hypotonic electrolyte solution. The normal kidney can form urine which is dilute enough to excrete much of the administered water with only a small portion of the administered electrolyte. The electrolyte depletion is repaired by selective retention by the kidneys.

Approach to Fluid and Electrolyte Therapy

In treating patients with dehydration, the amount of fluid needed to restore normal hydration can be calculated by estimating the extent of the deficit that has occurred due to the illness and adding to this the maintenance requirement. If continued extra losses occur during treatment this amount is added to the total requirement.

Since it is very difficult to estimate with any degree of accuracy the amount of dehydration that exists in a given patient, and even more difficult to estimate the extent of electrolyte losses, advantage can be taken of the kidney's ability to retain needed water and electrolytes and excrete excesses. The patient is, therefore, supplied with water and electrolytes in excess of maintenance requirements in the form of hypotonic electrolyte solution. The correction then rests upon the wisdom of the kidney rather than upon the rough guess of the physician.

The amount of fluid needed in excess of the maintenance requirements will depend upon the extent of dehydration, whether mild, moderate, or severe. The type of fluid to be given will depend somewhat upon the specific disease state, but the vast majority of conditions can be satisfactorily treated with a hypotonic multiple electrolyte solution.

In a recent paper, Talbot et al[16] explained in detail the rationale for this treatment. The body has limits within which it can handle administered fluid and electrolyte. If the amount of fluid and electrolyte given falls within these limits, and circulatory adequacy and renal function are established, the kidney will act to conserve what is needed and allow the remainder to be excreted. It is wise in practice to use amounts nearer the lower limits rather than the higher limits of tolerance, thereby decreasing the excretory burden upon the kidney.

The administration of the hypotonic solution at the rate of 2400 cc/m^2/24 hrs., to a patient who is moderately dehydrated, will furnish enough water and electrolyte in excess of maintenance needs to correct a large portion of the deficit within the first twenty-four hours. A severely dehydrated patient may require 3000 cc/m^2/24 hrs.

In the Department of Pediatrics of the University of Minnesota Hospitals, an intravenous fluid therapy program, based upon the rationales described above, has been satisfactorily used for the past seven years.

The electrolyte solutions used are:

1. *Initial Hydrating Solutions*

(a) ⅓ isotonic saline, ⅔ 5 per cent Dextrose in distilled water.*

This solution is designed to furnish free water with electrolyte to restore hydration and urine output to a point where potassium-containing solutions can be safely used.

(b) ⅓ isotonic saline, ⅔ 5 per cent Dextrose in distilled water, with 26 ml. Molar sodium lactate per liter.**

This solution contains sodium and chloride in the same relative proportions as exist in plasma (1.5:1) and is used in patients with severe acidosis to avoid the acidotic effects of isotonic saline.

2. *Hypotonic Multiple Electrolyte (Polyionic) Solutions*

(a) Butler's solution (1950)[3]† containing in each liter: Sodium—55 meq., potassium—23 meq., chloride—45 meq., Lactate—26 m-moles, magnesium—5 meq., phosphate—12 m-moles, in 5 per cent dextrose and distilled water. (Electrolyte No. 2 in Travert® (Baxter) is similar to Butler's solution. However, 10 per cent invert sugar is employed in place of 5 per cent Dextrose).

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*Prepared by adding 150 cc. isotonic saline to 300 cc. of 5 per cent Dextrose in distilled water. This 450 cc. should be sufficient for the initial rehydration of most children.

**Prepared by mixing 150 cc. isotonic saline and 300 cc. 5 per cent Dextrose in distilled water, and adding 12 cc. of molar sodium lactate. (Each cubic centimeter of molar sodium lactate contains 1 meq. of sodium.)

†Prepared by adding one ampule of Butler's solution (1950) and 10 cc. of 3 per cent magnesium chloride to 1000 cc. of 5 per cent Dextrose in distilled water.

(b) ⅓ Darrow's solution, ⅔ 5 per cent Dextrose in distilled water, and 11 cc of 10 per cent potassium acetate per liter.‡

This solution will contain in each liter: sodium 41 meq., potassium 23 meq., and chloride 35 meq. (Darrow's solution is available under several trade names). This solution is used for infants whose renal immaturity might preclude phosphate administration. It may also be used as an alternative to Butler's solution in older children and adults.

These fluids can be satisfactorily used as the routine solutions in the treatment of patients requiring intravenous fluid therapy whether acidosis or alkalosis is present. There are, however, a few notable exceptions to their universal employment; adrenal insufficiency, where potassium containing solutions are contraindicated; severe hypotonic dehydration from any cause (i e., adrenal insufficiency, prior subcutaneous treatment with hypotonic solutions, and heat prostration); unusually high gastrointestinal losses as in ulcerative colitis, where electrolyte losses are greater than can be replaced by hypotonic solutions; renal insufficiency; and in the immediate postoperative period where the amount of water and electrolytes must be greatly restricted.

The determination of the amount of fluid needed is facilitated by the use of a graph, such as Figure 1, which eliminates the usual steps in the calculation, and allows the fluid needed to be determined directly from the weight. For example, a baby weighing 10 kg (22 lbs.) who is moderately dehydrated will receive 1100 cc. of fluids in twenty-four hours (0.47 m^2x2400 cc.).

The initial hydrating solution of ⅓ isotonic saline may be given at a rapid rate for a short period of time to establish the adequacy of renal function before the polyionic solution is started. It may be given at a rate of 8 cc/m^2/minute for forty-five minutes following which, if the patient has not voided, the rate is decreased to 2 cc/m^2/minute until voiding occurs.

If evidence of circulatory collapse is present or appears imminent, blood or plasma should be used at the beginning of therapy. Amounts of blood and plasma given are not included as a part of the total fluid to be administered.

After the dehydration and electrolyte deficits have been corrected, and if no continued extra losses occur, the rate of fluid administration may be decreased to a value nearer the maintenance volume of 1500 cc/m^2/24 hrs. for the duration of the intravenous fluid therapy.

Routes of Administration

Parenteral fluids are usually given either intravenously or subcutaneously. However, the use of subcutaneous fluids should be limited to short term therapy of non-dehydrated or slightly dehydrated children. The most serious consequence of dehydration is circulatory collapse, and only by placing the repair solution directly into the vascular compartment can the physician be certain that the fluids are immediately available to begin rehydration and prevent shock.

The absorption of subcutaneous fluids is too uncertain to depend upon when the patient is dehydrated. Patients treated by clysis will sometimes go into shock with enough fluid to have prevented the shock still pooled in the subcutaneous tissues.

If circulatory collapse is present or imminent when the patient is seen, intravenous therapy is mandatory, since blood or plasma must be given immediately to restore cardiac and renal function.

Subcutaneous fluids are suitable for a child with mild dehydration, if the abnormal loss of fluid has already stopped. The patient can often be sustained until he is able to tolerate oral fluids. Solutions given by clysis should have an electrolyte concentration isotonic with plasma. Hyptomic fluids may actually make the patient worse, at least temporarily, by drawing electrolytes from the plasma before the fluids can be absorbed. *None of the solutions listed above should be given subcutaneously.*

Sites for Administering Intravenous Fluids

In small children or infants, the veins in the scalp are usually the most dependable. The hair must be shaved, and a small needle, either No. 22 or No. 23 should be used.

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‡Prepared by mixing 150 cc. Darrow's solution and 300 cc. 5 per cent Dextrose in distilled water, and adding 5 cc. of 10 per cent potassium acetate (each cc. of 10 per cent potassium acetate contains 1 meq. of potassium). If a large amount of solution is required, mix 500 cc. Darrow's solution and 1000 cc. 5 per cent Dextrose in distilled water, and add 17 cc. of 10 per cent potassium acetate.

The veins on the dorsum of the hand can also be used, although in obese infants, the support for the vein may be so lax that difficulty is encountered in trying to insert the needle.

The saphenous vein as it courses anterior to the medial malleolus is an excellent site, because the position of the vein is quite constant. This vein can be entered in children of almost any age, even if it is not visible.

In older children, the antecubital veins are used most frequently, but the saphenous vein or the veins on the dorsum of the hand can be substituted if necessary.

If the child is in shock and a vein cannot be readily located, a cut-down may be done immediately. The saphenous vein is usually used. The incision is made just anterior and superior to the medial malleolus, and the polyethylene tubing is inserted through a small nick in the vein.

It should be emphasized that the daily intravenous fluid requirement should always be given over a twenty-four-hour period, rather than intermittently, to avoid the danger of overloading the vascular compartment.

References

1. Adolph, E. F., and others: Physiology of Man in the Desert. New York: Interscience Publishers, Inc., 1947.
2. Astwood, E. B., Flynn, J. M., and Krayer, O.: Effect of continuous intravenous infusion of glucose in normal dogs. J. Clin. Investigation, 21:621, 1942.
3. Butler, A. M.: Diabetic coma. New England J. Med., 243:648, 1950.
4. Butler, A. M., Talbot, N. B., Burnett, C. H., Stanbury, J. B., Mac Lachlan, E. A.: Metabolic studies in diabetic coma. Tr. A. Am. Physicians, 60:102, 1947.
5. Butler, A. M., Talbot, N. B., Mac Lachlan, E. A., Appleton, J. E., and Linton, M. A.: Effect of testosterone proprionate on losses incident to inadequate dietary intake. J. Clin. Endocrinol., 5:327, 1945.
6. Danowski, T. S., Austin, A. C., Gow, R. C., Mateer, F. M., Weigand, F. A., Peters, J. H., and Greenman, L.: Electrolyte and nitrogen balance studies in infants following cessation of vomiting. Pediat., 5:57, 1950.
7. Darrow, D. C.: The retention of electrolyte during recovery from severe dehydration due to diarrhea. J. Pediat., 28:515, 1946.
8. Elkington, J. R., and Tarail, R.: Present status of potassium therapy. Am. J. Med., 9:200, 1950.
9. Flink, E. B., and Zimmerman, B.: Fluid and electrolyte balance: I. Basic considerations. Minnesota Med., 37:545-557, 1954.
10. Gamble, J. L.: Physiological information from studies on the life raft ration. Harvey Lectures, 1946-1947, pp. 247-273. Lancaster, Pa.: The Science Press Printing Company, 1947.
11. Gamble, J. L., and Butler, A.: Measurement of renal water requirement. Tr. A. Am. Physicians, 58:157, 1944.
12. Holt, L. E., Jr., and McIntosh, R.: Holt Pediatrics. 12th Ed. New York: Appleton-Century-Crafts, Inc., 1953.
13. Lowe, C. U., Rourke, M., Mac Lachlan, E. A., and Butler, A. M.: Use of parenteral potassium therapy in surgical patients. Pediat., 6:183, 1950.
14. Newburgh, L. H., and Johnson, N. W.: The insensible loss of water. Physiol. Rev., 22:1, 1942.
15. Stewart, J. D., and Rourke, G. M.: Effects of large intravenous infusions on body fluid. J. Clin. Investigation, 21:197, 1942.
16. Talbot, N. B., Crawford, J. D., and Butler, A. M.: Homeostatic limits to safe parenteral fluid therapy. New England J. Med., 248:1100, 1953.

FELLOWSHIPS IN HEART DISEASE RESEARCH

The Life Insurance Medical Research Fund announced in June, that it has this year awarded $878,000 in grants and fellowships to be devoted to heart disease research.

This year's allocation includes $734,000 as grants-in-aid for fifty-four research programs and $144,000 to support thirty-eight fellowships for young men and women in training as research workers. Studies under these grants will be conducted in forty-seven institutions in twenty-four states, the District of Columbia, Canada and Denmark.

The grants will enable scientists to study the causes of heart failure, high blood pressure, surgery of the heart and blood vessels, virus infections of the heart, hardening of the arteries, obesity, and other highly specialized basic research. As in the past, findings of these investigations will be reported in scientific journals for the widest possible distribution to other researchers and to physicians.

The Life Insurance Medical Research Fund, which receives contributions from more than 140 large and small U. S. and Canadian life insurance companies, is the pioneer agency supporting the scientific study of diseases of the heart and blood vessels. These diseases, now the nation's No. 1 health problem, cause over half of all deaths.

NON-TUBERCULOUS CAVITIES IN THE LUNG

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IT IS MY purpose to review the causes and some of the perhaps not so well known signs, symptoms and diagnostic criteria of certain non-tuberculous cavities in the lung, with illustrations from a selected group of cases. At the outset I would like to emphasize one very important point, very valuable, I believe, for the general practioner. For many years, it has been a good clinical rule that any patient with a fairly considerable amount of sputum negative for tubercle bacilli on many repeated examinations, especially if there was a cavity on x-ray examination, did not have pulmonary tuberculosis. This has been a good general rule but it has failed often enough to make it very necessary to be extremely cautious in the exclusion of tuberculosis as the cause of pulmonary cavity. This is all the more true today when chemotherapy may temporarily render the sputum negative.

I shall present instances of cavity formation in patients with Friedlander's bacillus pneumonia, coccidioidomycosis, actinomycosis, histoplasmosis, paragonimiasis, Hodgkin's Disease, cancer and tuberculosis masquerading as simple abscess of the lung.

Friedlander's Bacillus Pneumonia

Case 1. A man, aged fifty-seven, hospitalized on September 5, 1951, was desperately ill with high fever, mental confusion, and sputum containing the Friedlander's bacillus (Fig. 1). Physical examination showed dullness and absence of breath sounds over the left upper lobe.

He was treated with streptomycin 4 gm daily for four weeks and then 2 gm daily for four more weeks. On November 8, 1951, the left upper lobe was removed because it had become an abscess.

Comment.—This Friedlander's bacillus *(Klebsiella pneumoniae* or *b. mucosus capsulatus)* is a non-motile, Gram-negative, non-spore-bearing organism. It is found in the respiratory passages in 2 to 25 per cent of normal persons and also in the intestinal tract of both infants and adults. It causes slightly over 1 per cent of clinical pneumonias. Jaffe[7] classified 198 Friedlander bacillus infections: 79 per cent were about equally divided between the genito-urinary tract, the gastro-intestinal tract and the liver and bile tracts; 12½ per cent involved the lungs and the respiratory tract; 5 per cent the skin and appendages, and 3½ per cent the female internal genitalia and the vagina.

Pulmonary Friedlander infections may be divided into four groups: (1) acute, (2) chronic, (3) focal, and (4) septicemic. There is a marked predilection for the elderly, the alcoholic and the diabetic. The upper lobes were affected in 80 per cent of the cases where the lungs were involved and more than one lobe was involved in 30 per cent. An abscess develops in the lung in 60 per cent and an empyema in 23.4 per cent of the cases. Characteristically the thick viscid sputum blocks the bronchi and interferes with ventilation so that the breath sounds over the involved areas are suppressed. Persistent thin-walled cavities with metastatic lesions elsewhere in the lungs simulates chronic pulmonary tuberculosis but the sputum is consistently negative for tubercle bacilli. Blood cultures are positive in more than half the cases of Friedlander's bacillus pneumonia.

The course of the disease roughly may be divided into two stages: first, the acute stage in which death may come quickly; the second, resembling very closely pulmonary tuberculosis with chronic course due to varying amounts of pulmonary fibrosis, suppurative pneumonitis, cavitation, bronchiectasis and perhaps empyema. One-sixth of the patients with pulmonary involvement recovery satisfactorily; two-sixths develop chronic suppurative pneumonitis; three-sixths die.

Coccidioidomycosis

Case 2 is presented because it illustrates the persistence of the organism, *coccidioides immitis,* in the body after approximately eight years without symptoms. This patient has shown no pulmonary involvement at any time. Figure 2, of another patient, shows a typical residual cavity from primary coccidioidomycosis. While a soli-

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tary thin-walled cavity with little reaction around it is characteristic and highly suggestive of a cyst, multiple coccidioidal cavities do occur and the cavities are by no means all thin-walled.

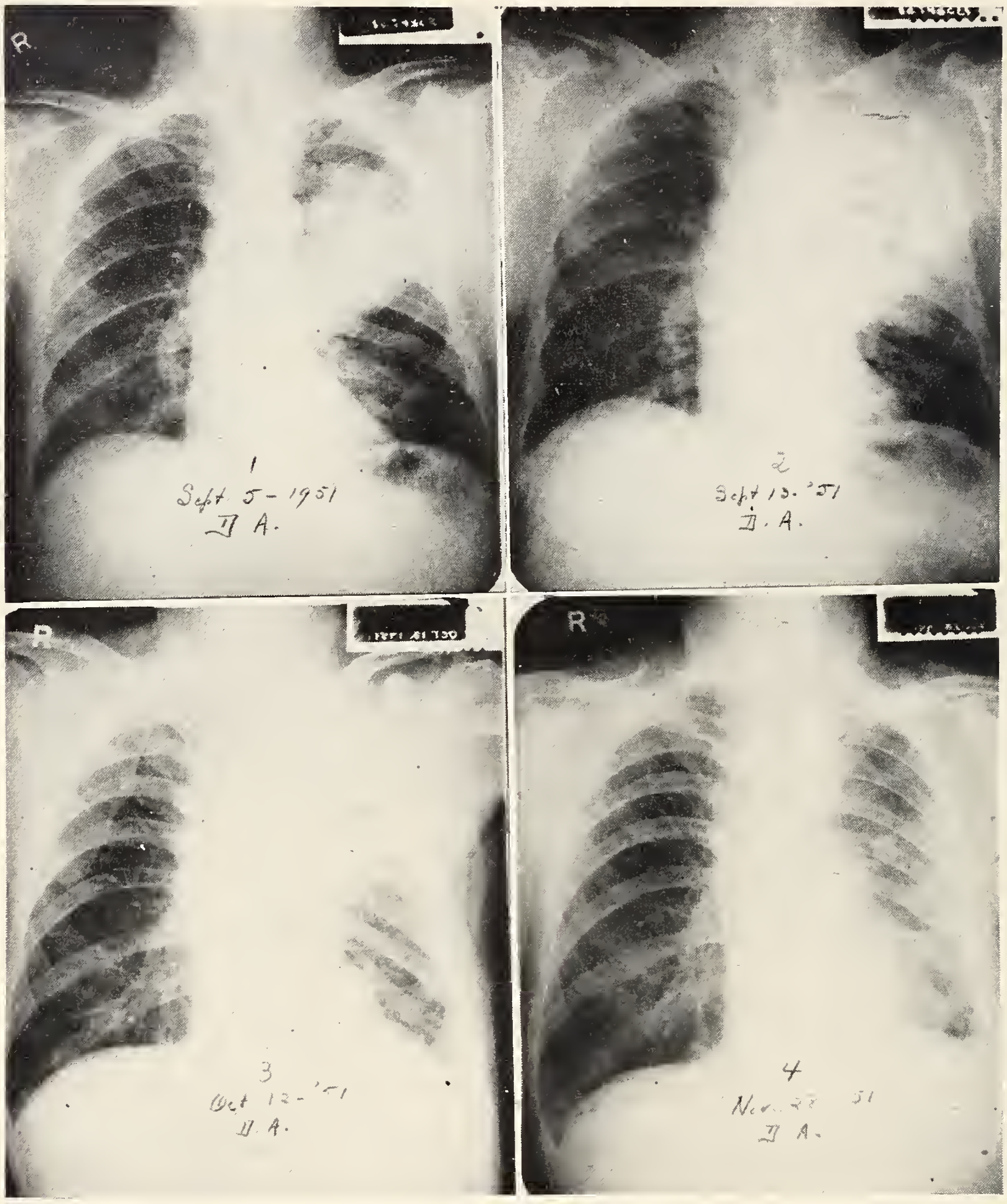

Fig. 1. Friedlander's bacillus pneumonia, abscess left upper lobe. Man aged fifty-seven years.

Case 2. E. W., a white man, aged fifty-six in 1953. In the early fall of 1940, two and a half months after arrival in California where he worked in the San Joaquin Valley, he became ill with what his doctors called "Valley Fever." He stayed in bed at home two weeks. One week after returning to work, he developed an abscess on the middle finger of his left hand and had to quit work. He returned to Colorado.

He was admitted to Colorado General hospital, April, 1941, with six abscesses, and bone involvement of the left middle finger, right olecranon, right acromion process and sternum; one abscess of the left hip with involvement of the ischium, one abscess of the left groin and one soft tissue abscess of the left forearm. During the following year he had many other soft tissue abscesses without additional bone involvement. *C. immitis* was recovered in pure culture many times from these abscesses. At no time have X-rays of the chest shown any lung or mediastinal glandular involvement.

He was in Colorado General Hospital about one year; was treated with increasing doses of coccidioidin at first subcutaneously and later intravenously and made a remarkable recovery.

He resumed regular work in January, 1945, and has since been well until October, 1952, when he bruised the skin on the right elbow. A sore developed from which *C. immitis* was recovered in pure culture. This healed in a few weeks and no further symptoms have developed.

Comment.—Coccidioidomycosis is a fungus infection resembling closely in many ways pulmonary tuberculosis. The primary form of the disease is an acute, benign, self-limited respiratory infection. According to Winn,[19] in this primary stage pleural pain may be extremely severe suggesting occasionally a coronary occlusion, gall stones or renal colic or a broken rib. The headache sometimes is very severe and may suggest the headache of acute poliomyelitis or tumor of the brain. Toxic erythemas resemble measles and scarlet fever. The pain in the joints and in the limbs may suggest acute rheumatic fever. The typical rashes of erythema multiforme or erythema nodosum are highly suggestive, especially when they occur in recent visitors to the endemic area of California. The erythema nodosum may be used as an index of the incidence of the disease. Five per cent of newly arrived persons in the endemic area who become infected and about 20 per cent of the patients who have proven coccidioidomycosis develop erythema nodosum.

The secondary stage of the disease, coccidioidal granuloma, is a chronic, progressive, disseminated disease with a 50 per cent mortality. When dissemination takes place it is almost always early in about 1 per cent of patients clinically diagnosed with this infection and in about one in 500 of all those infected. During the first week of the infection 50 per cent of the cases have a positive precipitin test, 15 per cent have a positive skin test and only 10 per cent have a positive complement fixation test. Cases with erythema nodosum react very strongly to the skin test. No case of fatal dissemination has been reported from the residual cavity. Smith[15] says: "None of our military coccidioidal patients, of whom we had records of thousands, has ever been reported to us as having undergone a post-war dissemination."

A cavity may develop after completely inapparent infection. Few cavities produce sufficient symptoms to warrant the patient seeking medical advice. The most common symptom of cavity is hemoptysis. A cavity may appear transiently during the acute infection and then disappear or one may develop months after the acute infection is over and thus be missed. For these reasons the true incidence of cavity is difficult to determine.

Criteria for proof of coccidioidal nature of cavity: (1) recovery of fungus, (2) positive serology, precipitins during acute phase and complete fixation of complement in at least first 1:2 dilution of serum, (3) a positive coccidioidin skin test with a negative tuberculin test.

A small number of double infections, coccidioidomycosis and tuberculosis, are recorded. Coccidioidomycosis rarely activates quiescent tuberculosis. Tuberculous patients hospitalized in endemic areas have acquired coccidioidomycosis from the ever-present dust containing chlamydospores (Smith).[15]

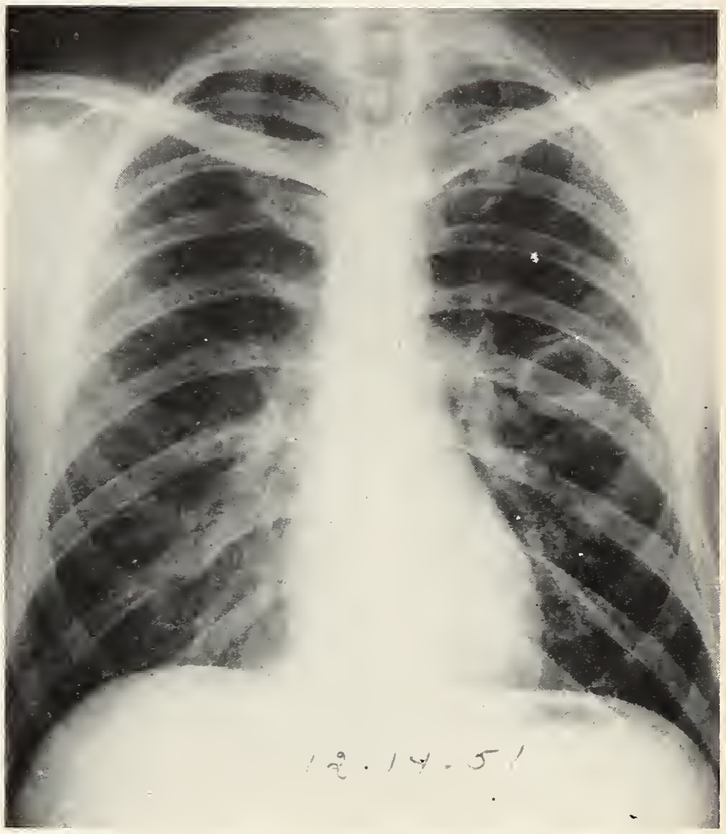

Fig. 2. Coccidioidomycosis. Residual cavity.

The pulmonary segment or lobe most frequently involved with a primary coccidioidal cavity is not known since many cases have not had special x-ray examinations to identify accurately affected lobes. Roughly, in the films 70 per cent of the cavities are in the upper lung fields and 30 per cent in the lower. Spontaneous hydro-pneumothorax during the initial infection even without demonstration of cavity has been reported. In recording the history of the patient, residence two to three months preceding onset of symptoms should be carefully noted. Since 25 per cent of cavity cases may be serologically negative and the skin test give only "a faint blush," a positive diagnosis can be made only by recovery of the fungus. The sedimentation rate may be normal in 75 per cent of coccidioidal cavity cases but, in contrast, it is infrequently normal in tuberculous cavity cases. Surgical procedures on the lung do not stir up the infection even to the extent of increasing the titer of the complement fixation reaction. As in tuberculosis, terminal cases of histoplasmosis, coccidioidomycosis and blastomycosis may become anergic and give negative skin tests, but the complement fixation test rises in these cases (Smith).[16] Only patients with excessive sensitivity to coccidioidin, positive reactions to 1:10,000 dilution, show cross reactions with histoplasmin or blastomycin. Persons with marked reactions to histoplasmin may give cross reactions with blastomycin.

Winn[19] in a study of ninety-two coccidioidal cavities says 25 per cent closed spontaneously, 10 per cent persisted without further complications and 5 per cent filled and became "solid" lesions radiologically. The balance required surgery.

Racial predilection in this disease is so conspicuous that it is worthwhile recording: Dissemination occurs in one in 500 infected Caucasians, three and one-half times as frequently in Mexicans, fourteen times as frequently in Negroes and 180 times as frequently in Filipinos (Jillson).[8]

There is no evidence of person to person transmission. The return to normal of the "sed rate" is a dependable clinical sign of cure.

The coccidioidin skin test is a valuable and at the same time a very safe test. It differs from the tuberculin test in certain important particulars. Its virtues and significance may be summarized as follows:

1. It does not sensitize.

2. Unlike tuberculin, it does not cause focal reactions.

3. Unlike tuberculin, it does not stir up quiescent lesions.

4. It does not interfere with serologic tests.

5. As with the tuberculin test, conversion of the coccidioidin test from negative to positive is highly significant. In doing the tests one must carefully avoid using tuberculin contaminated syringes. Syringes used for tuberculin tests should be reserved solely for that purpose!

6. The tuberculin test is helpful in differential diagnosis.

7. Simultaneous skin tests should be done for blastomycosis, histoplasmosis and coccidioidomycosis; if all react positively, the strongest test is the significant one.

8. After primary infection there is remarkably solid immunity to exogenous reinfection. The acquired immunity after naturally acquired primary infection with tuberculosis or BCG vaccination is only relative.

Indications for surgery in coccidioidomycosis include: (1) a giant cavity, a block cavity or an infected cavity, (2) rupture of a cavity into the pleural space with pneumothorax or empyema, (3) an unexpanded lung after rupture, (4) repeated severe hemoptysis, (5) formation of a coccidioima, (6) some surgeons recommend that all residuals should be removed surgically regardless of activity. J. A. Weinberg advises delaying surgery if the complement fixation test is high.

We are so disappointingly aware of the great tendency of primary tuberculosis to progress or to become latent and later to relapse that it is hard to appreciate the solid immunity conferred by uncomplicated primary coccidioidomycosis. All clinical experience is against exacerbation or late dissemination from residual lesions. However, it is still remotely possible that dissemination *may* take place from a primary focus since: (1) patients have been observed with coccidioidal meningitis and an associated old focalized lesion, (2) patients dead of disseminated coccidioidomycosis may have fresh lesions and also older focalized lesions, (3) lymph node, bronchial or blood vessel ulceration furnish the opportunity for lymphogenous, bronchogenic or hematogenous spread, (4) organisms remain viable and can be cultured from these early lesions even after fifteen years, (5) Puckett[11] has found the mycelial form in a majority of resected coccidioidal lesions both solid and cavitary, (6) Forbus[3a] says as long as the primary lesion remains, the danger of dissemination is possible. Winn[19] has never seen a fatal dissemination from a residual pulmonary cavity. It should be emphasized again that early infection and subsidence of this early infection evidently produces a strong immunity both to exogenous reinfection and to endogenous exacerbation and dissemination.

Craddock[3] has itemized the general features of the mycoses and some associated conditions as follows (with modifications and additions):

1. Calcification of pulmonary lesions is common in tuberculosis, coccidioidomycosis, and histoplasmosis.

2. Cavity formation is common in tuberculosis, coccidioidomycosis and actinomycosis and infrequent in histoplasmosis.

3. Mediastinal glandular involvement is common in tuberculosis; in 16 per cent to 65 per cent of the cases of coccidioidomycosis; in actinomycosis and blastomycosis and histoplasmosis and, incidentally and conspicuously, in sarcoidosis.

4. Central nervous system involvement is par-

ticularly important and frequent in tuberculosis, torulosis, actinomycosis and nocardiosis and histoplasmosis.

5. Blastomycosis is prone to involve the skin, the lungs, the bones. It also should be noted that cases are appearing secondary to antibiotic chemotherapy.

6. Histoplasmosis may involve the mucous membranes anywhere, the mouth, the gastrointestinal tract. It also may involve the lungs, the adrenals, the central nervous system. Histoplasmosis is now recorded as one of the causes of Addison's Disease. It should be noted that histoplasmosis occasionally complicates or is complicated by tuberculosis, diabetes, leukemia or lymphoblastoma.

"Round lesions" discovered by the x-ray are found in about 2 per cent of persons in mass surveys. These round lesions may be divided as follows: tuberculomas 45 per cent; asymptomatic cancer of the lung 30 per cent; benign tumors 15 per cent; miscellaneous inflammatory lesions including mycoses 10 per cent.

Histoplasmosis

Space does not permit many illustrations so I omit an illustration of cavity formation in histoplasmosis. In any case, it would look almost like chronic fibro-cavitary pulmonary tuberculosis. I include a brief discussion of histoplasmosis, since Furcolow[4] and others have reported the occasional confusion of this fungus disease with tuberculosis. This confusion is not surprising since histoplasmosis (1) may produce a chronic pulmonary disease with cavity formation, (2) sometimes appears under both epidemic and non-epidemic conditions as a widespread miliary pulmonary disease resembling miliary tuberculosis, (3) not infrequently involves the larynx and has been erroneously diagnosed tuberculosis, sarcoidosis or coccidioidomycosis, (4) may involve the adrenals and produce the clinical picture of Addison's Disease, (5) shows calcification of old lesions and, (6) finally, may complicate tuberculosis.

In a valuable paper, Israel[6] described four principal types: (1) active primary histoplasmosis, (2) healed primary, (3) active disseminated and (4) chronic disseminated. Jillson[8] suggests a convenient classification: I. Pulmonary, both symptomatic and asymptomatic (non-epidemic and epidemic forms); II. Disseminated (non-fatal and fatal); and III. Localized (skin, penis, tongue and larynx). Furcolow and Grayston[4] have collected 116 cases of the epidemic form in a retrospective study of eleven epidemics. A strikingly consistent symptom complex accompanied the illness in all the epidemics. The x-ray typically showed numerous widely disseminated discrete infiltrations varying in size from small, finely granular lesions through snowflake appearance to discrete nodules. A high percentage eventually healed with miliary calcification.

Clinically, histoplasmosis usually begins with lesions in the mouth or the lungs. *Cavitation is uncommon.* Half of the reported cases were diagnosed at post mortem. The organisms have been isolated from 50 per cent of the cases. In fatal cases the gastrointestinal tract is almost always involved. Up to 1950 there were 123 cases reported in the literature and the lungs were involved in sixty-two. Up to August of 1952, 178 cases have been reported in the literature, and the proportion of those with lungs involved remains the same, at about 50 per cent. Cultures of the organisms in the sick patient are usually fairly reliable. Of 138 cases reported up to January, 1950, 20 per cent were in infants. Other diseases are not infrequently associated with or complicated by histoplasmosis infection. For instance, of 123 cases, concomitant tuberculosis was found in thirteen, Addison's Disease in six, Hodgkin's Disease or other lymphoblastomas in four, leukemia in two, diabetes in three, erysipelas in three. The incidence of concomitant malignant lymphomas (chiefly Hodgkin's Disease) in cryptococcosis and disseminated histoplasmosis is about 10 per cent (Jillson).[8]

The tuberculin test may become negative with the onset of Hodgkin's Disease and a similar depression of the serologic reactions for syphilis has been noted. It may be surmised that the malignant lymphomas predispose to cryptococcosis and histoplasmosis by alterations in the host's resistance.

Note that this disease, histoplasmosis, heavily involves the reticuloendothelial system, with a marked frequency of involvement of the orifices of the body, the gastrointestinal tract, the adrenals, the lungs and the lymph nodes and the central nervous system. Schulz reported involvement of the central nervous system in twelve of 120 cases. There is no evidence of person to person communication but dog to dog transmission is reported. Bone marrow cultures are sometimes helpful in detecting the organism. Mice should be injected intraperitoneally with the proper material. After three to six weeks the mouse is killed and the spleen and liver are streaked on media suitable for the growth of fungi. Puckett[12] has found the Periodic-Acid-Schiff Stain very valuable in identification of the organisms in resected lung lesions.

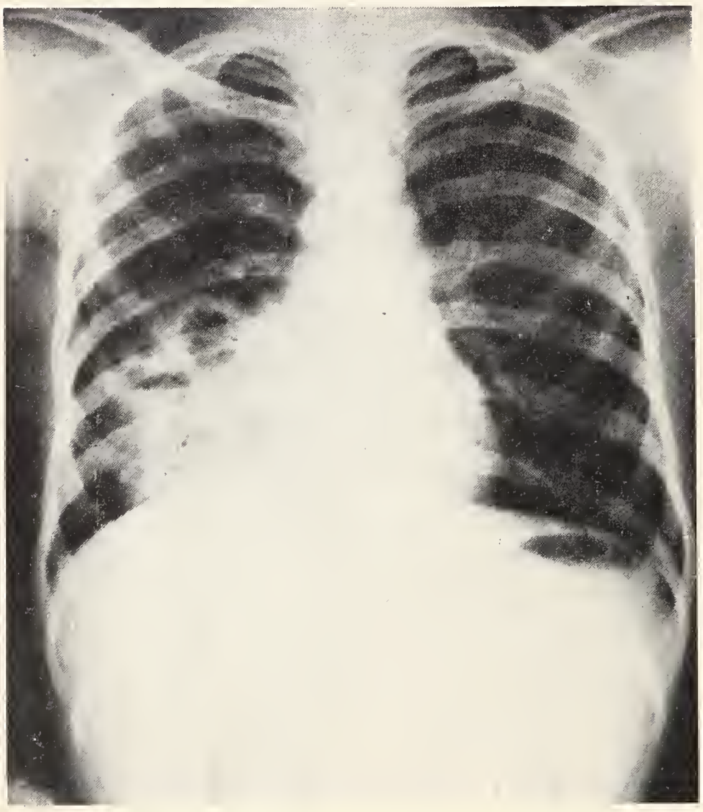

Fig. 3. Paragonimiasis. Cavity right lower lobe.

Christie[2] records his observations on the treatment of twelve cases of histoplasmosis with a fatal prognosis. These cases were treated with a preparation called ethyl-vanillate. This preparation is used orally as a 40 per cent solution in olive oil; dose given 0.5 gram every five to six days until 1.5 gram per kg. body weight per day is attained. Of twelve cases with fatal prognosis, five recovered and are alive and well after the use of this drug ethyl-vanillate.

Paragonimiasis

From the material of Roque, Ludwick and Bell,[13] Figure 3 shows a radiograph of an American soldier returned from Korea demonstrating a pulmonary cavity due to paragonimiasis. Since this patient had the highly suggestive combination of hemoptysis and a cavity in the lung, it is not surprising that it was originally thought that he had pulmonary tuberculosis. Because it is probable that pulmonary paragonimiasis will be seen, though rarely, among our American soldiers returned from Korea, it is important that we know of the existence of this disease and be on the alert not to confuse it with tuberculosis.

Paragonimiasis is called *endemic hemoptysis. Schistosomiasis* is *endemic hematuria.* Pathogenesis: Ingestion of a crab in the endemic area results in the liberation of the fluke from the cyst. The fluke migrates through the diaphragm and matures in cysts and in tunnels in the lungs. Diagnosis is dependent upon five criteria: (1) residence in the endemic area in Korea or Japan, (2) generally good condition of the patient, (3) a story of chronic or intermittent hemoptysis, (4) an x-ray showing lesions in the lungs, (5) the demonstration of the typical ova in wet preparations of the sputum or in some other tissue. The ovum is operculated (has a lid) and resembles the ovum of the fish tapeworm.

Important clinical causes of hemoptysis that the general practitioner should bear constantly in mind these days are: (1) tuberculosis, (2) bronchiectasis, (3) mitral stenosis, (4) infarction, (5) abscess of the lung, either simple or cancerous, (6) fungus infections of the lung, (7) paragonimiasis.

Actinomycosis

The vast majority of fungus infections of the lungs are caused by coccidioidomycosis, histoplasmosis and actinomycosis. "Valley Fever" and coccidioidal granuloma, as has been pointed out, represent only a very small part of those infected with *coccidioides immitis.* Cases of clinical histoplasmosis are being recognized more frequently but their absolute number is still small, and again inapparent infections, cases recognized only by a positive histoplasmin test and/or serologic tests, with or without few or many calcified pulmonary lesions, far outnumber those with manifest active disease. These inapparent infections of coccidioidomycosis and histoplasmosis have been identified by serologic reaction and skin tests. The situation with actinomycosis is different. Attempts to devise serologic reactions and skin tests have been unsuccessful. *Coccidioides immitis* and *histoplasma capsulatum* are widely distributed in nature, the former in endemic areas in California, Arizona and Texas, the latter mainly but not exclusively in the Mississippi Valley. Perhaps, because "lumpy jaw" of cattle was a common, as well as the first recognized, form of actinomycosis, it was originally thought (Bostroem, 1891) that actinomycosis results from traumatization with grass or straw carrying the infective agent and, therefore, the rural incidence must be greater than urban. Today we know better. The true actinomyces has not been found in nature "apart from a parasitic habitat," but is commonly present on human tooth surfaces, in the tartar on teeth, in tonsillar crypts and perhaps on mucous membranes. Aerobic actinomyces (Genus nocardia) is found in nature. Though Glover et al. have reported an instance of sensitivity to a suitable antigen of *N. asteroides* in human infection, reliable skin and serologic tests have not received general application. The number of inapparent actinomycotic infections is therefore conjectural, but must be comparatively few.

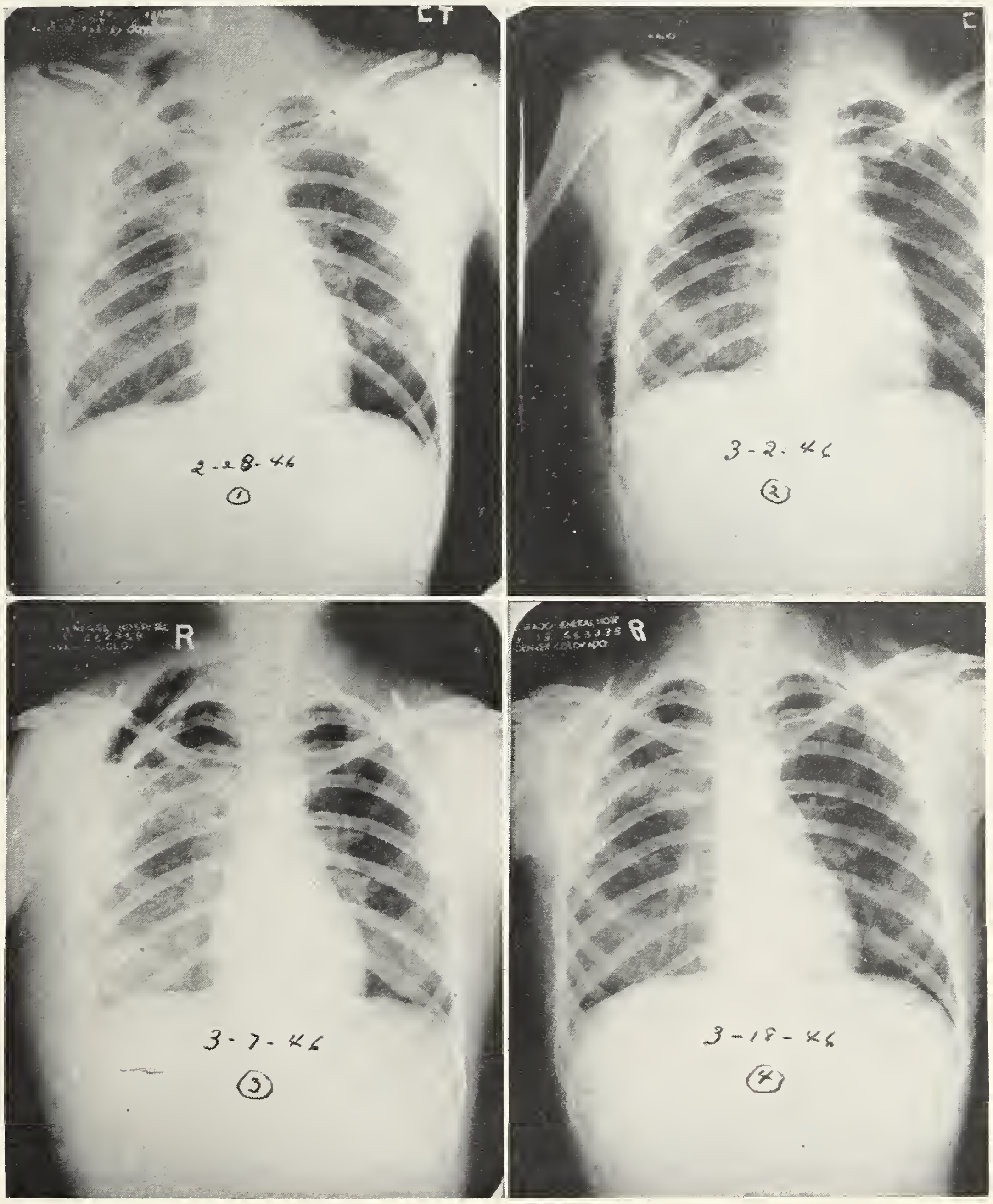

Fig. 4. Actinomycosis. Abscess left lung with broncho-pleuro-cutaneous fistula and large abscess chest wall. Note subcutaneous emphysema No. 2 and No. 3 and fluid level No. 3.

Case 3. R. G., a single Spanish-American girl, aged twenty-three, entered the hospital February 25, 1946, in a comatose state with high fever, frequent cough with bloody sputum, a tremendous fluctuating and crepitating abscess under the soft tissues of the back extending from the shoulder to the buttock, and abscesses on the left wrist and right elbow; bloody diarrhea and urinary incontinence.

On March 8, 1200 cc of pus were evacuated from the abscesses and *A. bovis* successfully cultured from this material. The patient was treated with large doses of penicillin and sulfadiazine and made a dramatic improvement. Note the subcutaneous emphysema in films 1, 2 and 3 and the fluid level extending beyond the chest wall

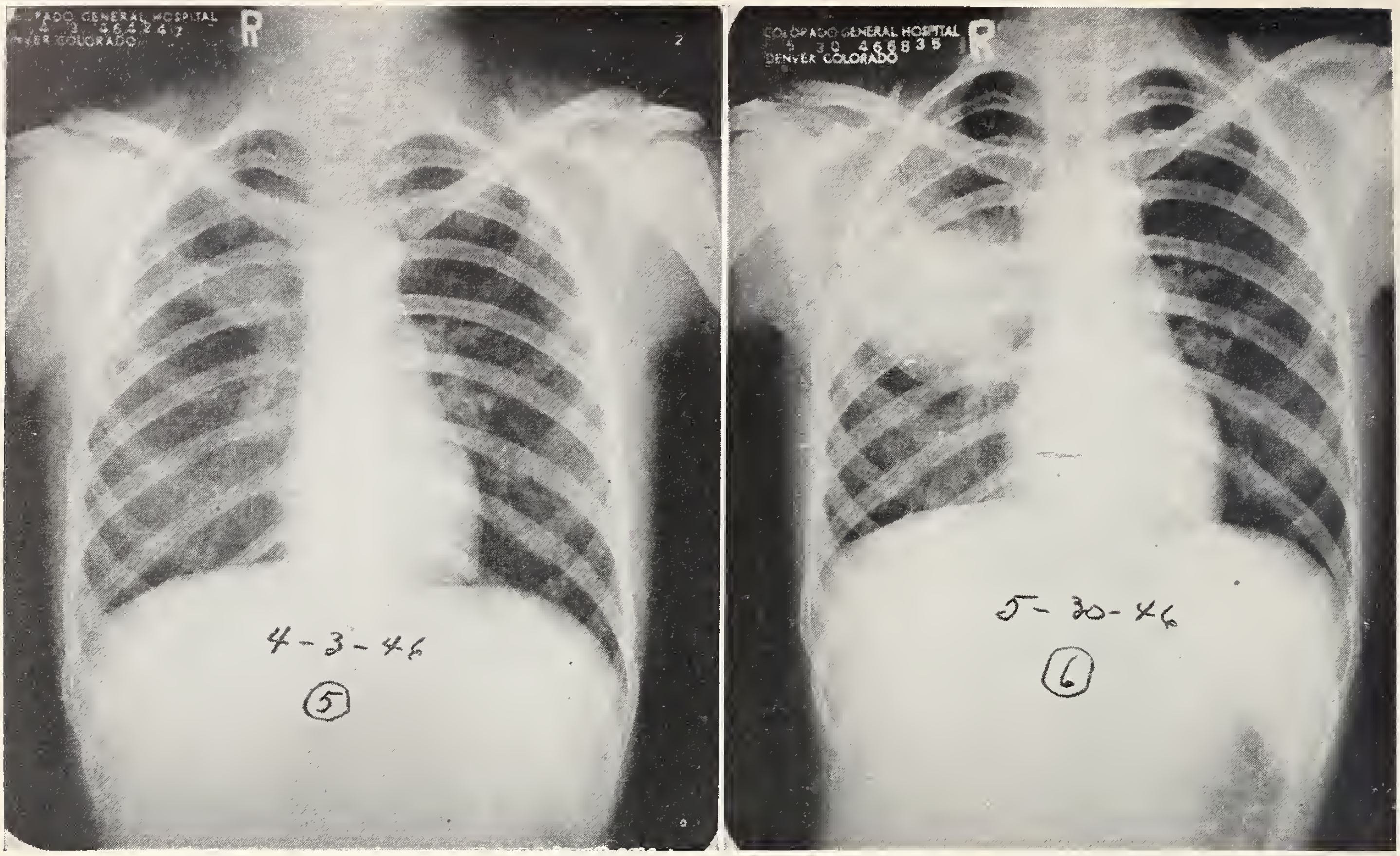

Fig. 5. Actinomycosis. Same case, showing improvement No. 5 and abscess No. 6 after hemorrhage.

in film No. 3, dated March 7, 1946, before surgery (Figs. 4 and 5).

She was so much improved by April 11, 1946, that, unwisely, I let her return home, under injunction, however, to continue the penicillin and sulfadiazine. She shortly began to hemorrhage very severely, returned to us and despite prompt surgery, died on August 16, 1946. It would have been much wiser to have done a lobectomy before letting her return home.

Comment.—A classification of the clinical cases of actinomycosis shows that: (1) cervico-facial, 56.8 per cent, (2) abdominal 22.3 per cent, (3) thoracic, 15 per cent, (4) elsewhere, 5.9 per cent. Note that the tongue is involved in 3 per cent of the cases. Pathogenesis: The disease originates (1) from the mouth by aspiration of infected material from around the teeth and gums (2) by extension of the disease up through the diaphragm into the lungs and (3) from metastasis. Characteristic pathology of the disease includes sinus formation and scar formation, all being of chronic nature.

Actinomycosis may be classified mainly from a clinical standpoint into two groups: Infections due to the anaerobic organism *a. bovis* which is Gram positive and is the agent in 90 per cent of the clinical infections of actinomycosis, and infections due to the aerobic organism called *nocardia asteroides* which is a partly acid-fast organism pathogenic for animals. Ordinary methods of concentration of sputum interfere with the growth of this nocardia organism. Neither form is communicable from person to person.

Kirby and McNaught[9] reported two cases of nocardiosis. Reviewing the literature they collected thirty-four cases of which the lungs were involved in twenty-five; the brain was involved in eleven; the diagnosis was made at post mortem in twenty-one; and thirty of the thirty-four patients died. The organism causes a necro-suppurative segmental pneumonitis with numerous small abscesses. Fortunately, some of the newer broad spectrum antibiotics are proving helpful. Formerly the main remedies consisted of potassium iodide and the combined use of penicillin and sulfadiazine. McVay and Sprunt[10] reported seven cases of actinomycosis treated successfully with aureomycin intravenously. It should be added that irradiation may be helpful for superficial lesions and surgery may be helpful for some of the local lesions.

McLean reported on the successful treatment of seven cases of oro-cervical actinomycosis with terramycin. They gave 750 to 2000 mg daily for fourteen to forty-two days, for an average total dose of 46 gm in an average period of twenty-four days. They said they saw no severe toxic reactions.

In the *J.A.M.A.* for May 9, 1953, there is a report on the treatment of another fungus disease, blastomycosis, with a preparation called stilbamidine. The first day 50 mg were given in 10 cc of 5 per cent dextrose. From the third to the twenty-ninth day 150 mg were given daily in 500 cc of dextrose solution. Also the patient at this time was put on a low protein and low purine diet. David Smith says stilbamidine is first choice in the treatment of pulmonary and systemic blastomycosis regardless of the immunological status of the patient. This drug is not harmless and must be used with caution. In a fairly high percentage of patients the drug selectively damages the sensory root of the fifth nerve.*

Hodgkin's Disease

Case 4. M. R., a woman, aged forty-three, suffered onset of the disease in 1946 with severe generalized pruritus. Diagnosis of Hodgkin's Disease by axillary node biopsy was made in September, 1947; four courses of irradiation therapy and three courses of nitrogen mustard; continuous fever, right pleural effusion, many thoracenteses; forty-three pound weight loss in the past year; non-productive cough. X-ray: enlarged mediastinal nodes and widespread parenchymal involvement both lungs with cavity right lower lobe (Fig. 6). Sputum negative for acid fast bacilli. Death, October 31, 1950. Anatomical diagnosis: Generalized Hodgkin's Disease. Cavity right lower lobe, hydrothorax. No pathologic evidence of tuberculosis was found.

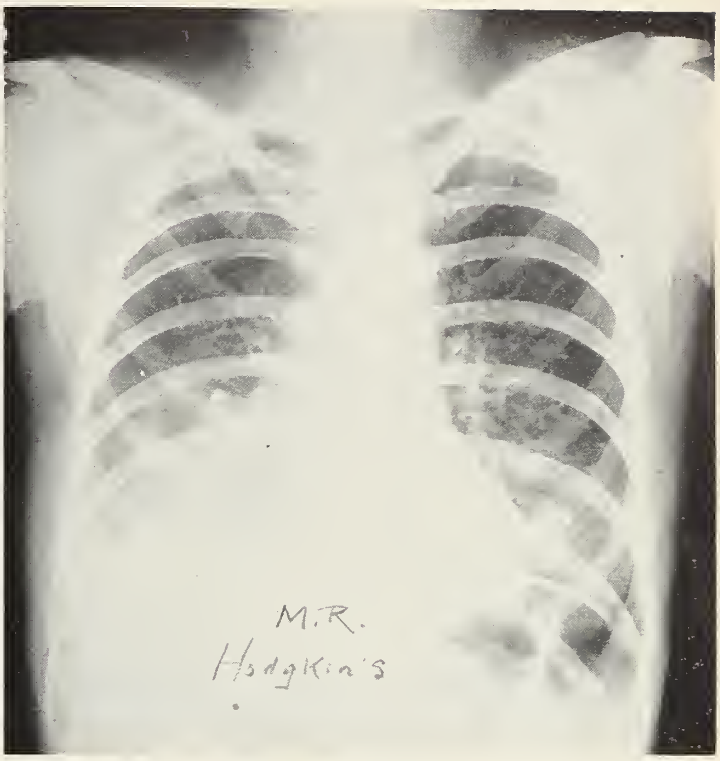

Fig. 6. Hodgkin's disease with cavity.

The lungs are involved in 15 to 40 per cent of all cases of Hodgkin's Disease. Pleural effusion is not infrequent. Granulomatous changes and thickening of the bronchial wall may lead to stenosis of the bronchus and obstructive pneumonitis which may suggest cancer, pneumonia or tuberculosis with cavity formation. Much later, nodules throughout the lungs may suggest metastasis from some malignancy originating outside the chest. Enlarged mediastinal nodes so commonly seen may suggest tuberculosis, lymphoma, erythema nodosum and sarcoidosis. Higginson and Grismer[5] reported a biopsy proven case of Hodgkin's Disease with a pedunculated endobronchial tumor.

Tuberculosis and Hodgkin's Disease.—Wallhauser[18] collected from the literature the post-mortem reports of 151 cases of Hodgkin's Disease. Active or healed tuberculosis was found in thirty-one or 20 per cent of those 151 cases. Clinically active tuberculosis was found in twelve or 4.5 per cent of 267 cases. Healed or active tuberculosis was found at post-mortem in other forms of lymphoma in 5.3 per cent; in cases of cancer in 14.6 per cent; at general post-mortem examination in 19.3 per cent; and in patients with Hodgkin's Disease 33.3 per cent. In Hodgkin's Disease the tuberculin test is frequently negative in the presence of a complicating tuberculosis. However, a strongly positive tuberculin test is good evidence of tuberculosis. The tuberculin test may become positive after effective x-ray treatment has been given.

Because I believe that one understands a disease best if he knows something of the history of the development of our present knowledge of it, or even our deficient knowledge, I summarize here some landmarks which reveal, if nothing more, the baffling nature of this disease.

1. In 1832, Thomas Hodgkin confused the disease with tuberculosis.
2. In 1926, Fox re-examined some of the tis-

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*In a recent report, Snapper and McVay call attention to three mistaken notions about blastomycosis. Briefly, (1) this disease is not such a rarity that it need not be considered seriously in differential diagnosis, (2) laboratory procedures for diagnosis are neither difficult nor expensive and, finally, (3) the prognosis is by no means hopeless since new drugs are available. They note the toxicity of stilbamidine and recommend the use of 2-hydroxystilbamidine which they found of therapeutic value and without toxic sequels. (Snapper, I. and McVay, L. V., Jr.: *Am. J. Med.*, 15:603 (Nov.), 1953.

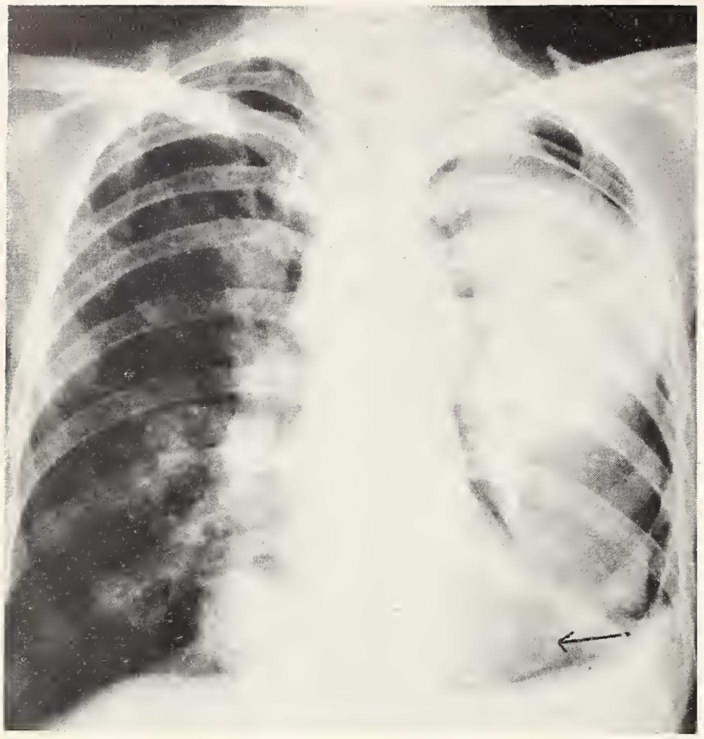

Fig. 7. Cavernous metastasis from seminoma of the testis.

sues from Hodgkin's original cases and found definite evidence of tuberculosis in some of them.

3. In 1898, Sternberg said that Hodgkin's Disease was a peculiar form of tuberculosis.

4. In 1928, James Ewing, in a memorable sentence dramatically emphasized the frequent association of these two diseases: "In New York where Hodgkin's Disease is common, tuberculosis follows Hodgkin's Disease like a shadow."

5. In 1931, L'Esperance said the disease was due to an avian tubercle bacillus.

6. In 1910, Fraenkel and Much found acid-fast rods in tissues of patients with Hodgkin's Disease.

7. In 1933, Wallhauser said that Sternberg had recently reaffirmed his theory that the cause of Hodgkin's Disease was tuberculosis.

8. In Hodgkin's Disease a factor is present not found in normal or cancer tissue which can be passed serially in fertile chicken eggs. The amniotic fluid from the eggs can interfere with the growth of the influenza virus in eggs.

Carcinomatous Abscess of the Lung

Abscess of the lung is found most frequently in association with primary carcinoma of the lung, especially those forms leading to bronchial obstruction and consequent suppurative pneumonitis. Rarely, a metastatic carcinoma of the lung may break down and form a cavity. Brock[1] says a cancerous cavity may be caused by necrosis of the primary growth itself, or it may arise in the obstructed and infected portion of the lung distal to the growth, or it may result from bronchial embolism or spill-over from the infected primary growth.

From the material of Salzman, Reid and Ogura,[14] the x-rays in Figure 7 illustrate the more rare condition: cavernous metastatic carcinoma of the lung. This is the picture of the lung of a man with pulmonary metastasis from seminoma of the testis. One year after removal of the testicular tumor, his chest film showed many solid and cavernous lesions in the lungs. Terminally, rupture of one of the cavities into the pleural space caused spontaneous pneumothorax. Five cases of spontaneous pneumothorax associated with metastatic carcinoma have been reported. According to Salzman et al., rapid growth of the metastatic pulmonary lesions with central necrosis, combined with infection and bronchial erosion, seem to be important factors in the pathogenesis of cavernous pulmonary metastatic carcinoma.

Abscess of the Lung due to Primary Cancer.—Strang and Simpson,[17] between 1946 and 1951 collected seventy cases of cancerous abscess of the lung from a British chest clinic. These cases were selected from a total of 1930 cases of bronchial carcinoma. Cancerous cavity was found in 3.6 per cent of 1930 cases of bronchial carcinoma. Cancerous cavity is found at post mortem in 12 per cent to 25 per cent of cases of cancer of the lung. According to Brock,[1] bronchial carcinoma was the cause of abscess of the lung in 13.8 per cent of 405 cases of lung abscess.

Of the seventy cases of cancerous lung abscess reported by Strang and Simpson, sixty-seven were in men and 70 per cent of the entire group were between the ages of fifty and sixty-five years. Squamous cell carcinoma was the most common cancer to cause cancer abscess of the lung; 30 per cent of forty cases of squamous cell carcinoma of the lung had abscess of the lung; 70 per cent of twenty-two cases of cancer with abscess were squamous cell carcinomas. Note that cancer of the middle lobe and, therefore, cancerous abscess of the middle lobe are rare. Sixty-three per cent of cancerous abscesses of the lung were in the upper lobes. Finally, one-third of lung abscesses in persons over forty-five years of age are due to cancer. Of Brock's

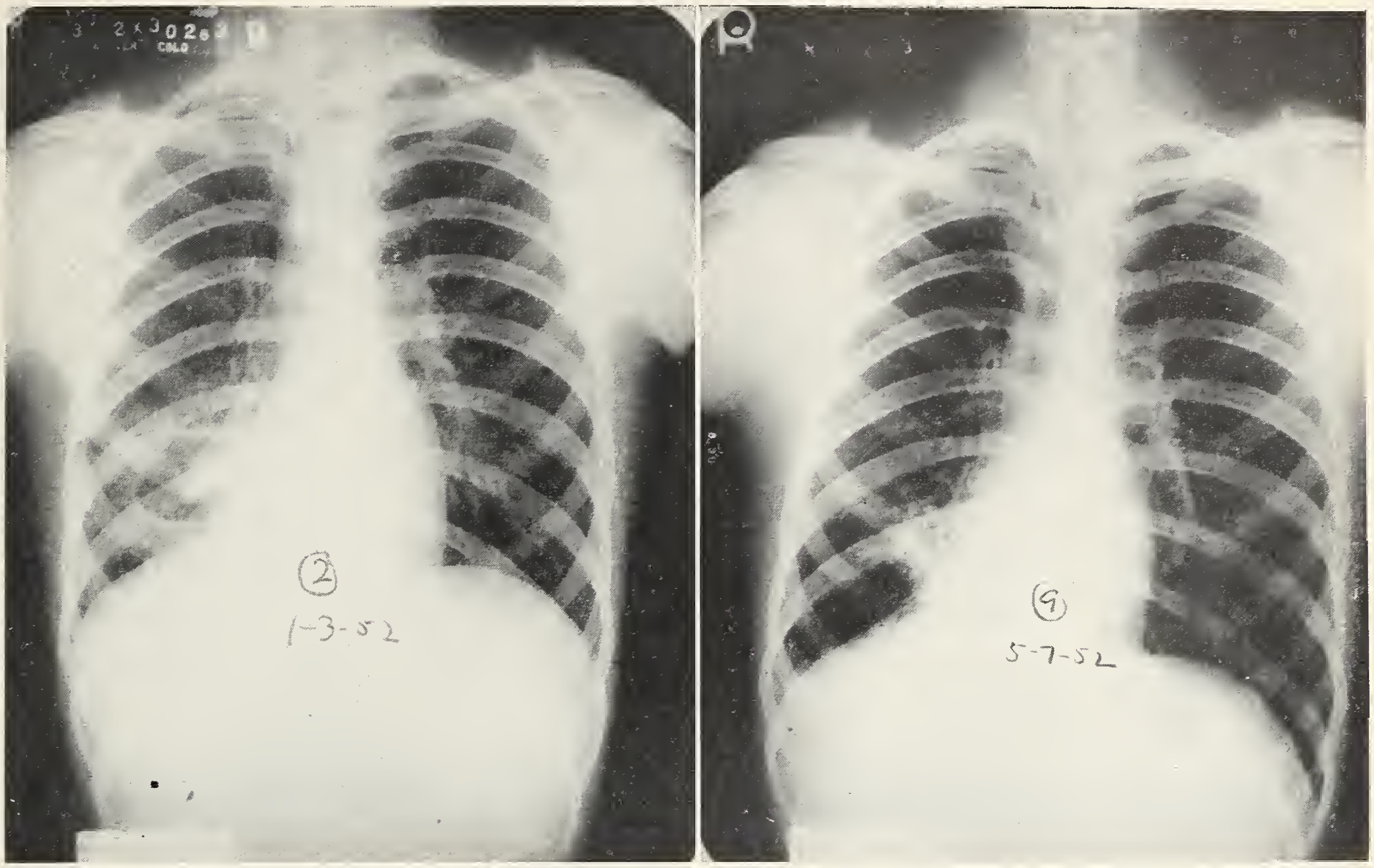

Fig. 8. Basal tuberculous cavity simulating non-tuberculous abscess of the lung.

eighty-three patients with abscess of the lung due to cancer, only four were in women, the other seventy-nine were in men. Therefore, age and sex are important influences. Eccentric cavitation is highly suggestive and rib erosion diagnostic.

Basal Tuberculous Cavity

Finally, a case is reported of a tuberculous cavity (Figure 8) in the lower lobe of the right lung masquerading as a non-tuberculous abscess of the lung.

Case 5.—R. J., an eighteen-year-old Spanish-American boy, entered the hospital's surgical service, December 28, 1951 with chief complaint of twenty-five pound loss in weight in the preceding year, increasingly productive cough and fatigability and pain in the right chest anteriorly. Since his pre-admission diagnosis was non-tuberculous abscess of the lung and since the "abscess" was in the lower lobe and the rest of the lung on both sides was apparently clear radiologically, he was forthwith bronchoscoped by an enterprising surgical resident without waiting for further sputum examination, which the next day, to the consternation of the bronchoscopist, showed innumerable tubercle bacilli.

Comment.—Tuberculous Cavity vs. Simple Abscess of the Lung: Several lessons can be drawn from this case. First, do not be too quick to accept the other fellow's diagnosis; second, if time permits, and it usually does, always examine the sputum carefully before bronchoscopy; third, since tuberculosis is ubiquitous and often unpredictable, all bronchoscopies should be done with as much care as if tuberculosis was already proven; fourth, tuberculous cavities do occur in the lower lobes and tuberculosis can be present even if repeated sputum examinations have been negative.

Conclusions

In general, in trying to make an etiologic diagnosis of cavity in the lung, the following should be borne in mind:

1. Record the presence or absence of putrid odor to sputum and the presence or absence of putrid odor during bronchoscopic examination. This means a putrid abscess.

2. Record presence or absence of dentures and "oral sepsis," e.g., pyorrhea. Although one must without fail inquire carefully for a history of aspiration of a *"foreign body,"* such history is relatively rare. Brock records only three foreign body abscesses in his 235 cases of abscess of the lung. Adults with upper dentures and children

are the most frequent "victims." Aspiration of *foreign material* notably vomitus at any time and infected material from bad teeth and gums during dental extraction or other oral surgery, are much more frequent causes of aspiration pneumonia and simple and putrid abscess of the lung. (See No. 6 below).

3. The early correction of oral sepsis in pregnant women and the correction of oral sepsis before any elective operation are desirable. Thus can one best avoid aspiration pneumonitis.

4. Record presence or absence of thrombophlebitis in legs. Bland or septic pulmonary infarction may lead to cavity formation.

5. Record presence or absence of heart murmurs. Mural thrombi dispose towards pulmonary infarction.

6. Record presence or absence in history of loss of consciousness including particularly injury, especially fracture of the jaw with bleeding (traffic accidents), operative procedures with or without general anesthesia, intoxications (morphine, barbiturates, alcohol, diabetic coma, carbon monoxide, illuminating gas, et cetera), epilepsy, immersions, labor and delivery with or without vomiting, foreign body aspiration, prolonged use of oil, sprays or laxative oils. With any of these, aspiration pneumonitis can easily occur.

7. The advisability of gastric lavage before emergency operative procedures and the necessity of prompt bronchoscopic aspiration of any person vomiting during anesthesia should be considered.

8. Cavities behind the heart or in the vertebral gutter may be and often are missed because they are unsuspected and lateral and oblique films of chest are not made. Precise lobar localization of cavities is helpful in diagnosis, e.g., a cavity in the middle lobe is not likely to be cancerous, but is much more likely tuberculous since the middle lobe bronchus is peculiarly vulnerable to compression from tuberculous glands as part of a primary complex. Tuberculous cavities are most apt to be found in the apical and posterior segments of the upper lobes and the superior segment of the lower lobes. Posture of the patient at time of aspiration plays an important part in determining where aspirated material will lodge. *Tomograms often reveal unsuspected cavities.*

9. Record residence or not in endemic areas for Coccidioidomycosis and Histoplasmosis.

10. Sputum and gastric washing cultures and blood cultures, all repeated in timely fashion, are fundamental to accurate diagnosis. Finally, never overlook the possibility of *tuberculosis!*

Acknowledgment

Case 1, Friedlander's bacillus pneumonia, is shown through the courtesy of Dr. John R. Durrance, Chief, Tuberculosis Section, Veterans Administration Hospital, Denver, Colorado.

References

1. Brock, R. C.: Lung abscess. Charles C Thomas, publisher, Springfield, Illinois, 1952.
2. Christie, Amos, Middleton, J. G., Peterson, J. C., and McVickar, D. L.: Treatment of disseminated histoplasmosis with ethyl vanillate. Pediatrics, 7:7-18, (Jan.) 1951.
3. Craddock, W. L.: Pulmonary traits of systemic mycoses. Mississippi Valley M. J., 75:28-37, (Jan.) 1953.
3a. Forbus, W. D., and Bestebreutje, A. M.: Coccidioidomycosis; a study of ninety-five cases of the disseminated type with special reference to the pathogenesis of the disease. Mil. Surg., 99:653-719, 1946.
4. Furcolow, M. L., and Grayston, J. T.: Occurrence of histoplasmosis in epidemics. Am. Rev. Tuberc., 68:307-320, (Sept.) 1953.
5. Higginson, J. F., and Grismer, J. T.: Obstructing intrabronchial Hodgkin's disease; case report. J. Thoracic Surg., 20:961-967, (Dec.) 1950.
6. Israel, H. L., et al.: Chronic disseminated histoplasmosis; an investigation of its relationship to sarcoidosis. Am. J. Med., 12:252-260, (Feb.) 1952.
7. Jaffe, S. A.: Extrapulmonary Klebsiella pneumoniae infections; analysis of literature; report of two unusual cases with recovery. J.A.M.A., 122:292-296, (May 29) 1943.
8. Jillson, O. F.: Mycology Medical Progress. New England J. Med., 249:523, (Oct. 1) 1953.
9. Kirby, W. M. M., and McNaught, J. B.: Actinomycosis due to nocardia asteroides; report of two cases. Arch. Int. Med., 75:578-591, (Nov.) 1946.
10. McVay, L. V., and Sprunt, D. H.: A long-term evaluation of aureomycin in the treatment of actinomycosis. Ann. Int. Med., 38:955-966, (May) 1953.
11. Puckett, T. F.: Hyphae of coccidioides immitis in tissues of the human host. (In press).
12. Puckett, T. F.: Pulmonary histoplasmosis; a study of twenty-two cases with identification of H. capsulatum in resected lesions. Am. Rev. Tuberc., 67:453-476, (April) 1953.
13. Roque, F. T.; Ludwick, R. W., and Bell, J. C.: Pulmonary paragonimiasis; a review with case reports from Korea and the Philippines. Ann. Int. Med., 38:1206-1221, (June) 1953.
14. Salzman, E.; Reid, J. H., and Ogura, G. I.: Cavernous metastatic pulmonary carcinoma; a report of two cases. Dis. of Chest, 23:678-685, (June) 1953.
14a. Schulz, D. M.: Histoplasmosis of the central nervous system. J.A.M.A., 151:549-551 (Feb. 14) 1953.
15. Smith, C. E.: (a) Diagnosis of pulmonary coccidioidal infections. California Med., 75:385-391, (Dec.) 1951. (b) Pathogenesis of coccidioidomycosis with special reference to pulmonary cavitation. Ann. Int. Med., 29:623 (Oct.) 1948.
16. Smith, D. T.: The diagnosis and therapy of mycotic infections. Bull. New York Acad. Med., 29:778, (Oct.) 1953.
17. Strang, C., and Simpson, J. A.: Carcinomatous abscess of the lung. Thorax, 8:11-28, (March) 1953.
18. Wallhauser, Andrew: Hodgkin's disease. Arch. Path., 16:522-562, (Oct.); 672-712, (Nov.) 1933.
19. Winn, W. A.: Pulmonary mycoses—coccidioidomycosis and pulmonary cavitation; study of 92 cases. Arch. Int. Med., 87:541-550, (April) 1951.

THE EFFECT OF PENICILLIN UPON THE VIRULENCE OF C. DIPHTHERIAE

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THE PRESENT study was undertaken to investigate the inhibiting effect of penicillin upon the virulence of *Corynebacterium diphtheriae*. The need for such an investigation arose from the widespread use of antibiotic therapy in upper respiratory infections. In many instances, cultures taken from patients with suspected clinical diphtheria and under antibiotic therapy failed to show *C. diphtheriae*. The purpose of the study was to record the effect of antibiotics not only on the growth but also on the virulence of *C. diphtheriae*. Penicillin was chosen as the antibiotic for study since it is the one most commonly used.

The management of the patient with acute diphtheria and of the convalescent carrier is the joint responsibility of the clinician and the public health worker. The diagnosis of the disease and the treatment of the patient are prerogatives of the physician. The bacteriologist in the public health laboratory must furnish the physician with corroborating evidence by means of cultures from cases of acute and convalescent diphtheria. The bacteriologist should be informed whenever cultures are taken during penicillin therapy. If they are taken during that period, the physician should be cautioned that this is the period of greatest bacteriostasis and that cultures taken then may give rise to false or unreliable negative results.

Negative cultures at this period may mean that diphtheria goes undetected and that persistent carriers of *C. diphtheriae* may be released into the general population. The Minnesota Department of Health has recognized this danger and in recent years has added the query "Antibiotics used? When?" to the data cards accompanying diphtheria culture outfits. If the answer to the first question is affirmative, the following warning is typed on the report sent to the attending physician: "We note that this patient is receiving (antibiotics, penicillin, aureomycin, et cetera). We would advise that further nose and throat cultures be submitted at least forty-eight hours after such treatment is completed or terminated."

General Background

That penicillin has no influence upon the toxic effects of *C. diphtheriae* in the patient has been well established. Some investigators believe that penicillin therapy prevents complications from pyogenic organisms. All workers agree that the bacteriostatic effect of penicillin has a pronounced influence in reducing the persistence of *C. diphtheriae* in the upper part of the respiratory tract. The following is a brief review of more recent investigations on this subject.

Skinner,[4] in 1945, working with twenty patients, found that penicillin had a definite bacteriostatic effect upon *C. diphtheriae*. With adequate dosage he was able to eliminate *C. diphtheriae* in twelve of the patients in the first course of treatment.

Karelitz and associates,[3] in 1947, treated eighty diphtheria patients with antitoxin and intramuscular injections of penicillin. He found that 27.5 per cent remained positive at five days, in contrast to 100 per cent positive in controls receiving antitoxin only; 7.5 per cent remained positive at ten days, controls being 92 per cent positive; at fifteen days fewer than 5 per cent remained positive, controls being 85.7 per cent positive. Ten of fourteen convalescent carriers treated with intramuscular injections of penicillin cleared up by the twelfth day of treatment. These workers found some recurrence of positive cultures one to five days after penicillin therapy was stopped. They were convinced that some persistent carriers could be missed unless cultures were taken after cessation of therapy.

Weinstein,[5] in 1947, treated twelve patients with clinical diphtheria with antitoxin only. He found that *C. diphtheriae* disappeared in an average of 33.25 days. Twenty-six patients treated with antitoxin and penicillin failed to show *C. diphtheriae* in an average of 3.96 days.

Bruyon and Brainerd,[1] in 1950, found that sixty-two patients with clinical diphtheria treated with antitoxin only had the first consistent negative culture in 24.5 days on an average. In contrast, eighty-five patients receiving antitoxin and

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From the Minnesota State Board of Health, Division of Medical Laboratories.

penicillin became consistently negative in 17.7 days on an average.

Recent Minnesota Experience

In 1952, only forty-five cases of clinical diphtheria were reported in residents of Minnesota; two cases in nonresidents were diagnosed in the state. There were only five deaths. This is a gratifying record, but its maintenance depends upon continued vigilance and cooperation of clinicians, laboratory workers and epidemiologists. An analysis of morbidity figures shows that sixteen of these forty-seven cases of diphtheria were reported from the age group one through nineteen years of age, whereas thirty-one cases occurred in patients twenty years or more of age. All the deaths occurred in the age group forty to sixty-five. It is significant that most of the cases and all the deaths occurred in the population that had a minimum of protection by immunization. Checking the records of release cultures completed at the beginning of the present work, we found complete culture reports in twenty-one; the results of virulence tests were positive in all twenty-one. Of these, seven were released from quarantine in a period of four to nine days and ten were released in a period of ten to twenty days; four were released in a period of twenty-one to thirty-one days. One patient received a second negative report in four days, two in five days, one in six days and two in seven days. The longest period of quarantine was thirty-one days. The average length of time before release was 13.05 days.

Data in regard to penicillin therapy were provided in only four of these instances. The studies of Hartley and Martin[2] on 457 diphtheria patients treated only with antitoxin indicate that 85 per cent were positive at five days and forty-two per cent remained positive at twenty days. These figures and the previously quoted data suggest the possibility that penicillin therapy was operative in securing negative release cultures in an average of 13.05 days in the Minnesota series. Not included in the group of twenty-one are two cases of clinical diphtheria in which the organisms failed to appear in cultures.

If it is assumed that penicillin is the cause of failure of *C. diphtheriae* to appear in cultures of clinical diphtheria and that its bacteriostatic effect has materially reduced the length of persistence of the organism in convalescence, then two serious problems arise: (1) Does the use of penicillin as a bacteriostatic agent result in undetected clinical diphtheria? (2) Will the release of convalescent patients at an early date furnish a backlog of virulent carriers that may burst into epidemic proportions at a future date? On the other hand, does the bacteriostatic effect of penicillin on *C. diphtheriae* render the organisms nonvirulent?

Present Study

In an attempt to throw some light on these problems, the present study on the inhibiting effects of penicillin on *C. diphtheriae* was undertaken.

Procedures: For the purpose of the study, six virulent strains of *C. diphtheriae* isolated in the current year were selected. Strains 1, 2 and 3 were obtained from patients who had clinical diphtheria with positive virulent tests reported respectively three, two and one-half and two months previously. Strain 4 was isolated from a person who has been a carrier for the past six years and whose last positive virulence test had been reported two months previously. Strain 5 was isolated from a newly detected carrier whose positive virulence test was reported one and one-half months previously. Strain 6 was isolated from a patient who had clinical diphtheria and whose positive virulence test was reported one and one-half months previously.

Serum agar was selected as the medium in which to incorporate varying amounts of penicillin. To check the suitability of the medium before the penicillin was added, the six strains of organisms were inoculated upon the serum agar. Abundance of growth and normal microscopic appearance of the organisms were noted. Virulence tests were done on the twenty-four-hour cultures. A test guinea pig was inoculated intracutaneously with 0.1 cc of a suspension of the cultures. A control guinea pig that previously had received 250 units of antitoxin was inoculated with identical material. All cultures showed strongly positive virulence. The control animal showed no reactions.

After these tests were completed, penicillin was incorporated in the serum agar in amounts of 5, 10, 100, 1,000 and 2,500 units per milliliter respectively. The six strains were inoculated upon the media, and growth was observed at the end of a forty-eight-hour incubation.

Results. Moderate to fair growth was noted in the tubes containing 5 units of penicillin. Micro-

EFFECT OF FIVE UNITS OF PENICILLIN ON GROWTH AND VIRULENCE OF VIRULENT C. DIPHTHERIAE IN EIGHT SERIAL TRANSFERS

Transfer	Cultures Viable in 5 u. Penicillin	Cultures Viable on Transfer to Loffler's	Cultures Nonviable on Both Media	Positive Virulence Test
1-2-3	6	—	0	6
4	3	2	1	5
5	2	3	0	5
6	2	2	1	4
7	2	2	0	4
8	2	0	2	2

scopic examination showed definite distortion of the organisms. No growth was discernible in the tubes that contained 10 to 2,500 units of penicillin. These cultures that failed to show growth in the presence of 10 to 2,500 units of penicillin were then transferred to penicillin-free Löffler's medium. Abundant growth was established in forty-eight hours. These transfer cultures on Löffler's medium were inoculated into guinea pigs, and positive virulence tests were obtained.

The serum agar cultures with penicillin concentrations of 10 to 2,500 units and with no apparent growth were placed in a refrigerator for a week. Then transfers again were made on penicillin-free Löffler's medium. Good growth was obtained after a forty-eight-hour incubation. Guinea pigs inoculated from the subcultures from tubes containing 10 to 2,500 units of penicillin showed positive results.

To show the effect of partial inhibition of growth of virulent *C. diphtheriae*, further studies were made on the six cultures that showed moderate to fair growth in the tubes containing 5 units of penicillin. These cultures were transferred to media containing 5 units of penicillin in a series of eight transfers over a period of two weeks. The accompanying table summarizes the results of such transfer upon the growth and virulence of the organism.

The third transfer showed a decided decrease in growth and an increase in the morphologic distortion of the organisms. This phenomenon increased with repeated transfers. On the fourth transfer, two of the cultures failed to grow on the penicillin medium but were established by transfer to Löffler's medium. One of the cultures failed to establish on both media. On the eighth transfer, only two cultures remained viable on the penicillin medium.

Even though the growth on penicillin medium was scant and rough, positive virulence tests were obtained. In instances in which no apparent growth was obtained on penicillin medium but in which growth could be re-established by transfer to Löffler's medium, positive virulence persisted.

Summary and Conclusions

The bacteriostatic effect of penicillin in amounts of 10 to 2,500 units on six strains of virulent *Corynebacterium diphtheriae* was demonstrated by the failure to produce visible growth after forty-eight hours of incubation. Transfers from these apparently nonviable cultures to Löffler's medium produced abundant growth with typical morphology. These cultures showed no loss of virulence as tested by inoculation of guinea pigs. Storage of the inhibited cultures in a refrigerator for a week did not result in loss of viability on transfer to Löffler's medium. Virulence of the organisms was not affected.

Cultures carried on medium containing 5 units of penicillin showed some inhibition of growth and some distortion of organisms at the initial stages. Progress over eight generations showed more scanty growth and more pronounced distortion. At the eighth transfer only two cultures remained viable. As long as cultures remained viable on the medium with 5 units of penicillin or from transfers therefrom, the virulence test remained positive.

Conclusions

The following conclusions were reached as a result of this study:

1. The virulence of *C. diphtheriae* is not lost by partial or apparently complete bacteriostasis induced by penicillin.

2. Organisms that are partially inhibited or apparently completely inhibited on penicillin medium may be revived on Löffler's medium.

3. It is reasonable to assume that patients whose cultures are negative during antibiotic therapy may harbor virulent *C. diphtheriae* after therapy is terminated. Therefore it is recommended that:

(a) Cultures submitted from acute upper respiratory infections should be taken before antibiotic therapy to enable the laboratory to furnish reliable culture reports to the physician.

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Seminar

EISENMENGER'S COMPLEX

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Victor Eisenmenger, in 1897, reported the case of a cyanotic adult who at autopsy was observed to have a large ventricular septal defect. Twenty-five years later, Maude Abbott brought into focus the fact that in Eisenmenger's case the aorta was overriding the defect of the septum. She separated this malformation from the simple ventricular septal defect on one hand and the tetralogy of Fallot on the other and named it "the Eisenmenger complex." In the last few years, this syndrome has attracted much attention, as the diagnosis of the Eisenmenger complex is considered to be a contraindication for the performance of surgical procedures devised for the relief of cyanosis. Abbottt recorded the basic anomalies to consist of high interventricular septal defect, dextroposition of the aortic valve overriding the septal defect associated with clinical manifestations of cyanosis, dyspnea, enlargement of the heart, diastolic murmurs, clubbing of the fingers and polycythemia. The embryological basis was documented by Abbott. The circulatory dynamics and physiology was discussed by Bing and associates.[2] Cournand and co-workers did cardiac catheterization studies in cases of Eisenmenger's complex.

Definition

The Eisenmenger complex is usually presented as a cyanotic form of congenital heart disease consisting of a high defect of the ventricular septum, the aorta overriding the defect and dilatation of the pulmonary vessels, with an unobstructed pulmonary orifice. As in the tetralogy of Fallot, the hypertrophy of the right ventricle may be added as the fourth feature and the term tetralogy of Eisenmenger has been used by several authors.

The most important feature distinguishing the Eisenmenger complex from a simple ventricular defect is the presence of anoxemia in the former and its absence in the latter syndrome.

Clinical Features

Altogether, thirty-eight cases with clinical and postmortem pathological findings have been reported in the literature. According to Selzer and Laguer,[13] these cases could be divided in three morphological groups: those fitting the classic concept of the syndrome (Group A); those in which the aorta was completely dextroposed (Group B); and those complicated by aortic valvular deformities (Group C). The second group, that of cases of ventricular septal defect with aorta arising from the right ventricle is included as representative of the Eisenmenger's complex and its inclusion will be justified in the following discussion.

Three patients survived over the age of fifty, and the majority died in the third or fourth decade of life. There were eighteen males and fifteen females. In five of the reported cases, sex was not mentioned (all of these five were between a few weeks of age to fourteen months). Twenty-six cases fall under Group A, eight cases under Group B and four cases under Group C.

Cyanosis was a constant feature, as it was present in Groups A and B in all patients with the exception of one infant and one adolescent. In the majority of cases of Group A, cyanosis was described as "moderate" in intensity. In most instances in Group B, it was severe. In sixteen cases of Group A the age at the onset of cyanosis was reported. It was present from birth or infancy in six cases; it developed in childhood in five and in adolescence or adult life in five. In Group B, cyanosis was present from birth or infancy in all cases. Group C was thought to be non-cyanotic. Cyanosis was specifically stated to be absent in three cases, and in the fourth one it was reported as present in association with severe cardiac failure. Polycythemia was reported in nine cases.

Clubbing was reported in twelve cases. A few signs and symptoms, such as chest deformity in five cases, underdevelopment in six cases, weakness and fatigue as a major symptom in four cases, epistaxis in three cases and hoarseness in one case due to pressure on the recurrent laryngeal nerve by the enlarged heart, have been reported in the literature.

Auscultatory findings were reported in twenty-six cases. In two cases, no murmurs were found; in two cases, a diastolic murmur only was noted. In all other cases, systolic murmurs were present but were variable in intensity, quality, location and conduction. The loud systolic murmur at the fourth left costal cartilage characteristic of the

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isolated ventricular septal defect was infrequently encountered. In Groups A and B, there was a frequent association of the systolic murmur with a diastolic blowing murmur best heard at th left sternal border. In Group C, of course, loud diastolic murmurs characteristic of aortic insufficiency were present. As a rule, in the cases of the Eisenmenger complex, there was a loud second sound present in the pulmonic area.

Electrocardiographic reports indicate that right axis deviation, the pattern of right ventricular hypertrophy or a right bundle branch block was, as a rule, present. Exceptions were rare and usually accountable for by complications.

The roentgenographic appearance of the cardiac shadow showed a remarkable uniformity. Of the twenty instances in which roentgenograms were described or reproduced, enlargement of the pulmonary artery was observed in nineteen. The one exception was in the case of a child in whom severe aortic insufficiency was present, which may have been a clinically more significant lesion than the Eisenmenger complex. The pulmonary artery shadow and that of its branches were usually described as prominently enlarged, often extreme in degree. Pronounced hilar pulsations were usually noted on fluoroscopy, and the pulmonary fields showed varying degrees of congestion, often severe. The heart was almost always enlarged:

Cause of death was given as follows:

Heart failure	15	Cerebral abscess	1
Endocarditis	5	Ruptured pulmonary artery aneurysm	1
Pneumonia	3	Cerebral thrombosis	1
Thoracotomy	3	Asphyxia	1
Anoxia	1	Unknown	6
Leukemia	1		

A review of the pertinent features of the cases with necropsy reveals that the clinical picture of this syndrome is well defined. It was shown that the late development of cyanosis has been overemphasized. In all cases with complete dextroposition of the aorta cyanosis is present from birth or infancy and is severe; in those with partial dextroposition of the aorta it frequently developed in infancy or childhood, not unlike the cyanosis of tetralogy of Fallot. The degree of cyanosis on the whole, however, is less severe than in the tetralogy of Fallot, and there are higher degrees of polycythemia. Prominent clubbing of digits is less frequently encountered in the Eisenmenger complex. Cyanosis is usually not present in cases associated with aortic insufficiency, and for that reason it is suggested that this combination be classified as the simple ventricular septal defect with aortic regurgitation.

Pulmonary hypertension appears to be a constant and important feature of the Eisenmenger complex. This may be demonstrated by direct pressure measurements or suggested by the striking dilatation of the shadow of the pulmonary artery and its branches in the roentgenogram, usually associated with intense pulmonary congestion and large amplitude of pulmonary vascular pulsations. This is also supported by the constant finding of the pattern of right ventricular hypertrophy in the electrocardiogram.

Murmurs in the Eisenmenger complex seem to be less characteristic than in uncomplicated ventricular septal defect. Systolic murmurs, often loud and harsh, are usually present and vary in location. Early diastolic murmurs at the left border of the sternum are common and are probably due to relative pulmonary insufficiency. Very loud diastolic murmurs, almost continuous, and changes in systemic pulse pressure have been reported in cases associated with aortic valve deformity. As a rule, the second sound in the pulmonary area is prominently accentuated.

The prognosis of the Eisenmenger complex appears to be fair and is comparable to that of other congenital defects of the less serious type. The average patient survives to early or middle adult life. Heart failure and bacterial endocarditis are important causes of death.

The following determinations will allow a complete and accurate laboratory diagnosis:

1. Hypertrophy of right ventricle by x-ray and pulmonary hypertension by cardiac catheterization may be determined accurately.
2. Information relative to overriding of the aorta may be secured by several methods; the cardiac catheter may be passed from the right ventricle directly into the aorta; the arm to tongue circulation time may be abnormally short; diodrast may be seen to pass directly from the right ventricle into the aorta, and finally, as will be shown, by analysis of oxygen and pressure relationships.
3. A high ventricular septal defect may be demonstrated by the presence of increased oxygenation in the upper portion of the right ventricle or pulmonary artery or by passage of the catheter through the defect. Thus it will be seen that each of the abnormalities comprising the Eisenmenger complex is subject to reasonably accurate determinations by one or more of the special methods outlined.

With cardiac catheterization, overriding of the aorta may be demonstrated in three ways: (1) passage of catheter from the right ventricle directly into the aorta, (2) simultaneous pressure records and (3) by demonstrating admixture of left and right heart blood in the aorta.

Pathology

Pathological features of the Eisenmenger complex at autopsy were as follows: cardiac hypertrophy, almost uniformly affecting the right ventricle, a large basal ventricular septal defect, varying degree of dextroposition of the aorta and dilatation of the pulmonary arterial trunk to aneurysmal proportions.

The defect of the ventricular septum was always located in the typical area, involving the membranous septum, its anterior part or the muscular septum just anterior to it. Its shape was usually oval, occasionally crescentic or round. The size of the defect varied from 1 to 3 cm. in the longest diameter in the fully developed heart. Since the size of the defect is of crucial importance from the standpoint of pathogenesis of the malformation and its hemodynamic effect, it was thought of importance to compare the size of the defect with that of the defects unassociated with other malformations (Roger's disease) and those associated with dextroposition of the aorta and pulmonary stenosis (tetralogy of Fallot). It is seen that the majority of uncomplicated defects of the ventricular septum are small; in the Eisenmenger complex moderate-sized defects occur. With the exception of the rare isolated defects in the lower septum, the location and shape of the defects in the three syndromes are identical. It should be emphasized that the largest defect in the ventricular septum encountered in any case of these syndromes occupies only a small portion of the septum, probably less than 10 per cent of its surface, in the fully developed heart. There is no gradual transition between larger defects of the ventricular septum and the complete absence of the septum.

The position of the aorta in relation to the septal defect has been usually reported as "overriding." In some cases, the degree of dextroposition of the aorta was estimated, and it is seen that all degrees of transition may be found, from cases in which the aorta originates almost entirely from the left ventricle to those in which it is completely dextroposed, i.e., originates from the right ventricle.

The other two features of the Eisenmenger complex, dilatation of the pulmonary artery and hypertrophy of the right ventricle, were consistently present throughout the series. Five cases showed serious vascular changes in the pulmonary arterioles.

Other pathological findings on autopsy include conditions like patent ductus arteriosus (three cases), patent auricular septal defect (three cases), hypoplasia of aorta (three cases), aneurysm of cusp of aortic valve (two cases), atypical coronary artery distribution (two cases), coarctation of aorta (two cases), anomalous distribution of vessels from arch (one case), retraction and insufficiency of aortic cusps (one case) and finally congenital tricuspid fetal endocarditis (one case), have been recorded.

Morphological Relationship to Uncomplicated Defect of Ventricular Septum

It has been shown that the secondary features of the Eisenmenger's complex, the dilatation of the pulmonary artery and the hypertrophy of the right ventricle are consistently observed in large, simple defects of the ventricular septum. The morphological distinction between the latter and the Eisenmenger complex, based on the presence or absence of dextroposition of the aorta, may present more difficulty at the autopsy table than is generally appreciated. It is of interest to note that Eisenmenger reported his original case as one of a ventricular septal defect and in a later article clarified his position concerning the overriding of the aorta. He presented evidence that the aorta is normally located in such a relation to the ventricular septum that if the membranous portion of the latter is defective it comes in contact with both ventricles. Thus, he believed that overriding of the aorta is a normal by-product of a large ventricular septal defect and not a developmental dextroposition. The unity of the two syndromes has been accepted in the German literature.

In order to account for the morphological similarity between larger ventricular septal defects with or without the dextroposition of the aorta, and in consideration of the generally accepted fact that the aorta may be embryologically malposed in such cases, a different way of unifying the two syndromes has been suggested. It was thought that the same process of maldevelopment which brings about transposition of the arterial trunks in its severest forms, and is responsible for partial dextroposition of the aorta (overriding) with or without pulmonary stenosis, in less severe forms, causes the isolated high ventricular septal defect in its mildest form. This view finds further support from studies done:

1. It was shown that within the Eisenmenger complex all degrees of dextroposition of the aorta can be found.

2. The otherwise identical defects are, on the average, larger in the Eisenmenger complex (or tetralogy of Fallot) than in the uncomplicated form.

3. A striking example is presented in a case of Selzer,[13] in which the fetus had a defect of the ventricular septum appearing identical with that of the mother, except for the dextroposition of the aorta, which was present in the mother but not in the fetus. Yet it was the fetus that had an abnormal origin of the coronary ostia, which is considered characteristic for the transposition malformation.

Although Eisenmenger's original interpretation appears in part incorrect, his observation helps to emphasize the difficulty which is often encountered at autopsy in deciding whether or not dextroposition of the aorta was present during life. It is highly probable that the physiological overriding of the aorta can be accentuated by changes in pressure relationship, in topography of the arterial trunks related to gradual dilatation of the pulmonary artery and in other factors. Thus, overriding of the aorta need not be considered a fixed morphological feature.

Pulmonary Vascular Changes and Their Relation to Cyanosis

The uniform finding of pulmonary hypertension in cases of Eisenmenger's complex studied by cardiac catheterization suggests that these changes are an essential part of the syndrome; it was shown also that pulmonary arteriolar resistance is extremely high.

The reported changes in the pulmonary arterioles belong pathologically to the same group as those found frequently in other conditions associated with pulmonary arterial hypertension [mitral stenosis, cor pulmonale and those associated with large pulmonary arterial flow (septal defects, patent ductus arteriosus)]. The changes in the intima and media show some similarity to systemic arteriolar changes in systemic hypertension. In severe cases of pulmonary arteriolar disease, the changes resemble those of malignant hypertension.

From the physiological standpoint it is clear that the pulmonary arteriolar changes represent evidence of long-standing severe pulmonary hypertension. It has been suggested, however, by Burwell, Taussig and Blalock that pulmonary vascular disease interferes with oxygen exchange in the lungs and thus it is responsible for cyanosis. This opinion was based on consideration that oxygen inhalation raised the arterial oxygen content and that in the Eisenmenger complex the circulation to the lungs is not "inadequate." Yet, neither of these factors helps in determining the mechanism of cyanosis. There is no valid reason to separate the Eisenmenger complex from all other forms of congenital heart disease with cyanosis in which it is evident that anoxemia is caused by an admixture of venous blood in the systemic circulation. On the contrary, one can present evidence that in Eisenmenger complex, caval blood enters the aorta; this is shown by hemodynamic studies and by angiocardiography. Moreover, a causal relationship between pulmonary hypertension and arteriolar disease on one hand and cyanosis and anoxemia on the other has not been definitely established. Finally, Bing and his associates interpreted the finding of a close check between blood flow determination in Eisenmenger complex performed by the oxygen and carbon dioxide methods as evidence of a normal pulmonary oxygen exchange. One can further cite the case of Soulie and his collaborators as illustrating the lack of relationship between cyanosis and the pulmonary factors. In spite of proved severe pulmonary hypertension and arteriolar disease, the arterial oxygen saturation was normal, as apparently the co-existing aortic insufficiency "corrected" the overriding of the aorta to a point that caval blood could not enter it.

It seems reasonable to believe that anoxemia and cyanosis in the Eisenmenger complex are due to a "shunt" mechanism and the pulmonary venous blood is fully oxygenated, such as is shown directly in the tetralogy of Fallot and the anoxemic form of atrial septal defect.

Circulatory Dynamics[3]

A discussion of the hemodynamics of Eisenmenger's complex can best be opened by summarizing facts about which there appears to be reasonable certainty.

1. *Pathological Facts.*—(a) The defect of the ventricular septum is always located in the basal, subaortic portion of the septum and occupies only a minute portion of the septum, with most of it intact. (b) The aorta may originate mostly from the left ventricle, may arise mostly from the right ventricle, or may occupy an intermediate position. All degrees of transition can be found between the two extreme positions. (c) The pulmonary vascular changes indicative of high arteriolar resistance can usually be demonstrated.

2. *Clinical Facts.*—(a) Systolic pressure in the right ventricle and the pulmonary artery are equal and are always elevated in the Eisenmenger's complex but are occasionally normal in simple ventricular septal defect. This is shown by a collection of results of cardiac catheterization. (b) In many cases, perhaps the majority, the pressure elevation in the right ventricle-pulmonary artery system is such that systolic pressure readings are identical with the pressure readings in the aorta and the left ventricle. (c) Increased oxygenation of pulmonary arterial blood and decreased oxygenation of systemic arterial blood prove that there is a cross shunt, or premixing of caval and pulmonary venous blood. The systemic flow may equal the pulmonary flow or one may exceed the other, without any consistency in this relationship.

3. *Experimental Facts.*—Studies on experimental defects of the ventricular septum show that a fistulous connection between the two ventricles of a dog's heart comparable to a medium-sized human ventricle septal defect results in flow of blood through the fistula from the higher pressured left ventricle to the lowered pressured right ventricle. No equalization of pressure in the two chambers takes place, but a slight fall in the left and a rise in the right ventricular systolic pressure occur.

It would seem from the foregoing summary that the most characteristic feature of Eisenmenger's complex is the presence of severe pulmonary hypertension, which is invariably demonstrated in this syndrome, in contrast to the atrial septal defect, the ventricular septal defect and the patent ductus arteriosus where it is only occasionally present. The consistency in the observation of severe pulmonary hypertension and the frequency with which equal pressures in the two arterial trunks are demonstrated indicate that increased pulmonary resistance plays an important role in the dynamics of the circulation of the Eisenmenger complex.

It has recently become apparent that in certain anomalies of the heart, pulmonary hypertension may have to be present from birth as a condition for the survival of the patient. The reason for this is the fact that the physiological postnatal adjustment, during which an equal pressure in the pulmonary artery and the aorta changes into a high resistance and high pressure systemic circulation and a low resistance and low pressure pulmonary circulation, could prove fatal in those cases in which both trunks originate from a common source. The malformation in such a case creates a pressure pump which has two outlets; the quantity of blood entering each of them is in inverse proportion to the resistance offered. Thus, a low resistance pulmonary outlet could draw all the blood, with none available for the high resistance systemic circulation. In such cases, survival depends on equal resistance in the pulmonary and systemic circuits, which can be achieved by (a) systemic hypotension or pulmonary hypertension or by (b) a mechanical obstruction of the low resistance outlet, as pulmonary stenosis. The obvious conditions to which this concept of a "double outlet ventricle" applies are partial transposition of the arterial trunks, in which both great vessels originate from the right ventricle and the trilocular heart, in which both trunks arise from a common ventricle. A somewhat similar situation exists in some cases of patent ductus arteriosus with coarctation of the aorta in which the right ventricle maintains the pulmonary circulation and the lower part of the systemic circulation.

It is obvious that in the variant of the Eisenmenger complex with complete dextroposition of the aorta (Group B), one is dealing with a "double outlet ventricle" in which pulmonary arteriolar constriction and/or disease has to be present from birth for survival, since no protective pulmonary stenosis is present. However, in the classic Eisenmenger complex where the aorta is merely overriding the septal defect, one has to search for another factor explaining the pulmonary hypertension.

It was shown that an experimental ventricular septal defect produces a pathway for a left to right shunt. One can assume, however, that as the defect in the ventricular septum becomes larger and approaches the size of the aortic orifice the left ventricle becomes a "double outlet ventricle," in which case the low resistance right ventricle can draw all the blood away from the systemic circulation, unless the resistance in the right side of the heart is raised either by pulmonary stenosis or by pulmonary arteriolar constriction and hypertension. Pressure relationships between the two ventricles in cases of Eisenmenger complex and Fallot's tetralogy suggest that in the majority of them the defect is in effect large enough to name the left ventricle a "double outlet ventricle."

The concept of the "double outlet ventricle" as applied to the Eisenmenger complex, the trilocular heart and other similar syndromes is based on fundamental principles of hemodynamics and has to be postulated to explain the fact that survival in the post-embryonic life is at all possible with some of these malformations. However, the mechanism of the production of the protective pulmonary hypertension in such cases, the stimulus for the development of pulmonary arteriolar constriction and/or disease from infancy, is as yet unknown.

Another problem requiring analysis is that of the physiological significance of the "overriding" aorta. It is commonly assumed that an "overriding" aorta collects blood from both ventricles. Such a statement ignores the fact that normally the systolic pressure in the right ventricle is lower than the diastolic pressure in the aorta, and, if normal relationships were preserved, blood could not ordinarily flow from the right ventricle to the aorta. Deoxygenated caval blood can enter the aorta (a) if the systemic circulation is maintained by the right ventricle in complete dextroposition of the aorta; (b) if pressures in the two ventricles are equal, in which case the overriding position of the aorta facilitates turbulence and mixing of blood in the deficit; (c) if the pressure in the right ventricle is slightly lower than in the left, in which case turbulence may still throw right ventricle blood into the aorta, perhaps aided by the Venturi phenomenon.

The importance of the topographic relationship of the aorta to the ventricular septal defect in the production of anoxemia is exemplified by observations of Soulie and his associates in their cases of the Eisenmenger's complex associated with aortic valvular deformity. In one such case, the aorta was seen at autopsy to be dextroposed over the septal defect so that morphologically this was an instance of Eisenmenger's complex, but the longitudinal axis of the aorta was observed to be rotated counter clockwise, probably as a result of changes brought on by aortic valve deformity and insufficiency. This shift corrected the overriding position of the aorta, and the patient was found to have a normal oxygen saturation, even though severe pulmonary hypertension was present. Significantly, two other patients with the Eisenmenger's complex associated with aortic insuffi-

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THE SURGEON AND THE MYCOLOGIST

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THE USE of surgical procedures for treatment in certain selected cases of mycotic infection is well established. However, the surgeon must keep constantly in mind that certain fungous infections may be encountered unexpectedly during operations for therapeutic or diagnostic purposes. The selective therapeutic effect of certain drugs useful for treating such infections makes it necessary to know precisely the nature of the fungus.

Fungous Infections

The pathogenic fungi frequently important to the surgeon are *Blastomyces, Histoplasma, Coccidioides, Cryptococcus, Sporotrichum, Nocardia, Actinomyces* and *Aspergillus.* Recent work would indicate that *Actinomyces* may be classed more properly as a bacterium than as a fungus. However, for the sake of convenience, it will be considered as a fungus in this presentation. With the exception of *Blastomyces* and *Coccidioides,* infections due to fungi are world-wide in distribution. Except for *Sporotrichum,* fungi are likely to produce lesions in the lungs. These may be solitary lesions simulating bronchogenic carcinoma or they may be multiple, bilateral and widespread, simulating extensive tuberculosis. Many of them may excavate, producing cavities that enhance the clinical similarity to tuberculosis. Like other intrapulmonary lesions that become chronic, they may metastasize to vital areas, such as the brain, kidneys or spinal column. It is important, therefore, that the nature of the infection be established as early as possible because many of these metastatic lesions may be prevented by modern chemotherapeutic agents. Since tuberculosis is the most common cause of chronic infection in the lung and since most of the mycotic infections simulate tuberculosis, the intrapulmonary lesions frequently progress to fatal termination while extensive bacteriologic studies of sputum, gastric washings and bronchial secretions over months or even years continue to be reported "negative for tubercle bacilli."

Diagnosis

It is important, therefore, when studies are made relating to intrapulmonary lesions, that cultures for fungi should be made as routinely as are cultures for tubercle bacilli or smears for malignant cells. The added expense is small and is insignificant when contrasted with the benefits to the patient. It is indeed pitiful to see patients treated with first one and then another antibiotic, empirically, intermittently or inadequately, while the condition progresses and metastasizes to the brain because careful mycologic studies have not been made. Such studies may determine that the patient needs iodides, sulfonamides or stilbamidine, or that the dosage of penicillin should be increased severalfold. The surgeon who sees such patients in consultation frequently can be of great help by requesting that proper search be made for fungi.

In most cases the diagnosis of fungous infections must be made by isolation of the organism. Skin tests have been tried and in some instances have been useful. Most patients who have coccidioidomycosis are likely to give a positive cutaneous reaction after intradermal injection of coccidioidin, but in the experience at the Mayo Clinic most of those who have chronic localized forms of this disease give negative reactions, even when *Coccidioides immitis* is isolated from the lesion. In the remainder of the mycotic infections already mentioned, skin tests are unreliable in diagnosis; in some cases, they even may be misleading.

Dr. Weed is from the Section of Bacteriology of the Mayo Clinic.

Histopathology

Many times when a surgeon removes tissue for biopsy complete reliance for a diagnosis is placed on the histopathologic interpretation. Regardless of the site of the lesion, such a procedure may be entirely inadequate. The human body can react to stimuli in a limited number of ways and the response to infection with a fungus may closely simulate that seen in other infections. Many times the organisms are not visible in the histologic sections or, if found, may not be sufficiently characteristic for accurate identification. It is frequently impossible to determine on a morphologic basis whether a given organism is *Blastomyces dermatitidis, Blastomyces brasiliensis, Coccidioides immitis* or *Cryptococcus neoformans* but it is extremely important to know the true identity. *Sporotrichum, Nocardia* and *Actinomyces* are rarely, if ever, visible in sections stained with the usual hematoxylin and eosin. The masses of material frequently called "sulfur granules," when stained and subjected to culture, may be found to contain not *Actinomyces* but streptococci, micrococci, *Nocardia, Cephalosporium, Aspergillus* or no organism at all. It is extremely important to the patient to know the nature of these "granules." The surgeon should see that the proper specimen in adequate amount is sent for bacteriologic and mycologic studies. The patient's limb or life may depend on it.

Therefore, the surgeon should consider the possibility of a fungous infection in any condition simulating tuberculosis or neoplasm. He should attempt to establish the diagnosis as early as possible. This is best done by isolation of the organism, either from sputum, bronchial washings or material taken directly from the lesion.

* * *

This is the eighteenth in a series of editorial reports sponsored by the Minnesota Society of Clinical Pathologists and designed to foster closer relationships between the practicing physician and the pathologist.

Together with the preceding one, entitled "The Surgeon and the Bacteriologist," this report emphasizes the growing importance of bacteriologists and mycologists in the modern practice of medicine. The essential role played by such specialists in the field of clinical pathology has been spotlighted by the advent of new drugs and antibiotics.

Emphasis must be placed on the point that the histopathologic picture is not always specific in either bacterial or mycotic infections and that the appearance of tissue cannot be substituted diagnostically for isolation of the causative organism. Surgical pathologists were somewhat reluctant at first to accept this stand, but increasing experience has proved its correctness.

Reprints of most of this series of reports are available from the authors or the chairman of the editorial committee of the Minnesota Society of Clinical Pathologists.

George G. Stilwell, M.D.
Chairman, Editorial Committee,
Mayo Clinic.

POLIO VACCINE TRIAL NEEDS PHYSICIANS' AID AS IT MOVES INTO EVALUATION PHASE

More than 600,000 children have completed three inoculations, in the field test of the trial polio vaccine developed by Dr. Jonas E. Salk of the University of Pittsburgh. The emphasis now shifts to the evaluation study under the direction of Dr. Thomas Francis, Jr., University of Michigan School of Public Health. The validity of the evaluation is dependent upon data gathered on poliomyelitis cases in the test groups, *including those children in the first three grades who did not get vaccine.*

In addition, data on cases among family members of participating children are an integral part of the study. Since the number of poliomyelitis cases among the test groups may not be large, it is essential that all cases are completely reported. Early diagnosis, prompt reporting and follow-up, and the securing of *necessary epidemiological information and laboratory specimens* are important factors in the evaluation.

An outline of procedures and copies of necessary forms have been sent to local and state health authorities. It is important that physicians in areas where vaccinations were not given, co-operate in the study by notifying local or state health officers of cases occurring among children who participated in the trials and then migrated to another area and children who go to summer camps. Local health officials also need information on participating children who receive injections of Gamma Globulin.

This phase of the study will depend, to a large degree, on the whole-hearted co-operation of practicing physicians.

President's Letter

SUMMER SLUMP?

Most of us along about this time of year find ourselves a little on the lazy side. We are inclined to let the oppressive heat of summer capture our senses to some extent, and a consequent summer slump is likely to occur.

But there are many things in medical organization which should not be allowed to leave our consciousness entirely. Simply because late spring meetings are over, and a recess is in effect until fall, we should be aware that our attention is needed in many projects of vital importance.

One of these is the American Medical Education Foundation. Most of us have heard many pleas for contributions to this fund. One of the most recent was that presented to the House of Delegates at our June meeting in Duluth. The worthwhile nature of this project was emphasized there, and facts and figures were presented which pointed up the literal urgency of this program.

Since the fund was begun in 1951, 298 Minnesota physicians have given a total of $26,279.00 into this fund. In return, the University of Minnesota medical school has received $66,956.00—an excellent return for our investment.

The American Medical Education Foundation was founded and has been sponsored by the American Medical Association, which has given $500,000.00 annually to the fund, so that all money raised could be meted out to the nation's seventy-nine medical schools, without encumbrance.

One of the main reasons for the initiation of such a group was the threat of Federal subsidy. Because of the need for funds by medical schools throughout the country, Congress considered a bill which would provide these funds at government expense. Thus, the group was formed, and it was felt that if about ten million dollars could be raised over a period of years, the need would be eased sufficiently so that there would be no need for Federal subsidy.

Minnesota doctors have not given their full share toward this important fund, as the above figures indicate. The need is still great. It should be pointed out that all funds contributed can be earmarked by the giver for whatever medical school he wishes.

It is hoped that the traditional summer slump will not jeopardize this important work in Minnesota. This offers an excellent opportunity for Minnesota doctors to do their share in insuring the maintenance of high medical school standards in the United States. It is a national problem, for the seventy-nine medical schools in fifty-six cities are charged with the responsibility of turning out an estimated 6,000 physicians annually.

It is clearly our job to see that schools can maintain and improve these standards—a challenge which continues, regardless of the traditional summer slump.

President, Minnesota State Medical Association

Editorial

ARTHUR H. WELLS, M.D., *Editor;* HENRY G. MOEHRING, M.D., and JOHN F. BRIGGS, M.D.

STATEWIDE CANCER DETECTION

A RECENT EDITORIAL in MINNESOTA MEDICINE has emphasized the cost of finding early cancers in the Cancer Detection Center at the University of Minnesota. The same editorial also encouraged limitation of x-ray examinations of internal organs to persons presenting symptoms of visceral disease; this would be in contrast to the Cancer Detection Center program of studying symptom-free people for internal cancers by means of appropriate x-ray techniques when certain established criteria are present. The directors of the Cancer Detection Center wish to reply to this editorial and define the relationships of the Detection Research Center to a generalized community-wide cancer detection effort.

It is evident from past experience that for purposes of over-all service to the people of a state a single cancer detection center cannot hope to fulfill the needs of the population. However, statewide examination service is not the basis of existence of the Minnesota detection center. Rather, it functions dually as a testing unit to pioneer in improvements of practical diagnostic methods, and as a source of stimulation for both lay and professional groups. The reticence of already heavily overburdened physicians to take on the routine examination of apparently well persons in the manner now necessary for adequate cancer screening is readily understandable. Without a central effort of this kind, however, sponsored by the State Council of the Minnesota Medical Association and supported generously by the Minnesota Division of the American Cancer Society, any service program of early detection of cancer would drift about like a rudderless ship.

Undue emphasis upon cost factors is hard to defend in the light of present mortality statistics for most serious cancers. If we consider the over-all yearly expenditures in the Cancer Detection Center, compared with the number of examinations performed, it is apparent that the average cost of an examination has been slightly under $25.00. The cost per cancer found has been slightly over $3,000.00. If we add precancerous lesions to the cancers found, the cost for detection of either a precancerous lesion or a cancer is $180.00. Is it reasonable to believe that patients found to be free of cancer in the Detection Center receive nothing for the token fee which they contribute and the small expenditure of research funds allotted to their examination? I think not.

If one sets out to evaluate life in terms of dollars and cents, it would appear that the tremendous expenditure of money yearly in the United States for the treatment and care of poliomyelitis patients is a rather wasteful expenditure. By and large, severely affected polio cases are likely to be liabilities to the community after the cure of the disease, rather than an asset. Is such a thought to be the motivating factor in forsaking the cure and rehabilitation of poliomyelitis patients? Similarly, can anyone place a monetary valuation upon a group of lives, either *prolonged* for a few years or *saved* through early cancer detection?

Even though the numbers of cancers in any given organ found in our Cancer Detection Center during six years of operation is not great, an appraisal of the figures immediately reveals a significant increase of patients surviving cancer therapy when their disease was detected in the absence of symptoms. Such a simple comparison of results argues strongly for the benefits to be derived from earlier diagnosis. A review of current publications reporting five-year survivals for serious cancers, such as gastric cancer, impresses one with the significance of our present survivals in the asymptomatic patients found in the Detection Center. When we consider the great discrepancy between survivals of patients with cancer of the rectum and colon when the lesion is limited to the mucosa and submucosa as against lesions where cancer has spread to regional lymph nodes (eighty to ninety per cent in the first group and thirty to thirty-five per cent in the second group), the value of early diagnosis is again apparent.

If every doctor's office program were aimed at a thorough cancer examination only of patients with symptoms, results probably would not be much better than we currently see reported from medical and surgical centers around the United States. It is only with an increased stimulation and emphasis upon the *yearly examination of well people* that the every doctor's office program can be expected to achieve its maximum benefit with an increased percentage of long-term survivors.

With an increasing enlightenment of the lay public through the efforts of state and county chapters of the American Cancer Society, coupled with an earnest desire on the part of most physicians in the State of Minnesota to perform a searching and complete examination for cancer at the patient's request, we may well see a continuing improvement in the number of five-year survivals from serious malignancies. Coincident with this general effort, there should be a continuing search at the Cancer Detection Center for better, and more specific methods of diagnosis.

When a new specific and general cancer test appears, it can best be tried under the controlled conditions of a research center; in such ways your Cancer Detection Center has contributed and will continue to contribute to the general effort. The Director of the Cancer Detection Center urges doctors throughout the State to take an active part in the *examination of well people for early, asymptomatic cancers.*

Claude R. Hitchcock, M.D.
Director, Cancer Detection Center
University of Minnesota

A REACTION TO CRITICISM

During the last year, several articles appeared in the lay press concerning unethical surgical procedures, such as fee-splitting and ghost surgery. The impetus for these articles has become credited, rightly or wrongly, to the American College of Surgeons and its director, Dr. Paul Hawley. At the annual meeting in Chicago in October, 1953, college officials called in reporters and, to quote the *New York Times*, "Served notice on the *rest of the medical profession* that it was ready to meet all comers in its year-old campaign to eradicate surgical practices which the college considered unethical."

The reaction of most physicians to this announcement has been complete agreement with the objectives and complete disagreement with the methods. Following publication of an article in *Collier's* AMA Secretary George Lull sent the editor a telegram of sharp protest, as did the American Academy of General Practice; in contrast, the Director of the American College of Surgeons sent a message of commendation.

This sensational, dogmatically critical, quixotic approach to a problem of such seriousness is unfortunate. It is unworthy of the American College of Surgeons, an organization which has done more than any other to improve hospital care and raise surgical standards, yet which is now becoming an anathema to organized medicine. In doing so, the American College of Surgeons dissipated its great potential for accomplishing a fine and worthy task, one which in some sectors is urgently in need of doing.

This problem is rightly one for the Joint Commission on Accreditation and the American Medical Association; their efforts to be implemented by the American College of Surgeons, the American College of Physicians, the American Academy of General Practice, and others. These, with effective and sound methods to achieve a fully and correctly informed public, can accomplish the desired result. It is similar to the situation regarding Christian ethics—they will work miracles if we would but try them!

We all live together in this House of Medicine. Where this House is dirty, let's flush it out! If parts of it are rotten, then tear them down and build anew! But sensational, iconoclastic and misinterpreted articles in the lay press hurt us all. We can only hope that important and influential men *within the home* will see this in time, and do enough.

Chas. C. Cooper, M.D.

Editor's Note.—If the drastic action of the American College of Surgeons and Dr. Paul R. Hawley of calling upon public opinion to halt certain unethical practices fails to produce anything more than righteous indignation in the ranks of organized medicine in Minnesota, we shall have lost a most opportune time for action, and the exposé will then have been a miserable failure.

The responsibility for a planned and co-ordinated action in this field properly belongs in our County and State Medical Societies. Any organization so far removed from the individual physician as the Joint Commission on Accreditation will do no better than the American College of Surgeons.

"Our profession creates its eminence through deeds, not words, and through meeting human needs, not in satisfying our own" (from John P. Bowler, M.D.).

PHYSICIAN, HEAL THYSELF

LET US not allow self-interested, self-appointed groups unjustifiably to air our rare and minor defects in the public press. Let us seek remedies for our supposed ills through our own County and State Medical Societies, the American Medical Association, and the American Academy of General Practice, in accordance with rational medical ethics.

In 1903, the American Medical Association declared that the split of a fee without knowledge of the patient was an unethical practice. The American Academy of General Practice in Cleveland, in March, 1954, re-affirmed its unequivocal opposition to fee-splitting, ghost surgery, and other unethical medical practices, as it had done in previous meetings. It decried public statements and other publicity detrimental to medicine, recently aired by some apparently self-appointed spokesmen for the profession.

Certain basic intraprofessional rules should be generally accepted by physicians. No special selfish group should have a surgical or hospital monopoly. Every doctor in good standing should be able to hospitalize his patients. No group should inspire self-laudatory public statements for themselves or statements which imply incompetence of other groups. It must be recognized that general practitioner surgeons do most of the ordinary surgery in this country with excellent results, as shown by morbidity and mortality studies and patient satisfaction. Self-study, assisting competent surgeons, doing surgery under supervision, and observing surgery have produced surgeons equal in ability to those with more formal training, and should be recognized as methods of training.

The general practitioner who acts as family physician, as a rule, does only what he is qualified to do. He is the backbone of the practice of medicine. He can care adequately for most of his patients' illnesses, and he is more interested than anyone else in securing specialist assistance, when necessary, to promote his patients' welfare. Every patient should be under the direct and immediate supervision of his personal physician, with that physician making referrals when necessary.

The American Academy of General Practice requires that each member complete 150 hours of postgraduate training each three years, in order to retain his membership in the organization. All physicians should be required to keep up with the new developments in medicine. Physicians should follow the teachings of Christianity and Hippocrates, and be willing to teach other physicians their knowledge and techniques.

The referring general practitioner and the consultant or surgeon should each be adequately remunerated in proportion to the services rendered. The family general practitioner should have a place in the operating room as surgeon, assistant or observer, according to his abilities and desires, and the patient should be aware of the duties of each doctor in his complete care. This will induce harmony and satisfaction for the patient and his doctors, prevent the existing evils, improve the intraprofessional relations of physicians, and increase patient satisfaction.

The American Academy of General Practice, at the inspiration of Dr. Merrill Shaw, a member of its Board of Directors, who recently died of cancer, is fostering a new project, *"A Family Doctor for Every Doctor's Family,"* which proposes that every doctor secure a family physician for himself and his immediate family in the hope that physicians' early mortality can be decreased and their families' health protected.

A *"Family Doctor for Every Family"* would reduce the cost of medical care and greatly promote better intraprofessional and public relationships and understanding of physicians.

There should be more equitable representation of all branches of medicine on the Joint Commission on Accreditation of Hospitals, with its control vested in the House of Delegates of the American Medical Association. A democratically selected, joint commission could clear up the rare abuses. The hospital staffs have an important responsibility in encouraging physicians to promote voluntary high standards of medical care.

Postgraduate medical education should be available to any physician so that he may improve his ability in any line of work at any time he wishes. No group should deny this opportunity to the general practitioner. Most general practitioners have a major interest in some branch of medicine and a minor interest in others. In justice to themselves and their patients, there should be unlimited opportunities for them to improve their abilities.

Grievance Committees in county and state medical societies should be publicized and become more active in improving intraprofessional and public relations.

We physicians, through democratic processes, must work out improved methods of conducting our professional affairs and improving our personal health and the health of the public for the benefit of all.

J. A. Cosgriff, M.D.
President, Minnesota
Academy of General Practice

SEMINAR SECTION

Manuscripts of the miniature monograph type are being accepted for publication in the Seminar section. Practically complete presentations of limited medical subjects of wide interest are being solicited. "The Eisenmenger Complex" by Ananda Prasad in this issue is the first of a series. Your contribution will be appreciated.

CHEMOTHERAPY FOR TUBERCULOSIS

The Problem

Many patients now being discharged from sanatoria may continue to receive anti-tuberculosis chemotherapy; some may receive chemotherapy before entering the sanatorium and a few get this treatment without ever going to the sanatorium. These practices raise problems affecting the state and local health departments, the public health nurses, practicing physicians, and the patients themselves. The following recommendations are made to assist in the management of these patients.

Recommendations

Any patient who is to receive chemotherapy for tuberculosis should have a careful evaluation of his condition to insure the proper utilization of all available methods of therapy. Chemotherapy is only one important part of the total treatment of a patient with active tuberculosis. This is particularly important when, for some special reason, sanatorium care is not definitely planned for the patient. Under these circumstances the physician in charge is obviously responsible for prescribing the drugs, including the specific form of the drug, the exact dosage, the route and frequency of administration and the duration of chemotherapy. The person responsible for giving the treatment, such as a nurse, should be given this information in writing.

When a patient is to receive anti-tuberculosis chemotherapy after dismissal from the sanatorium, his private physician or the physician at the sanatorium, should be notified simultaneously, and in advance if possible, so that they may plan for the care of the patient at home. They should be given complete information about the drugs being used, including the specific form of the drug, the exact dosage, and the route and frequency of administration and total duration of treatment with drugs.

Although a nurse may be given responsibility for the administration of the drugs the patient must remain under the supervision of a physician, either his private physician or the physician at the sanatorium and should be examined by him at least once every two months. The patient or nurse in charge should report to the physician any untoward reaction to the drugs but the physician should make the decision to continue or stop treatment with the drug suspected of causing the reaction. This is particularly important as interruptions in the course of the treatment seem to increase the chance that drug-resistant strains of tubercle bacilli will occur.

The drugs used most frequently today are streptomycin, isoniazid (INH) and para-aminosalicylic acid (PAS).

Streptomycin may be given as streptomycin, dihydrostreptomycin, or streptoduocin, which is a mixture of equal quantities of streptomycin and dihydrostreptomycin. These drugs are available as the sulfate or the calcium chloride complex and may be bought already in solution or as a powder to be dissolved in sterile distilled water. They must be given intramuscularly, the usual dose for adults varying from one gram daily to one gram twice a week. The most common toxic reactions are dermatitis, drug fever, dizziness, and deafness. Many patients receiving some form of streptomycin complain of transitory numbness and tingling particularly about the mouth.

Isoniazid is usually given orally, parenteral preparations being reserved for critically ill patients who cannot swallow the tablets. The usual dose for adults varies from 150 to 300 mgm. per day, divided into two to three doses. The most common toxic reactions are peripheral neuritis, irritability, and drug fever.

PAS is usually given orally as the sodium or calcium salt, in tablets, granules, capsules, or powders. The usual daily dose is twelve grams of PAS divided into three or four doses. The equivalent daily dose of the sodium salt is sixteen grams. Preparations for parenteral administration are available. Irritation of the gastrointestinal tract producing anorexia, nausea, vomiting, and diarrhea may occur when the drug is given orally in the recommended dose. This can be diminished by giving PAS along with food. Other reactions are dermatitis and drug fever.

Reporting

All reports to the Minnesota Department of Health of cases of tuberculosis should include information as to anti-tuberculosis chemotherapy already received by the patient.

A statement prepared by the Committee on Tuberculosis, Minnesota State Medical Association, May 17, 1954.

The Dean's Page

SHORT COURSES IN TECHNOLOGY

A recent high school graduate was referred by her family physician to the secretary of the county medical society for information about training in medical technology. The secretary of the society told her about the splendid four-year course in medical technology at the University. She replied that she could not afford such a long course and asked about the one-year courses offered by the commercial schools. He advised against these schools but failed to mention to her the one-year program for laboratory aids inaugurated a year ago by the University. The result was that this young woman went to a business college.

I mention this because there is an urgent need for more trained young women in laboratories, in x-ray departments, and in doctors' offices. In fact, the course for laboratory aids was established at the request of the Minnesota State Medical Association and with the co-operation of the Minnesota Society of Medical Technologists, the Minnesota Society of Clinical Pathologists, and the Minnesota State Board of Health.

This course is one year in length, with six months spent in basic instruction at the University and six months in laboratory work under supervision in a hospital laboratory in the Twin Cities. The course does not prepare young women to be medical technologists, but it does prepare them to perform simple laboratory procedures under supervision in hospitals or in doctors' offices.

Somewhat similar to this is the new three-month course offered by the University in the basic sciences pertinent to x-ray technology. To be admitted to this course, an individual must be accepted for x-ray training in one of the approved courses in a Minnesota hospital.

Enrollment in these courses has been disappointingly small and must be increased to justify their continuation. The reason for the small enrollment probably is that the programs are new and not well known. We hope that this will be corrected by the physicians of the state, at whose request the courses were established.

HAROLD S. DIEHL, M.D.
Dean of Medical Sciences
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Medical Economics

Edited by the Committee on Medical Economics
of the
Minnesota State Medical Association
George Earl, M.D., Chairman

ILO CONVENTION GETS "THUMBS DOWN"

According to a recent *AMA Washington Letter,* President Eisenhower has forwarded to Congress the International Labor Organization's convention on minimum standards of social security, *with a recommendation that it not be ratified.*

It will be recalled that the convention, which was adopted by the ILO in 1952, if adopted by individual nations, would become similar to a treaty, and in effect, would be the supreme law of the land. The convention covers nine fields: Medical care, sickness benefits, unemployment benefits, old age benefits, employment injury benefits, family benefits, maternity benefits, invalidity benefits and survivor benefits. A government is considered to have ratified the convention if it promises to meet the requirements in three of the nine fields.

The *Washington Letter* states: "The medical care section stipulates that a country may qualify as ratifying if it agrees to provide one of the following: (a) a system of compulsory health insurance, (b) private, voluntary health insurance 'administered by public authorities under established regulations' set by law, or (c) private, voluntary health insurance administered by insurance companies but under government 'supervision.' Half the population would have to be covered."

Quoting the President's words in transmitting the convention to Congress, the letter states:

> "is . . . regarded as not suitable for ratification but rather for referral to the appropriate federal and state authorities for their consideration."

Accompanying the transmittal was a summary of comment from all affected federal departments and agencies, which pointed out that federal laws already are in accord with two of the points, old age insurance and survivors insurance. On the other points, the agencies came to the same conclusion as the President, namely that these issues are within the jurisdiction of state governments and "that therefore the convention is not appropriate for ratification" by Congress.

Because there is no time limit for ratification of the convention (treaty), this matter presents a constant possibility of thrusting compulsory health insurance, or some form of government-controlled health insurance on this country. Presumably, the convention has been studied from 1952 until the present time by various agencies and departments of the federal government.

Reconsideration Apparent

Apparently the delayed study of the implications in the treaty have caused the President and his advisors to reconsider the original U. S. delegation's favorable vote. The U. S. delegation voted in favor of the convention originally, but a spokesman for the delegation was quoted as saying at that time that this did not imply that the U. S. would accept the entire convention. Ratification would require a two-thirds vote of the Senate. Also, the State Department pointed out, in 1952, that the U. S. delegation agreed that the convention should not be recommended to the Senate for ratification as a treaty (which would be binding on this government and all the states), but should instead be passed along to Congress and the states for final disposition.

The U. S. delegation consisted of Senator Murray (D., Mont.) and Assistant Secretary of Labor, Philip M. Kaiser, representing government; George P. Delaney of the AF of L, representing employes, and Charles P. McCormack, president of McCormack Tea & Spice Co., Baltimore, employer representative.

The recent summary also noted that while signatories to the convention agreed to bring it before their respective legislative bodies, "it is entirely within the discretion of the competent authority of each country to determine whether any legislation is to be enacted." The federal agencies concurring in this opinion were Commerce, Interior, Justice, Labor, Navy and Health, Education and Welfare Departments, and the Civil Service Commission.

Although the outcome of the convention bears close watching, it can be hoped that the treaty, which caused so much concern among medical and other groups, will die a natural death because of the President's recommendation.

"GIVE-AWAY" SHOW GETS MEDICAL CHECK-UP

A new tack in medical public relations has revealed that medicine has become somewhat concerned over the impressions of medicine given by certain radio and television shows. Questioning the heartbreak stories of participants on "Strike It Rich," it is thought that the viewing audience, running into millions, has been left with the impression that financial assistance was needed in paying for the high cost of medical care.

In checking on these matters, representatives of the AMA and the Medical Society of the County of New York contacted the producer of the show, in an effort to reach some understanding. The interview was successful, apparently, for it was reported that "we were most impressed with the sincerity of . . . (the producer) in his desire to assist the medical profession in any way possible. He apparently has great admiration for the medical profession and in no way wished to discredit the profession on his show."

Subsequently, the producer asked the AMA to investigate two cases. In one case, in which the total medical bill had accumulated to $6,000, it was found that the family was covered by Blue Cross and had used this service. Also, it was found that at no time was the patient without medical care because of financial difficulties.

Case Dismissed

In both of these cases, according to a recent *AMA Secretary's Letter,* the investigations revealed that the individual had been "adequately taken care of at the community and state level, and, consequently, there was little basis for the appeal. This information was made available to (the producer) and both cases were considered ineligible to participate on his program."

This type of "preventive medicine" has again proved valuable to the profession as a whole. In averting an unpleasant situation before it occurs, the profession's lay representatives on a national level have demonstrated that an ounce of prevention against poor public relations is well worth more than a pound of cure.

JOINT COMMISSION ISSUES ACCREDITATION FIGURES

The May issue of the *Bulletin of the Joint Commission on Accreditation of Hospitals* carries a summary of accreditation figures as of December 31, 1953. The figures show that of the total number of registered hospitals, all types and sizes, 43.6 per cent are accredited. The bulletin points out however, that "if the hospitals of less than twenty-five beds are eliminated (and these hospitals are not now eligible for accreditation), more than half, or 51.8 per cent, are accredited."

The bulletin further points out that the figures show that the greatest emphasis must be placed on the area of the smaller hospital in order to improve the total picture in regard to accreditation of hospitals. A committee is now studying methods to assist the smaller hospitals in earning accreditation.

Basic Standards Developed

A set of standards for the Commission's guidance have been developed over the years of experience of the original accrediting agency, the American College of Surgeons and the work of the Joint Commission. This publication, *"The Standards for Hospital Accreditation,"* is the basic operating document for the commission. It is regarded as a sound and wisely-developed measuring stick for the evaluation of hospitals. The Board of Commissioners has stated that it would act slowly in making any changes in the basic standards, but that methods for review are being established to take changing conditions into consideration.

POPULATION SHOWS LARGE INCREASE

The population of the United States continues to show an annual increase over and above previous years. In 1953, the population increased 2,700,000, bringing the total at about 161,200,000. These figures are from a *Statistical Bulletin* of the Metropolitan Life Insurance company. The bulletin states: "This large increment, exceeding somewhat the previous high gain registered in 1952, reflected not only a record-breaking number of births, but also a low death rate and a moderate increase from immigration." Breaking down this statement further, the bulletin says:

> "The new high record for births was not far short of the four million mark. The actual number, including an allowance for unregistered births, was more than 3,950,000, or nearly twenty-five per 1,000 population. The births in 1953 were nearly 2 per cent greater than in the preceding year and more than 50 per cent higher than in 1940. The year just ended was the seventh in succession in which births exceeded three and one-half millions. In the past decade, the number of babies born reached the remarkable total of more than 35 million."

Infants Deaths Low

Also, according to the bulletin, infant mortality declined in 1953, establishing a new low estimated at twenty-eight per 1,000 live births, or slightly below the previous minimum recorded the year before. This decline in infant mortality represented a decline of 30 per cent from that a decade ago and of more than 50 per cent from that two decades ago.

The general mortality rate was also at a low level. The 1953 death rate was 9.6 per 1,000, or about the same as in the preceding year, which established a new minimum. The bulletin states that "this record is especially impressive because 1953 was marked by an outbreak of respiratory disease early in the year and a severe hot spell in late summer, both of which brought a rise in mortality over the seasonal normal.

. . . one of the purposes of a journal is to provide a means for the dissemination of the opinion of its readers . . . It is not likely that all the letters received can be published, therefore somebody has to select those most suitable for this purpose and to reject others. . . . Fortunately, there will be many who will not approve the editor's decision, for that keeps all concerned alert and on their toes, but he is fortified by the knowledge that a measure of disapproval is inescapable, and that all he can do is to endeavor to see that it moves round as much as possible, and does not stagnate in one place.—*Lancet*, April 3, 1954.

EISENMENGER'S COMPLEX

(Continued from Page 584)

ciency were non-cyanotic, which adds great weight to the observations and explanation of Soulie and his co-workers.

In the light of the previous discussion, one can define the Eisenmenger complex to consist of (a) a high localized defect of the ventricular septum, (b) a position of the aortic orifice in relation to the defect which permits an admixture of venous blood to enter the aorta, as evidenced by anoxemia and (c) an absence of significantly elevated resistance at the pulmonary outflow tract of the right ventricle. Seconary features of the syndrome, not included in the definition but constantly found in the entity are (a) strikingly elevated resistance at the pulmonary arterioles with organic changes within them; (b) severe pulmonary hypertension or even equalization of pressure in the two circulations; (c) hypertrophy of the right ventricle and (d) dilatation of the pulmonary artery and its branches.

Bibliography

1. Alexander, Fred and White, Paul D.: Ann. Int. Med., 1:64 (July) 1947.
2. Bing, R. J., Vandam, L. D., and Gray, F. D.: Bull. Johns Hopkins Hosp., 80:323 (June) 1947.
3. Bond, Vernard F. Jr.: Am. Heart J., 42:424, 1951.
4. Cosby, Richard S.; Levinson, David C.; Griffith, George C.; Zinn, Willard J., and Dimitroff, S. P.: Am. J. Med., 11:31, 1951.
5. Dillion, W. H., and Schreiber, Jr., H.: Am. J. Physiol., 159:281 (Aug.) 1948.
6. Glazebrook, A. J.: Brit. Heart J., 5:147, 1943.
7. Goldberg, H.; Silber, E. N.; Gordon, A., and Katz, L. N.: Circulation, 4:343, 1951.
8. Hawley, J. G.; Little, R. C., and Feil, H.: Circulation, 1:321 (March) 1950.
9. Hurst, W. W., and Schem, F. R.: Am. Heart J., 36:144 (July) 1948.
10. Jarcho, Saul: J. Mt. Sinai Hosp., 18:59 (May-June) 1951.
11. Plachta, Aaron and Speer, Francis, D.: J. Pediat., 42:325 (Mar.) 1953.
12. Saphir, O., and Lev, M.: Am. Heart J., 21:31, 1941.
13. Selzer, Arthur, and Laguer, G. L.: Arc. Int. Med., 87:218, 1951.
14. Taussig, H. B., and Semans, J. H.: Bull. Johns Hopkins Hosp., 66:156, 1940.

AMERICAN MEDICAL ASSOCIATION

House of Delegates—Résumé of Proceedings

San Francisco, June 21-25, 1954

Fee splitting, osteopathy, closed panel medical care plans, veterans' medical care and the training of foreign medical school graduates were among the major subjects of discussion and action during the sessions of the House of Delegates at the American Medical Association's 103rd Annual Meeting June 21-25 in San Francisco.

Named as president-elect for the coming year was Dr. Elmer Hess of Erie, Pennsylvania, who, until his election, was serving as a member of the House of Delegates and as Chairman of the Council on Medical Service. Dr. Hess will become president of the American Medical Association at the June, 1955, meeting in Atlantic City, succeeding Dr. Walter B. Martin of Norfolk, Va. Dr. Martin took office at the Tuesday evening inaugural session in San Francisco's Palace Hotel.

The House of Delegates voted the 1954 Distinguished Service Award of the American Medical Association to Dr. William Wayne Babcock of Philadelphia for his outstanding contributions to medicine and humanity. Dr. Babcock, who was professor of surgery and clinical surgery at Temple University School of Medicine from 1903 to 1944, received the award from Dr. Martin at the Tuesday evening inaugural ceremony.

The final registration total for the San Francisco meeting was expected to reach approximately 35,000, including more than 12,000 physicians.

Fee Splitting

The House adopted a supplementary report of the Reference Committee on Miscellaneous Business which recommended acceptance of a Judicial Council report on the subject of billing and made the additional recommendation "that the House of Delegates resolve that it firmly opposes fee splitting, rebating or payment of commissions in any guise whatsoever, and that it further opposes any mechanism that encourages this practice."

The Judicial Council report included the following statements:

> "The Judicial Council is of the opinion that the only new facet concerning this subject that has come up recently is the case of joint billing to some of the non-profit insurance companies. In many cases these insurance companies insist on a joint or combined bill, but the bill is being paid in most instances by two checks. This is not considered unethical and all insurance plans which do not pay the individual physician in this manner should be urged to do so.
>
> "The Judicial Council is still of the opinion that when two or more physicians actually and in person render service to one patient they should render separate bills.
>
> "There are cases, however, where the patient may make a specific request to one of the physicians attending him that one bill be rendered for the entire services. Should this occur it is considered to be ethical if the physician from whom the bill is requested renders an itemized bill setting forth the services rendered by each physician and the fees charged. The amount of the fee charged should be paid directly to the individual physicians who rendered the services in question.
>
> "Under no circumstances shall it be considered ethical for the physician to submit joint bills unless the patient specifically requests it and unless the services were actually rendered by the physicians as set out in the bill."

Osteopathy and Medicine

Four resolutions dealing with the osteopathic problem were considered. The House accepted a recommendation by the Reference Committee on Medical Education and Hospitals and adopted a Supplementary Report of the Board of Trustees on a Report of the Committee for the Study of Relations Between Osteopathy and Medicine:

> "The justification or lack of justification of the 'cultist' appellation of modern osteopathic education could be settled with finality and to the satisfaction of most fair-minded individuals by direct on-campus observation and study of osteopathic schools. The Committee, therefore, proposed to the Conference Committee of the American Osteopathic Association that it obtain permission for the Committee for the Study of Relations between Osteopathy and Medicine to visit schools of osteopathy for this purpose.
>
> "The Conference Committee favorably recommended this proposal to the board of trustees of the American Osteopathic Association which considered it at a special meeting on February 6-7, 1954. It has referred the question to its house of delegates which will act upon the proposal in July, 1954. If the action of the house of delegates of the American Osteopathic Association be favorable, the on-campus observations can be carried out in the fall of this year.
>
> "The Committee therefore recommends:
>
> "1. That no action be taken on the report at this time and that final action be deferred until December, 1954.
>
> "2. That the Committee be continued until December, 1954, in order to be available to evaluate education in schools of osteopathy should the house of delegates of the American Osteopathic Association act favorably upon the recommendation of its Conference Committee."

Closed Panel Plans

The much-publicized New York resolution, calling for several changes in the Principles of Medical Ethics relative to participation in closed panel medical care plans, was considered by the Reference Committee on Miscellaneous Business. That committee made the following recommendation, which was adopted by the House:

> "In the discussion before your reference committee on this resolution, it became apparent to the committee that clarification and interpretation of the Principles of Med-

> ical Ethics in relation to prepaid medical care plans are desirable. As set forth in the bylaws, the Judicial Council has jurisdiction on all questions of medical ethics.
>
> "Therefore, your reference committee recommends that the House of Delegates request the Judicial Council to . . . investigate the relations of physicians to prepaid medical care plans and render such interpretations of the Principles of Medical Ethics as the Council deems necessary, and report to the House of Delegates not later than the next annual meeting of the Association.
>
> "The committee further recommends that the New York resolution be referred to the Judicial Council for consideration in connection with this investigation."

The New York resolution, among other suggested changes, would add the following new paragraph to Chapter I, Sec. 4, "Advertising," of the Principles of Medical Ethics:

> "It should be understood that any medical care plan, company, or organization which advertises for subscribers and directs such subscribers to a restricted panel of physicians for medical care is advertising for the benefit of the physicians involved."

Veterans' Medical Care

Accepting a report by the Reference Committee on Legislation and Public Relations, the House adopted two strong resolutions condemning the present practice of establishing service-connection for veterans' disabilities by legislative fiat. In recommending passage of both resolutions, the committee said:

> "The study of the chronological expansion by law and regulation, together with evidence presented of pending legislation now before a Congressional Committee, emphasize all too clearly the imperative need of decisive action on the part of the American Medical Association.
>
> "It is the opinion of the Committee that the time is at hand when the American Medical Association and its component societies should go all out in preventing this unscientific method of determination of service-connected disabilities, and that we respectfully request that copies of these resolutions be transmitted to the Congress of the United States and other appropriate federal agencies."

In connection with veterans' medical care, the House also adopted recommendations by the Reference Committee on Insurance and Medical Service which reaffirmed the policy on non-service-connected disabilities, established at the 1953 annual meeting, and which commended the informational program carried out since then by the Committee on Federal Medical Services of the Council on Medical Service.

Foreign Medical Graduates

Three resolutions and a Board of Trustees supplementary report were submitted to the House regarding the evaluation of foreign medical school graduates, a subject which attracted major interest earlier this year at the annual Congress on Medical Education and Licensure in Chicago. The Reference Committee on Medical Education and Hospitals spent much of its time listening to the ideas and proposals of various state medical societies, state licensing boards, members of the Council on Medical Education and Hospitals and others. The reference committee recommended that "the intent and aims of this Supplementary Report and the three resolutions can best be met by referring the entire problem to the Council on Medical Education and Hospitals for further study. It is recommended that the Council report at the Interim Session in 1954 regarding the progress relative to this study." The House adopted the reference committee's recommendations.

Seal of Acceptance

The Council on Medical Service presented a supplementary report outlining the difficulties encountered in conducting the Seal of Acceptance program, and recommending discontinuance of the Seal of Acceptance for voluntary health insurance plans. The report said that the standards and principles of the program will be maintained as guides and recommendations for all groups operating or establishing plans. The House, on recommendation of the Reference Committee on Insurance and Medical Service, adopted the Council report, thus terminating the Seal of Acceptance program for voluntary health insurance plans.

Registration of Hospitals

The House also approved a Board of Trustees report calling for discontinuation of the registration of hospitals by the Council on Medical Education and Hospitals and suggesting that the Joint Commission on the Accreditation of Hospitals be requested to undertake the registration of hospitals in addition to its present accreditation activities.

Miscellaneous

Among a wide variety of other actions, the House also:

Voted to continue the holding of the annual Clinical Meetings;

Approved the establishment of a program of medical military scholarships with appropriate safeguards limiting the number of students involved;

Approved the extension, on a voluntary basis, of the Medical Education for National Defense program which currently is in operation in five medical schools as a pilot study, and

Authorized the Council on Scientific Assembly to conduct a thorough study of the use of tape recordings of the material presented at meetings of the Council, and asked for a report at the December meeting.

Opening Session

Highlights of the opening House session on Monday were selection of Dr. Babcock as recipient of the Distinguished Service Award and the addresses by Dr. Edward J. McCormick of Toledo, then president of the Association, and Dr. Martin, then president-elect.

Dr. McCormick called upon the medical profession to take the guesswork out of medical costs by adopting average fee schedules on an area or regional basis. The Reference Committee on Reports of Officers later suggested that the Board of Trustees make a study of such programs where they already are in operation, and the House approved.

Dr. Martin, in his opening session address, declared that the most urgent problem before the medical profession is that of financing hospital services to make them more generally accessible. In his presidential inaugural address, Dr. Martin said that physicians are duty-bound to keep themselves informed on public matters affecting the medical welfare of the people, and he also urged doctors to "reach back farther than the disease" in treating their patients.

Special Citations

Two special citations were presented by the Association during the San Francisco meeting. During the presidential inauguration ceremony Dr. McCormick presented an award to a fellow Toledoan, Dr. Nicholas P. Dallis, for his outstanding health educational service as the writing member of the team that produces the illustrated feature, "Rex Morgan, M.D." At the closing House session on Thursday, Dr. Martin presented a special citation to Smith, Kline & French Laboratories of Philadelphia for "pioneering use of television in bettering the health of the nation." The plaque was accepted for the company by Mr. Francis Boyer, president.

The closing session also brought the announcement that the California Medical Association had presented a check for $100,000 to the American Medical Education Foundation.

Election of Officers

The election at the closing session brought the following results, in addition to the selection of Dr. Hess as president-elect:

Dr. Clark Bailey of Harlan, Ky., was named vice president.

Dr. David B. Allman of Atlantic City and Dr. F. J. L. Blasingame of Wharton, Texas, were reelected to their positions on the Board of Trustees.

Also re-elected were Dr. George F. Lull of Chicago, secretary; Dr. J. J. Moore of Chicago, treasurer; Dr. James R. Reuling of Bayside, N. Y., speaker of the House of Delegates, and Dr. Vincent Askey of Los Angeles, vice speaker.

Dr. J. Morrison Hutcheson of Richmond, Va., was named by Dr. Martin as a member of the Judicial Council to succeed Dr. Edward R. Cunniffe of New York, who served as Council chairman for many years. Dr. Homer Pearson of Miami, Fla., was elected new chairman.

Dr. W. Andrew Bunten of Cheyenne, Wyo., was elected a new member of the Council on Medical Education and Hospitals, succeeding Dr. W. L. Pressly of Due West, S. C. Dr. Charles T. Stone, Sr., of Galveston, Texas, was re-elected to the same Council. Both terms run to 1959.

Dr. Floyd S. Winslow of Rochester, N. Y., was re-elected to the Council on Constitution and By-Laws for a term ending in 1959.

Dr. Joseph D. McCarthy of Omaha Neb., was re-elected to the Council on Medical Service for another term running to 1959. To fill the vacancy created on the same Council by Dr. Hess' resignation following his election as president-elect, Dr. Robert L. Novy of Detroit, Mich., was selected.

The House of Delegates also chose New York City as the place for the 1957 annual meeting, San Francisco for 1958 and Atlantic City for 1959. Previously selected were Atlantic City for 1955 and Chicago for 1956. The dates of next year's meeting in Atlantic City are June 6-10.

J. Arnold Bargen, M.D.
O. J. Campbell, M.D.
George Earl, M.D.
F. J. Elias, M.D.
(Delegates)

THE EFFECT OF PENICILLIN UPON THE VIRULENCE OF C. DIPHTHERIAE

(Continued from Page 579)

(b) Cultures taken for the purpose of releasing patients from restrictions should be taken no earlier than forty-eight hours after antibiotic treatment has been discontinued.

References

1. Bruyon, H., Brainerd, H.: Penicillin in the Treatment of Diphtheria. Am. J. Sc., 219:408, 1950.
2. Hartley, P., Martin, C. J.: Proc. Roy. Soc. Med. London, Sec. Epidemiol. and State Med., 13:277, 1919-1920.
3. Karelitz, S., Wasseman, L. R., Moloshok, R. E.: Penicillin in the treatment of diphtheria and diphtheria carriers. J. Pediat., 30:1-18, 1947.
4. Skinner, J. W.: Penicillin in persistent diphtheria. U.S. Naval M., Bull., 45:264-6, 1945.
5. Weinstein, L.: Treatment of acute diphtheria and the chronic carrier state with penicillin. Am. J. M. Sc., 213:308-14, 1947.

Public Health

THE PHYSICIAN AND VITAL STATISTICS

In almost every medical article he reads, the physician may see and study statistical tables relating to the measurements of human life. He probably gives little thought to the fact that his own cases play a significant part in the construction of such tables. Perhaps he does not fully realize the value of statistics as guides in the many factors influencing man's well-being.

Actually each individual physician's records form an essential part of the vast collection maintained by the State Board of Health. From this storehouse of facts, nosology brings back to the physician reports of current trends in causes of death, together with comparisons and interpretations of such trends that may have direct application to cases under his care. Many indications of answers to problems faced by the physician in his day-to-day practice may be found in the statistical bases that underlie a particular case. From correct reports amassed from all sources and statistically treated, many a physician may obtain sound public health guidance in the treatment of his individual patients.

Vital statistics constitute the basis of all medical research. For instance, the "Medical and Health use" items on the uniform Certificate of Live Birth are of great value in analyzing infant and maternal deaths. Such items are considered confidential and are therefore not included in the certified copy of a birth certificate given to a person requesting it. This information, however, is indispensable for accurately determining trends in natality and mortality throughout the state.

The Physician's Responsibility

Every practicing physician shares in the responsibility to help the statistician compile a body of accurate and complete vital data that may be of great value to other physicians. Lately there has appeared a tendency to abbreviate causes of death in the medical certification on Certificates of Death. This practice often causes confusion and delay. The state registrar of vital statistics has to communicate with the physician concerned in order to ascertain just what he meant by the letters he used to record a particular cause of death. Querying follow-ups are annoying to the physician, and answering them may well involve far more time than would have been needed to fill in the certificate completely in the first place. Furthermore, if the death certificate must be produced in court in a civil or criminal proceeding, the physician may be called upon to explain items that are not clear. Every practitioner will want to avoid such encroachments on his time. He can at least minimize them by writing out causes of death accurately and completely, thus removing all doubt as to what, in his professional judgment, brought about each death.

In some cases the physician may require extra time to complete his medical certificate—for instance, when autopsy findings require study or consultation. In such a situation he should write the words "diagnosis deferred" after the heading of the "Medical Certification." He should at the same time date and sign this certificate and forward it to the vital statistics section of the State Board of Health. Upon receipt of such certificates—incomplete but promptly filed—the state registrar will forward to the physician a special Medical Certification form on which he may record the cause or causes of the death after he has ascertained them to his satisfaction. The same procedure should be followed by coroners.

Help from Board of Health

All vital statistics offices in the United States are now operating under rules of the World Health Organization. The Minnesota State Board of Health is increasing its efforts to obtain on all death certificates the cause-of-death certification provided for under these rules. Some physicians in the state still appear to lack full understanding of these accepted procedures. Because the State Board of Health desires to remedy this situation, it proposes to provide help through this page in subsequent issues. The material published for the guidance of physicians will present a number of illustrative cases in an attempt to clarify coding points that may be troublesome.

J. W. Bower, M.P.H., *Director*
Departmental Administration
Minnesota Department of Health

Minneapolis Surgical Society

Meeting of January 7, 1954
President Robert Tenner, M.D., in the Chair

TUMORS OF THE HAND

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Chicago, Illinois

The hand is the site of a great variety of tumors. Some of these are among the most frequent tumors seen, e.g., ganglia, synovial cysts, epidermoid cysts. Others occur with great rarity on the hand, e.g., sarcoma, sebaceous cysts. Some seem to be almost peculiar to the hand, e.g., enchondromas, glomus tumors, ganglia, implantation cysts and isolated xanthomas of the tendon sheaths.

The diagnosis of tumors of the hand should be made early because of the ease of examination and as a corrollary the cure rate should be high. The removal of benign appearing skin lesions will appreciably reduce the incidence of malignant tumors since in many instances it appears that benign lesions are the precursors of malignancy.

In scarcely any other part of the body does trauma and irritation seem to play such an important role in the development of malignancy. Sunlight and other types of irradiation, chemical and mechanical irritants, old scars, and puncture wounds have all been shown to be factors leading to the development of malignant tumors, particularly of carcinoma. This observation certainly points the way to prophylactic measures and likewise emphasizes the possible medicolegal aspects of skin cancer in industry.

All tumors of the hand are treated preferably by surgery. While it may be admitted that some will respond to irradiation therapy, this treatment often fails and is followed by the development of an irradiation damage to the skin often of more serious import and demanding more extensive surgery than excision of the original lesion. Biopsy of tumors of the hand should consist in their complete removal, and if the lesion is sufficiently serious, frozen section should be carried out while the patient is still on the table. In this way further surgery, if indicated, can be carried out then and there. Removal of the deep tumors of the hand demands a thorough knowledge of the anatomy of the hand. Many of the benign tumors simply displace nerves and tendons, grow around them or insinuate themselves between them. A digital nerve, for example, may be deeply buried in a groove of a benign xanthoma from which it must be carefully dissected free. The deep lipomas may follow tendon sheaths and require extensive dissection.

Some idea may be gained as to frequency of the various tumors of the hand from an analysis of some 700 tumors. Of these tumors, approximately one-third were ganglia. Another third was made up of four tumors in about equal numbers—xanthomas, epidermoid cysts, angiomas, and irradiation carcinoma. One-sixth was made up of carcinoma (not developing on the basis of irradiation), lipoma, fibroma, neuroma, and enchondroma, again in approximately equal numbers. The remaining 1/6 comprised the remainder of the hand tumors: synovioma, glomus tumor, fibrosarcoma, giant cell tumors, osteosarcoma, osteoma, Ewing's tumor, osteoid osteoma, lymphangioma, sweat gland carcinoma, myxoma, leiomyoma, metastatic tumors, et cetera.

It is convenient to classify these tumors according to the tissue from which they take their origin—skin, fibrous tissue, fatty tissue, bones and joints, tendons, tendon sheaths and joint capsules, nerves, blood and lymphatic vessels, and muscles. There is not 100 per cent agreement as to whether some of the tumors are actually tumors in the strict sense of the word, e.g., xanthoma, ganglion, angioma, and glomus tumor. Certain of them, like the fibroma and fibrosarcoma, neuroinoma and neurosarcoma, are still confused as to actual status.

Tumors of the Skin

The common *wart* while probably not a true tumor and usually treated by neglect, home remedies, or the dermatologist, comes to the surgeon's attention under one of several circumstances. The subungual wart is especially troublesome; it causes an ugly lesion under the nail, is resistent to ordinary therapy, and is awkward to deal with even surgically. It is a nuisance to the patient and becomes tender and inflamed quite easily. The surgeon also sees the wart which has failed to respond to treatment or which persists without change over many years and then begins to grow or to ulcerate. He also sees the common wart when it has failed to respond to usual treatment, has broken down and become infected, or has ulcerated after various types of home care. The surgeon likewise sees the wart when other more serious changes have occurred—when it develops spreading ulceration or a cauliflower-like proliferation, often following mechanical injury. The surgeon also sees the palmar wart which, because of its location, is frequently subject to trauma.

In all such instances noted above, it is my feeling that surgical treatment is indicated. Usually simple excision will suffice with suture closure, although a wide excision may be needed and occasionally a skin graft may be necessary to close the defect. In case of the subungual wart, the raw surface left beneath the nail must always be closed by a skin graft and a certain amount of nail deformity must be accepted as a sequel. If there is any question as to possible malignancy of the lesion, a frozen section may be indicated at operation. A subungual wart, which proves to be a carcinoma may require amputation of the digit. Also, what may at first instance appear to be a simple wart may prove to be a melanoma and likewise require amputation of a finger.

Kerotoses, hyperkerotoses and *squamous papillomas* are not infrequently on the backs of the hands of individuals who have been out in the sun and weather a great deal, in elderly people, sailors, farmers, et cetera. Such lesions in themselves are of slight moment, but they should not be ignored since they may be the precursor of malignancy.

The *epidermoid or inclusion or implantation cyst* is a quite frequent tumor of the hand which resembles clinically the ordinary sebaceous cyst. It differs from the sebaceous cyst in that it is derived strictly from epithelium, is lined by squamous cell layers as is the skin, and is filled with desquamated epithelial cells and debris. It occurs very frequently on the hand, whereas the sebaceous cyst is very rare on the hand. There is quite a literature about these tumors. Some authors have contended that they develop as the result of the implantation of bits of skin beneath the surface as the result of puncture wounds, hence the name traumatic epithelial cyst or implantation cysts, or, since occasionally foreign material is found in them, they are also called inclusion cysts. On the other hand, it would seem that some of them are the result of proliferation of cellular rests of epithelium which on proliferation produces cystic cavities. They have been produced experimentally. In a few instances the skin over them has shown a small scar as evidence of earlier trauma. In an occasional one a foreign body, such as the tip of a thorn has been found.

The *implantation cyst* occurs especially frequently on the volar surface of the hand and digits. A favorite location is at the base of the palm over or between the regions of the metacarpophalangeal joints. They are sometimes seen in association with Dupuytren's contracture in which case they may be due to drawing down and eventual pinching off of a tiny islet of skin by the attachments of the fascia to the corium of the overlying skin. They occasionally become infected, particularly those lying at the base of the palm. Ordinarily, however, they produce few symptoms and are simply a cosmetic blemish or interfere with the use of the hand. They should be removed intact surgically and do not recur.

The *sebaceous cyst* is a distinct rarity on the hand, although it may occur on the dorsum. It is treated by excision, as anywhere else in the body.

Carcinoma of the Hand

Carcinoma comprises about 90 per cent of all the malignant tumors of the hand. It likewise makes up close to 10 per cent of all hand tumors. It is one of the tumors of the hand in which an irritation is of great importance in its development. Some 70 to 80 per cent of carcinomas develop on the basis of some discernible irritative factor. Only some 20 to 30 per cent of carcinomas of the hand develop without known cause from previously normal skin.

Carcinoma of the hand may be classified etiologically into a number of groups as follows:

1. *Sun and Weather.*—Cancer of the skin of the hand occurs not infrequently in sailors, farmers, men who spend much of their time out of doors and exposed to wind, rain, and sunshine. Our great Southwest furnishes a great deal of cutaneous carcinoma and, although the face is the most frequent site for these tumors, the hands, especially the dorsum, suffers not infrequently. The lesion starts out as an apparently harmless area of keratosis which later becomes scaly, inflamed, and tends to bleed.

2. *Irradiation with X-Rays and Radium.*—In our experience, the most frequent cause of carcinoma of the hand is x-ray irradiation. It is seen particularly in those individuals whose hands are exposed over a period of months or years to often repeated but small doses of x-ray, either adventitious or therapeutic. Thus we used to see it very often in the older professional roentgenologists who were exposed to irradiation in the days before the dangers were clearly understood. It is still seen occasionally in the roentgenologist, but more often now in doctors who use x-ray in their practice for diagnosis, removal of foreign bodies, insertion of pins, and fracture reduction.

It is also often seen in dentists who up to 10 to 20 years ago were holding films in the patients' mouths while making dental x-ray films. It is seen from time to time in the roentgen and radium technician who for one reason or another has subjected his or her hands to excessive irradiation over a period of years.

We likewise see irradiation carcinoma occuring on the hands of individuals who have been subjected to x-radiation in the treatment of chronic recurring dermatitis of various sorts.

Carcinoma may develop in irradiation dermatitis incurred in other ways as well, but not with the frequency with which it follows chronic repeated small doses.

The majority of these carcinomas are of low grade but if they persist long enough and are subjected to further irritation they eventually metastasize to the regional nodes.

The very great frequency (50 to 60 per cent) with which carcinoma develops in irradiation dermatitis, especially that following frequently repeated small doses, makes excision of all involved skin advisable even in instances in which the patient apparently is keeping the process controlled with salves and oils. Wide excision and immediate grafting is to be counciled early before infection develops in the skin and when carcinoma, if present, is still confined to the skin and held in check by the dense subcutaneous fibrosis which characterizes the condition.

3. *Chemical Irritants.*—These causative factors have long been recognized in various occupations and in industry as leading to the development of carcinoma of the skin. These irritants are many and include acids and alkalis, tar, anilin, pitch, oil, paraffin, grease, paint, and coal soot. True, other parts of the body may be involved as well as the hand, but the hand suffers frequently enough and prophylaxis should be practiced.

4. *Burn Scars.*—Carcinoma has long been recognized as developing in scars of burns, especially of burns which were slow to heal, and are associated therefore with a

thick keloid-like mass covered with a thin and easily traumatized epithelium. It is probably the traumatization of this thin overlying skin which finally leads it to break down into a malignancy. Cracks, infections and possibly even solar irradiation may assist in the process. The carcinomatous breakdown occurs only after many years (40 to 60) following the burn. Here prophylaxis would consist in the removal of these thick keloid burn scars before breakdown has occurred.

5. *Internal Medication.*—Arsenic, taken over a period of many years as Fowler's solution, is well known to lead to the development of keratoses on the palms and soles, and also on other areas of the body. These keratoses are precursors of squamous cell carcinoma, developing in many foci.

6. *Chronic Infection.*—Tuberculosis of the skin, particularly if it has been irradiated, has long been known to break down into carcinoma. Chronic osteomylitic sinuses likewise may reveal areas of squamous cell carcinoma in the epithelial lining of the sinus tract. Puncture wounds, especially of the palm, have from time to time been reported as leading to carcinoma. It is not always clear whether this is due to foreign body irritation or to infection; or is purely coincidental.

7. *Irritation of Previous Benign Lesions.*—In this category the common wart is the most frequent offender. In some instances it would appear that the lesion was a carcinoma from the start, but in many it seems without question that the preliminary lesion was in fact a wart. Cauterization, the use of escharotics, or irradiation and peeling off with the pocket knife seem to be the most frequent initiative factors.

8. *No Known Factor.*—Some 20 to 30 per cent of carcinoma of the hand appear without any known previous preceding lesion or causative factor.

Carcinoma of the hand appears usually but not exclusively on the dorsal surface of the hand either as a flat indurated ulcerating lesion, or as a fungating cauliflower-like mass. Its development in areas of x-ray dermatitis cannot always be detected before surgery. The carcinomatous lesions here tend to be harder and more indurated than the lesions elsewhere, they are frequently ulcerative and many are fungating. However, a careful study of skin removed because of irradiation dermatitis, will frequently reveal many foci of carcinoma-*in-situ* producing little evidence of their presence.

Local invasion and lymphatic involvement may occur quite early in carcinoma of the hand but usually are later manifestations. Seldom does one see a rapidly invasive carcinoma of the hand. Enlargement of the epitrochlear and axillary lymphatics may be due to infection, which is always an element in roentgen dermatitis with carcinoma or any ulcerating or fungating tumors. If regional lymph nodes are enlarged, and at least 50 per cent will be found to be so, they must always be assumed to be involved by carcinoma. In the absence of palpable enlargement and particularly if the tumor is of low grade malignancy, the glands are practically never invaded.

Carcinoma of the hand is almost always squamous cell, but may be basal and is very rarely sweat gland carcinoma.

Treatment consists in wide local removal of the lesion and surrounding skin. Nail bed carcinomata, which tend to be somewhat more malignant than others, are best treated by amputation of the digit. Where local invasion has occurred wide resection must be carried out. It is often possible to carry this out in such a way as to leave functioning elements on the hand, but it occasionally proves necessary to amputate the hand.

The question of lymph node dissection is not always easy to settle. In the early cases of roentgen carcinoma it is not my practice to carry out axillary and epitrochlear dissection as routine. If there is deep extension however, and particularly with bone invasion, lymphatic dissection seems indicated even if the nodes are not palpable. In the presence of palpable nodes, dissection is always indicated. Because of its degree of malignancy one should carefully consider axillary dissection when the nail bed is the site of carcinoma.

Roentgen dermatitis offers a field for the prophylaxis of cancer of the hand. Complete removal of all involved skin and the immediate replacement with split or in early cases free full thickness grafts, rids the hand of this source of danger.

The prognosis is very good for carcinoma of the hand. Statistical studies show cure rates of 40 to 70 per cent.

Melanoma

The ordinary *mole* is not often seen on the hand, but if present it should be excised with wide margin. The hand is very subject to trauma and these lesions, if they become malignant following irritation, are so vicious, that they should never be tampered with. They should not be subjected to biopsy by cutting out a section but should be widely removed in their entirety. The use of cautery, electrodissection, or irradiation is to be deplored.

Melanomas of the hand occur most often in the nail bed where they are known at Hutchinson's melanotic whitlow. Unfortunately these are frequently not recognized as melanomas but treated as infection, cancers, exostoses, warts, fungus infection, et cetera. Unfortunately too, they may not be deeply pigmented or may be without pigment although Hutchinson called our attention to the fact that the periphery of the unpigmented lesion is usually black or at least shows evidences of pigmentation.

As melanomas go they represent a better prognosis than any other melanomas, probably because they can be diagnosed early, and because the offending digit is amputated early.

Treatment consists in amputation of the involved finger followed either at once or within three to four weeks by epitrochlear and axillary node dissection.

Subungual Tumors

Various other subungual tumors which may occasionally be confused with melanoma include carcinoma, glomus tumor, exostosis, warts, fibroma, and in rare instances metastatic tumors.

Fibrous Tissue Tumors

Fibromata of the hand occur in a number of locations including the nail bed. They usually form isolated solitary nodules but occasionally appear lobulated. Sometimes they project as pedunculated mass from the skin. They may be movable or fixed, hard or occasionally soft and are usually painless. They grow slowly and cause trouble only because of size. They may be confused with neurofibromata and vice versa; both on examination and microscopically. They are occasionally found in tendon sheaths or on tendons although here one should be certain the tumor is not actually a xanthoma. Periosteal fibromas have been described. The early nodule in Dupuytren's contracture is often mistaken for a fibroma.

Fibromata do not recur after removal which is usually rather easily accomplished in case of the superficial tumors. The deep ones however, present a problem in dissection.

If there is a recurrence after removal of what appears to be a fibroma on gross and microscopic section, the tumor is probably the so-called recurring fibroma which should probably really belong in the group of sarcomas.

Fibrosarcomas present a somewhat perplexing group of tumors variously classified and confused with the neurosarcomas. It is apparent, however, that there are various varieties of fibrosarcomas; some undoubtedly taking origin in connection with nerve sheaths, others with no such apparent association. Some grow with great rapidity, others grow very slowly or not at all for many years and then take on a rapid accession of growth. They may start out as small isolated subcutaneous nodules in the skin (rarely) or in the subcutaneous tissues (more often), or they may take origin very deeply from intermuscular septa and other fascial planes. Other sarcomas may be hard, fixed and painful, and evidence rapid growth with early invasion of blood vessels and pulmonary metastases.

Two main pathologic types are described, the spindle cell and the round cell. Either may be rapidly invasive and prove fatal quite early.

One recurrent fibrous tumor has been noted in an earlier paragraph as a recurring fibroma which might possibly be better classed with the sarcomas. This tumor is usually diagnosed as a fibroma which is not too well circumscribed but which occurs promptly after what appears to be adequate excision. This process continues after successive removals and the diagnosis microscopically will change possibly to neurofibroma or neurosarcoma. The process will eventually require extensive excision and often amputation, although distinct metastases are rare.

As the cellularity increases the tumors become more and more malignant. Early removal of any tumor of the hand is advisable. For the sarcoma, wide excision or amputation offers best chance of cure.

Lipomas

The *lipomas* are a moderately frequent and usually rather typical tumor of the hand. They form slow-growing, usually well-encapsulated masses either superficially and then often on the dorsum or deep within the tissues where they form indistinct swellings most difficult to diagnose accurately. The superficial ones may be mistaken for epidermoid cysts or even xanthomas but may usually be suspected by their consistency. The deep masses have been divided into the epivaginal or those lying without the tendon sheaths and the intravaginal, those lying within the tendon sheaths. The epivaginal may press upon neighboring structures such as nerves or tendons causing some symptoms or they may infiltrate or push into muscle masses likewise leading to some interference with function. Ordinarily there is little disturbance other than that caused by size or location due to mechanical interference with use of the part by the obstructing mass.

The intravaginal lipomas are unusual and rare tumors which follow the tendon sheaths. They usually appear in the palm where they may be easily mistaken clinically for tuberculous tenosynovitis. Others may lie deep within the muscles, may infiltrate them and produce a very confusing clinical picture seldom diagnosed before operation. These intramuscular lipomas may occasionally lead to rupture of tendons. An occasional one may become malignant.

The removal of the tumor is not difficult in case of the superficial ones. The deep tumors may present difficulties in dissection. The arborescent lipomas may require extremely wide exposure and a careful dissection similar to that required in removal of a tuberculous tenosynovitis. The deep intramuscular tumors may present out-growths resembling the sproutings of potatoes and cannot be simply shelled out. The perineural lipomas may completely bury a nerve which may be easily traumatized in removal.

The prognosis is obviously favorable except in the rare instance of a liposarcoma of which only a few have been reported at any location on the body—the hand being extremely rarely involved.

Tumors of Joint Capsules and Tendon Sheaths

The tumors taking origin from the joint capsules and tendon sheaths are represented by the most common and by some of the rarest of hand tumors.

The *ganglion* has been intensively studied over the years and its exact nature is still somewhat obscure. Some believe that it is a degenerative process occuring in the dense tissue of joint capsules and tendon sheaths and that it owes its origin to trauma. The traumatic factors leading to its development are thought to be of chronic recurring nature. Those who support this idea point out its frequent occurrence in people who use their hands for frequently repeated motions, e.g., violinists, typists, pianists, et cetera. On the other hand, it is found frequently in people whose hands are not used in this manner. In the early days, when tuberculous tenosynovitis was looked upon as a compound carpal ganglion there was some tendency to ascribe the simple ganglion to tuberculosis. The French authors earlier in the century, prepared a pretty good case for such an assumption, which has no supporters today. The older idea that the ganglion was due to herniation of the synovial lining of joints and fibrous tendon sheaths is no longer held. Actual communications between joint or tendon sheath and the ganglionic cavity cannot be demonstrated in operation.

King, of Australia, has studied tumors coming from joint capsules and tendon sheaths and his conclusions seem most logical and correlate well with clinical observation. King feels that ganglia are in fact tumors which spring from tissue entering into the formation of joint capsules and tendon sheaths, synovial membranes, and the fibrous tissue about them. King feels it is hardly correct to call them degenerative processes but prefers to look upon them as actual tumors whose product, the cyst (early multiple and late single cystic spaces) is an expression of the cells from which the growth is derived; in other words they are synovial tumors.

Ganglia are seen most often on the dorsal surface of the wrist where they take origin usually from the capsules of the intercarpal joints. They are not infrequently seen in the anatomic snuff box and their deep origin here is in close anatomic relation to the radial artery, which may be damaged during operation for removal. On the volar surface of the wrist they are often seen in the region of insertion of the flexor carpi radialis tendon and the tissue of origin here seems to be the fibrous sheath of the tendon and the underlying joint capsules. Also, at this area, the ganglionic tissue or parts of it may lie deep to the thenar muscles and may escape detection, one explanation for recurrence. Another typical but often not recognized site for ganglia is the fibrous tendon sheath of the flexor tendons over the proximal phalanges. These small pearl-like tumors are frequently mistaken for sesamoid bones. Ganglia are also seen springing from other joints of the hand. The synovial or mucoid cyst of the distal phalanx, to be described below, while similar to ganglion appears to take its origin from the derma rather than joint capsule.

Symptoms of ganglia are largely due to their size and location. They produce cosmetic blemishes, are painful and tender as would be any hard mass in the tissues and may interfere with the use of the hand. The tiny volar digital ganglion at the base of a finger may interfere with grasp. At times, a ganglion may produce symptoms of pain and tenderness of the wrist before a mass becomes evident. Diagnosis may then be difficult. Recurrence after operation may be heralded in by the onset of pain and tenderness after a period of relief from trouble.

Treatment is excision. This is not the simple little operation that it might seem to be. There is no trick in removing the main cystic mass, although this is not easy to accomplish without rupturing it. The difficulty is in following the basal tissue down to its site of origin and removing all of the involved tissue. Tiny cysts may be found scattered throughout the thickened tissues from which the large cyst arises but otherwise the distinction between surrounding normal tissues and tumor is very difficult. Complete removal will entail excision of a segment of joint capsule or fibrous tendon sheath. If this is not done recurrence can be anticipated, and may even take place when a supposedly complete excision has been accomplished. The operation may be done under local anesthesia and should always be done in a bloodless field secured by the use of a blood pressure cuff. The incision for removal of volar carpal, volar digital and dorsal carpal ganglia should be transverse so as to avoid the contractile scars which are sure to occur following longitudinal incisions.

The wrist or involved digit is splinted for a period of approximately ten days following operation.

Methods of treatment other than operative excision, such as rupturing by a blow, aspiration and injection, carry a high percentage of recurrences.

The *synovial* or *mucus cyst* resembles very much the ordinary ganglion. It occurs close to the nail at the distal interphalangeal joint and may be mistaken for Heberden's nodes. They differ from ganglia in origin, appearance and behavior. Many appear to be derived from the subcutaneous tissues or the cutis and not from joint capsules. The skin overlying them is very thin and almost translucent so that one can almost see the greyish mucoid contents. They recur after excision with the greatest of rapidity unless excision includes the overlying skin. Pressure of the growth on the nail bed leads to broad grooves on the nail.

Treatment consists in complete excision. Here it is necessary to remove the skin and the tumor, since it appears likely that the tumor may well arise from the lower layers of the derma. The defect thus left must be filled in by a small skin graft. Recurrence is unlikely with this type of removal but is very frequent with less complete procedures in which the skin is saved.

The *xanthoma* (giant cell tumor of tendon sheaths, benign synovioma, et cetera) occurs as an isolated tumor on the hand, occasionally as a manifestation of generalized xanthomatosis. There is some question whether or not this is a true tumor, although actual cholesterol metabolic disturbance is not present in case of the solitary masses as it is in the generalized process.

The histologic pathology of the tumors is typical but variable both from tumor to tumor and from one area of the tumor to another. The tumors are characterized by intermingled areas made up of large multinucleated foreign-body type giant cells, so-called foam cells filled with cholesterol and giving the tumor its yellowish appearance because of the dissolved coloring matter in the cholesterol, areas of spindle shaped endothelial cells and areas with hemosiderin deposition. Various spaces within the tumor which may resemble vascular spaces, are thought to represent synovial spaces. These varying areas are recognizable in the gross specimen by the variably colored lobules (yellow, reddish brown, grey) which present themselves. The tumor is not a sarcoma even though it is often diagnosed as such.

These tumors occur on the volar, lateral, or dorsal surface of the digits, where they form firm evidently encapsulated and lobulated masses which may stretch but do not involve the skin overlying them. They are also seen in the palm or wrist, always in association with joint capsules or tendon sheaths. They are rather firmly attached and do not move on the underlying tissues. They are painless and are not tender, and except for actual size and cosmetic appearance cause little disturbance. They may be confused with the epidermoid cyst although this latter is not lobulated and it is usually possible to palpate lobules in the xanthoma.

Treatment of the xanthoma is surgical removal which must be done carefully in a bloodless field, both to ensure removal of the entire tumor and to avoid damage to nerves and blood vessels. The digital nerves and vessels will be found to be deeply embedded in between lobules of the tumor to lie flattened and thin on its surface. The tumor is easily recognizable when it is exposed, the areas and lobules of reddish black, yellow, orange, brown and grey are immediately striking. As the tumor is shelled free from its bed the surgeon must be very careful to follow all of its lobular proturberances and to expose and remove every trace. The tumor may be found to take origin from the joint capsule and its site of origin must be excised. Not infrequently a small prolongation of the tumor will be found to penetrate the digital tendon sheath and lobules of the mass must be followed into the sheath and removed.

The prognosis is good since these tumors are always benign but the recurrence rate is likely to be high unless great care is taken to follow and remove every bit of the tumor mass and particularly the basal tissues from which it springs. Very rarely, and then usually only after a number of recurrences, the histology may change and a sarcoma may develop. Occasionally a digit will require to be sacrificed not because of malignancy but because of destruction of blood supply subsequent to the dissection required to remove the tumor growth.

The *synovioma* is a not too uncommon sarcoma springing from tissues of the tendon sheaths, joint capsules and bursae. These tumors are made up of spindle cells with a variable amount of collagen; mitoses are not common. They occur as painless, slow growing masses near joints which are not especially striking at first appearance. When uncovered they may be encapsulated or diffuse, solid or fleshy tumors, occasionally even cystic with areas of hemorrhage or even of butter yellow tissue. They may be very vascular.

The diagnosis may be suspected expecially if the origin from joint capsule is determined. The history of injury is occasionally elicited, but the significance is not clear. It is an actively malignant and metastasizing tumor which promptly recurs after simple excision. Amputation is the treatment of choice.

Tumors of Blood Vessels

The *vascular* tumors of the hand, as those elsewhere, are relatively frequent. They vary from the simple telangiectatic angiomas to the diffuse and progressive arteriovenous or racemose aneurysms. There is some reason for doubting whether most of them are in fact tumors. Mont Reid from his extensive experience with them, concluded that the majority, if not all, were essentially arteriovenous communications of varying extent and degree. Most of them can be correlated with the various stages of development of the vascular tree, which embryologic study has been so beautifully depicted in the studies of Sabin. Thus we have the angioblastic type, which is made up apparently of cells similar to the primordial angioblastic cells from which the blood vascular system develops. They occur as angioblastic cords, which, by proliferation, may compress normal blood vessels. Other of the tumors are reminiscent of the capillary plexus stage, other of the retiform stage and still others of the stage of stem formation.

Clinically they are classified into various groups which tend to merge into each other and to be composed of mixed elements of various types.

The *capillary or* telangiectatic type occurs as a tiny vascular spot or as the port wine or strawberry nevus. These not infrequently are associated with areas of cavernous tissue to produce sponge-like masses in the subcutaneous tissues or even deeper, covered with telangiectatic skin. These tumors may be destructive by the simple mechanical pressure of their expanding vessels and sinuses but are not malignant. Unfortunately some of these mixed capillary and cavernous tumors may take on the character of arteriovenous aneurysms in later life and spread out over large areas—a good reason for eradicating them early.

The *cavernous* angioma is made up of large vascular sinuses, usually containing venous blood which lie deep to the skin and tend to infiltrate or push aside neighboring tissues. They may extend from volar to dorsal surface of the hand, infiltrate and replace muscles and take over the vascular elements of associated nerves and tendons.

The *arteriovenous aneurysms* frequently involve a certain area of vascular supply such as for example, a digit or several digits. They differ from the traumatic arteriovenous aneurysms in that there are multiple vascular communications throughout the whole involved area. It is not possible to eradicate them by tying a single arteriovenous communication. They form one of the most difficult to treat of all the vascular tumors of the hand.

The capillary and cavernous angiomas are usually amenable to surgery but may require sacrifice of skin and immediate replacement. The capillary type is amenable to x-ray therapy but one is always hesitant to use irradiation on the hand because of the skin damage it may cause. Also, since many of these tumors occur in young people, one would avoid irradiation for fear of the damage which might occur to the growing bones. Injection therapy of the cavernous angioma is recommended by some but I have had no experience of it. Treatment of the arteriovenous (or racemose or cirsoid) tumors is a very difficult problem. The direct attack upon the tumor mass itself is a most heartbreaking experience. It is practically an impossibility to discover and ligate all or even a fair number of the communications. The structure of the hand is such that searching for them is impossible without absolutely wrecking the hand. One can hardly speak of brilliant success in eradicating the tumor but we have helped these patients the most by measures directed toward diminishing the blood flow to them. Ligation of the arteries in stages combined with partial excision while not perfect seems to offer best chances of help. When a single digit is involved amputation is often the best solution.

The *telangiectatic granuloma,* also known as the pyogenic granuloma, is considered by some to be a true angioma, although others think it is simply granulation tissue tumor of infectious origin. It is seen as a mushroom-like growth of what appears to be granulation tissue. The "stalk" of the mushroom pushes up through a small opening in the epidermis which forms a thin collar about the neck of the tumor. Apparently many of these tumors disappear following roentgen therapy, others disappear spontaneously under pressure dressings continued for many weeks. A more certain and rapid method is to excise them and suture the small elliptical defect in the skin. They do not tend to recur.

The *glomus tumor* takes its origin from the neuromyoarterial glomus found as normal heat and circulatory regulating bodies in the skin. These glomi are especially numerous on the hand especially in the nail bed, but are found in varying numbers in practically every area of the body. The glomus or neuromyoarterial glomus is an arteriovenous short circuit found only in warm-blooded animals and is especially abundant in the feet of ducks. Fine arterioles directly connecting with venules form the canal of Suquet-Hoyer. The vessels are surrounded by epitheloid cells which may be modified smooth muscle cells. Many myelinated and non-myelinated nerve fibers are to be found coursing through this structure. The tumor which develops from these bodies evidences the same make-up as the normal glomus and there is some question as to whether this is a true tumor or a hamartoma.

Glomus tumors appear typically in the nail beds where 50 per cent of them are to be found. They appear however in many locations on the extremities, trunk and even rarely on the head and neck. They are usually single, may however be multiple. They are ordinarily 0.5 to 2.0 cm in diameter but occasionally a very large one is found.

They produce a rather typical symptomatology. They develop apparently rather insidiously to produce small painful nodules or painful spot on an extremity usually under a nail where they may be visible as reddish or purplish discolorations. They may change color with temperature change; may be visible at times and invisible at other times. When in the nail bed the overlying nail is raised or rounded so that when the finger is seen "end on" it has a circular shape rather than the semicircular shape typical of a finger tip.

The area is usually always tender but is more so at some times than at others and as time goes on its periods of sensitiveness increase. Sometimes it is worse in cold weather, at other times warmth makes it worse. Because of the tenderness the patient hesitates to use the digit, keeps it flexed in his palm or keeps his hand in his pocket. Paroxysmal attacks of radiating pain beginning in the tumor pass up the forearm and arm and occasionally even into the neck. A Horner's syndrome has been observed occasionally.

The diagnosis is usually suggested by the typical history and the finding of a tender spot in the area complained of. Occasionally no tumor can be seen, or sometimes warming or cooling of the hand will bring out the color. The rounding of the overlying nail in case of subungual glomus tumors has been noted above. X-ray of such a finger tip will occasionally reveal a depression of the distal phalanx. Similar tender spots may be caused by leiomyomas, and rarely by fibromas and neuromas. The amputation neuroma of course should never be confused with the glomus tumor.

Treatment consists in removal of the tumor. They usually shell out quite easily from the surrounding tissues. The subungual ones require removal of the nail which grows back readily. When operation is carried out under local infiltration anesthesia, the tumor may suddenly pale out as the anesthetic agent is injected, and the tumor may be difficult to locate. Under these circumstances, it resembles a small fibrous tumor.

The glomus tumor does not recur when removed completely and has never been reported to have become malignant.

Lymphangiomas are quite rare tumors of the hand, occuring as congenital thickening or soft swelling involving large areas of the hand and forearm. They are of slow growth and are not malignant. It has been advised to excise them, although their usual extent would often make this a difficult procedure.

Tumors of Bone

Tumors of the bones of the hand are not frequent except for the enchondromas. The *enchondroma* is rather typical of the hand, it being seldom seen elsewhere, even in the bones of the foot. The tumor occurs particularly in the shafts of metacarpals or proximal phalanges, never in the carpal bones. They may be multiple and it is usually well to x-ray both hands as well as other parts of the skeleton. In our experience, however, they are almost always solitary.

They develop as slow, usually painless swellings of the shaft of one of the tubular bones of the hand, usually near the head of a metacarpal or base of a proximal phalanx. There may be some dull aching pain to call attention to the tumor, but not infrequently the tumor is first recognized in x-rays taken because of a fracture which has followed rather minimal trauma.

X-ray examination reveals a vesicular area of rarefaction in a fairly well-circumscribed region of the bone. The cortex of the bone will be seen to be expanded and very thin but is intact except in case of fracture. The tumor may resemble a giant cell tumor, somewhat, in x-ray although the lateer shows a more soap bubbly appearance.

The enchondroma is benign and requires only thorough currettage. If the patient is seen with a fracture, the fracture is first treated by immobilization on a splint until healing occurs. This follows about the same course of healing as any other fracture, although the cortex remains wafer thin even after healing.

The bone is exposed, preferably where it is thinnest, and after stripping back periosteum for a limited area, the cortex is bitten away over an area large enough to admit a small curet. The pearly white contents of the tumor are then removed. A surprising amount will be found. The surgeon should carefully explore the whole cavity until he is able to bring out no more tumor substance. It is not necessary to fill the cavity thus made with bone chips or a bone graft. It is soon filled with clot which quickly organizes, new bone quickly forms and the thinned-out shaft thickens.

The prognosis is good in that these tumors do not recur if they are thoroughly removed and they are not malignant.

Exostoses appear from time to time on the hand, usually in the neighborhood of joints at a point of tendon insertion. They may be multiple. In the x-ray the tumor appears smaller than gross examination would indicate since they are usually capped with cartilage. They are occasionally encountered in the nail bed, the so-called Dupuytren's subungual exostosis, which pushes up the overlying nail and erodes through it, leading to the development of a granulomatous lesion.

The *osteoid osteoma* was established as a definite entity by Henry L. Jaffe in 1940. It occurs with moderate frequency in the bones of the hand, where Robert Carroll has reported twenty-eight cases from the literature and his own service. Previous to Jaffe's reports, this tumor was confused with other areas of bone rarefaction and sclerosis such as localized osteomyelitis, bone abscess, lues, et cetera. It must still be differentiated from these conditions. It occurs as a small transluscent area in the spongy bone or cortex surrounded by denser areas and thickening. The central area or nidus is composed of osteoid tissue about which is found sclerosed bone.

Clinically the tumor is characterized by the development of chronic naggy nocturnal pain which may occur a year or more before the tumor can be recognized in the x-ray. The pain is not severe and usually responds to small doses of aspirin. X-ray examination may have to be repeated from time to time and in various positions, since the tumor can be easily missed, or may not show up in its early stages. It may be mistaken on X-ray examination for abscess, lues, osteochondritis dessicans, xanthoma or even callus. The radiolucent center and associated cortical thickening are the characteristic features.

The tumor, if left alone, may gradually run its course, reach a peak and over a number of years subside as reported by Moberg.

Treatment consists in removal of the tumor mass. The nidus or soft radiolucent center must be removed entirely else recurrence is certain to follow.

Giant cell bone tumors do not often occur on the hand. They have been reported in the metacarpals where they develop in the epiphysis as slow growing tumors causing boring pain more severe at night. The appearance in x-ray is fairly characteristic and can usually be differentiated from enchondroma by more definite fine trabeculation. Treatment is surgical removal. Usually the involved bone or section of bone requires removal and may be replaced at once by a bone graft. Curettage of the current-jelly-like contents of the tumor may occasionally prove curative.

Bone cysts, myxomas, osteomas, bone fibroma and hemangioma have been described in the bones of the hands. Ewing's tumor may also involve the bones of the hand, as a diffuse process, which may be confused with chronic diffuse osteomyelitis.

Bone sarcoma is of great rarity on the hand. Five only are recorded in the literature.

Metastatic tumors of the bones of the hand are likewise rather rare but have been reported from primary tumors of the lung, prostate, testis, breast, parotid, endometrium, kidney, colon and adrenals.

Tumors of Nerves

Tumors of the peripheral nerves occur clinically as four different types—isolated nodules (single in 95 per cent of instances), diffuse spindle-like enlargement of a nerve, plexiform thickening of all the nerves in an area, and as von Recklinghausen's disease.

The *nodular neuromas,* neurinomas or neurofibromas are proliferation of elements of the nerve sheath and usually lie subcutaneously in association with fine rami of sensory nerves. They are usually painless and may be multiple over the whole body. They cause few symptoms and are easily excised without any disturbance of function.

The *fusiform swellings* of a peripheral nerve, in our experience most often affects the median in the lower forearm and wrist, and may extend into the palm and involve the terminal rami of the median in the palm. Although it is said that these tumors often simply expand the nerve which lies spread out and thinned over its surface, in our experience of such tumors such has not been the case. The tumor tissue and the nerve fibers are so inextricably interwoven that it is impossible to separate them and removal of the tumor entails excision of the entire thickened nerve.

The *plexiform neuroma* produces also a thickening of a nerve or group of nerves in an area. However, this thickening extends into the finer branches to the terminal twigs in the skin. The skin itself and the subcutaneous tissues are thick and nodular and there may be gross distortion of a digit or part of a digit or several digits or of the whole hand. This condition is spoken of as *elephantiasis neuromatosa.* Complete removal of all tissue would often lead to severe mutilation which in view of the benign nature of the process is not necessary. The appearance of the hand may be considerably improved, however, by excision often associated with skin grafting.

von Recklinghausen's disease is seen involving the hand as it does other parts of the body. Its importance lies in the fact that in about 15 per cent of cases sarcoma develops.

Neurosarcoma or neurofibrosarcoma starts usually as a small nodule which promptly recurs after excision. In some instances these recurrences seem to be relatively benign and the surgeon is tempted to carry out rather conservative though wide excision. Some have been followed over a period of many years, eventually requiring amputation. Others, however, are extremely malignant and prove rapidly fatal. The initial operation is the most important one and should be radical and extensive.

Résumé

In résumé, we have gone over very briefly the tumors of the hand as they have presented themselves. There is a great variety of such tumors, many quite common, others quite rare. About 12 per cent of hand tumors are malignant and of these 90 per cent are squamous cell carcinoma, of which well over half are squamous cell carcinoma developing on the basis of irradiation dermatitis. Irritation of one type or another plays a definite role in the development of tumors of the hand, especially in the case of carcinoma. This fact points toward prophylaxis in this condition. The ease of examination of the hand makes for early diagnosis and early surgery for good prognosis. The anatomic structure of the hand may make adequate exposure and removal difficult.

Minnesota Academy of Medicine

Meeting of November 11, 1953

The regular monthly meeting of the Minnesota Academy of Medicine was held at the Town and Country Club on Wednesday evening, November 11, 1953. Dinner was served at 7 o'clock and the meeting was called to order at 8:15 p.m. by the President, Dr. E. A. Regnier.

There were fifty members and one guest present.

In the absence of the secretary, the Minutes of the October meeting were omitted.

Upon ballot, the following men were elected as candidates for membership in the Academy:

Robert Tenner, M.D., Minneapolis
Harold Buchstein, M.D., Minneapolis
Wallace Armstrong, M.D., University of Minnesota

Dr. Ohage moved that, inasmuch as the May meeting of the Academy always conflicts with the Annual Golf Tournament held at the Town and Country Club, the May meeting in 1954 be held at the Minneapolis Club in honor of the present President, and that on alternate years the meetings be held at the Minnesota Club, replacing the regular January meeting. Motion seconded and carried.

The scientific program followed.

Dr. David Anderson, of Austin, then read his Inaugural Thesis on "Non-operative Treatment of Acute Perforated Peptic Ulcer." Lantern slides were shown.

THE NON-OPERATIVE TREATMENT OF ACUTE PERFORATED PEPTIC ULCER

DAVID P. ANDERSON, M.D.

Austin, Minnesota

The treatment of acute perforated peptic ulcer was considered a settled matter until 1945. It was agreed that the only logical treatment was to operate, and the sooner the better, in order to surgically close the perforation. The few surgeons who performed a gastroenterostomy in addition to closure of the ulcer had been overruled by 1945, because of the increased mortality and the high incidence of late marginal ulcer which followed gastroenterostomy. The advocates of primary gastric resection for the treatment of acute perforated peptic ulcer were in the minority. It was generally accepted and taught that in dealing with an acute perforation of a peptic ulcer, it is the surgeon's duty to save the patient's life, and not to attempt to cure the ulcer. This viewpoint was emphasized forcefully by Dr. Roscoe Graham, whose simple suture technique for the closure of a perforated peptic ulcer was widely used. Significantly, Doctor Graham also demonstrated that peptic ulcer perforations of twenty-four hours' duration or longer hardly needed an operation, except for any such late complications as localized intraperitoneal abscesses. In addition, Doctor Graham offered convincing proof that it was worthwhile to delay operation a few hours, if necessary, to adequately prepare the critically ill, debilitated, or aged patient with an acute perforated peptic ulcer.

Taylor, in 1945, and later Seeley, disturbed the prevailing calm of surgical thought in regard to the treatment of acute perforated peptic ulcer. Prior to the reports of Taylor and Seeley, a few patients here and there had been inadvertently treated non-operatively for acute perforated peptic ulcer, and Wangensteen had given a deliberate non-operative regime some serious thought as early as 1935. However, it was Taylor's report of a series of selected patients deliberately treated without operation that actually started the new trend of thought on this subject.

Reasoning that a completely empty stomach, like a deflated balloon, will not leak, the advocates of the non-operative treatment of acute perforated peptic ulcer empty the stomach and keep it emptied. The initial soiling of the peritoneum, which is largely chemical rather than bacterial during the first few hours, is treated with antibiotics. Fluids, of course, are administered parenterally and narcotics are used for the relief of pain. Sealing-off of the perforation is left to the natural reparative processes. As all experienced surgeons know, the sealing of a site of perforation does occur quite rapidly, often within a matter of a few hours. If the stomach is kept deflated, the natural reparative processes can be depended upon to firmly seal a perforated peptic ulcer.

Dr. Robert R. Wright of the Austin Clinic treated our first patient by Taylor's non-operative method in April, 1947. Since that time we have treated thirty-seven patients with acute perforated peptic ulcer by the non-operative method. We have been so favorably impressed with the advantages of this method over the operative management that we believe a report of our experiences is justified.

Criteria for Accepting the Diagnosis of Acute Perforated Peptic Ulcer in Patients Treated Non-Operatively

The statement that we have treated thirty-seven patients with acute perforated peptic ulcer by the non-operative regime cannot be accepted as a statement of fact without further proof. It is a fact, attested by the records of the patients reported, that a final diagnosis of "acute perforated peptic ulcer" was made for each patient, and the results of the treatment are a matter of record. It remains to be proved that these patients did, in fact, have acute perforated peptic ulcer rather than some illness simulating this disease. Direct proof of the actual existence of an acute perforated peptic ulcer, such as one can obtain at operation, is naturally not available in patients treated non-operatively. It behooves us, therefore, to be very strict in selecting patients, who were treated without operation, that we report as having had acute perforated peptic ulcer. So-called "perforating" or "penetrating" petic ulcers must be excluded, as well as other diseases which commonly simulate acute perforated peptic ulcer.

We feel that there is no reasonable doubt that twenty-three of the thirty-seven patients diagnosed as "acute perforated peptic ulcer" did, in fact, have the disease. Each of these twenty-three patients fulfilled the following rigid standards that we have established for ourselves as proof of the actual existence of a frank, acute perforated peptic ulcer in patients treated non-operatively:

1. A clear-cut clinical history of acute, severe abdominal pain.
2. Physical findings of diffuse, board-like rigidity of the abdomen; tenderness; and rebound tenderness.
3. X-ray proof of a gastric or duodenal ulcer *with an active crater.*
4. Absence of early or late evidence of other diseases which might simulate acute perforated peptic ulcer.

The treatment of each of these twenty-three patients considered to have proved acute perforated peptic ulcer was supervised by the author, or one of his surgical associates in the Austin Clinic. Confirmation of the clinical and x-ray diagnosis was aided by other relevant findings:

1. Twenty-two of the twenty-three patients had definite peptic ulcer symptoms for a few days to several years prior to the onset of the acute abdominal catastrophe. Fifteen of these had definite x-ray proof of chronic peptic ulcer.
2. Eight of sixteen patients on whom an upright or left lateral decubitus x-ray was done had free gas in the peritoneal cavity. Only 5 per cent had free gas.
3. Three patients in this group have had late elective operations for peptic ulcer (gastric resection or gastroenterostomy with vagotomy) performed by the author. Seven others have received medical treatment at the Clinic for recurrent peptic ulcer.

We have classified the remaining fourteen patients, who were diagnosed "acute perforated peptic ulcer" and treated non-operatively, as *"probable"* acute perforated peptic ulcer. We feel that an unequivocal diagnosis cannot be made in this group because of lack of personal observation of the patient, or because of lack of late x-ray evidence of an active peptic ulcer. However, each of the fourteen patients presented findings that were considered a typical picture of an acute surgical abdomen, presumably due to an acute perforated peptic ulcer. Operation ordinarily would have been advised for these patients.

TABLE I. FINAL CLASSIFICATION OF FIFTY PATIENTS WITH DIAGNOSIS OF ACUTE PERFORATED PEPTIC ULCER

23	"Proved" cases treated non-operatively.
14	"Probable" cases treated non-operatively.
1	Patient, initially treated non-operatively, operated on after seven hours.
2	Patients operated on initially.
5	Patients, treated non-operatively, reclassified as having "perforating" or "penetrating" peptic ulcer.
5	Patients, treated non-operatively, reclassified as having "other diseases."

In order to rigidly re-examine our evidence, we have reviewed the records of all patients with the final diagnosis, "acute perforated peptic ulcer," who were treated in the St. Olaf Hospital, Austin, during the period of this study (April, 1947, to October, 1953). There were fifty patients so diagnosed during this period. A final classification of the diagnosis in these fifty patients is given in Table I. This was made after re-evaluation of the hospital record, late x-ray examinations, and the patient's subsequent medical course.*

Method of Treatment

Our method of non-operative management of acute perforated peptic ulcer follows the basic principles outlined by Taylor, Seeley, and others who have pioneered this treatment. Certain minor modifications have been made. The regime is as follows:

1. *The stomach is completely emptied and kept emptied.*—Emptying of the stomach, of course, is absolutely essential. We have found that an ordinary Lavin tube is satisfactory for the initial aspiration of the stomach, as well as for the continuous gastric suction. The stomach is thoroughly irrigated with sterile normal saline and aspirated until we are certain that it is empty. Continuous gastric suction is then maintained for forty-eight to seventy-two hours, or longer, until any abdominal distention has been relieved, peristalsis is active, and all signs of peritoneal irritation have subsided. Extreme care is taken to be certain that the stomach tube is patent and functioning properly at all times.

After forty-eight to seventy-two hours, the stomach tube is clamped, but left in place, and oral feedings of two ounces of milk and two ounces of water are given every two hours while the patient is awake. The stomach contents are aspirated two hours after the oral feedings and the residual gastric content recorded. If the residual stomach contents are less than an ounce on repeated aspirations during a twelve-hour period, it is assumed that there is no pyloric obstruction and the Levin tube is removed. If pyloric obstruction persists, the stomach aspirations are continued until it subsides. After the Levin tube is removed the patient is placed on the usual medical regime for the management of an active duodenal ulcer. A stomach x-ray can be done safely, with a little caution on the part of the roentgenologist, on the seventh to tenth day.

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*Several hospital staff members who are not associated with the Austin Clinic have adopted the non-operative management of acute perforated peptic ulcer. The author wishes to express his appreciation to these men for the courtesy of being informed of or seeing their patients during the course of treatment: Drs. P. C. Leck; P. A. Lommen; C. L. Sheedy and Leo Twiggs.

2. *Narcotics are used for the relief of pain.*—Surprisingly, many of the patients in this series, who were admitted to the hospital with intense pain, required no narcotics after the first twelve to twenty-four hours of non-operative treatment.

3. *Parenteral fluids are administered until oral intake is adequate.*—The average patient requires parenteral fluids for the first two to four days only.

4. *Antibiotics* are administered prophylactically, usually for five days. A combination of 300,000 units of penicillin and 0.5 grams of streptomycin, administered twice daily, has been our favorite.

5. *Careful observation* at four- to six-hour *intervals* is mandatory during the first twelve to twenty-four hours. This observation should be conducted by a surgeon familiar with the management of an acute surgical abdomen. The surgeon should keep an open mind during this early period and be willing to reverse his opinion and recommend operation if progress is not satisfactory. We have made it a practice to inform the patient, his relatives, and the referring physician that the election of non-operative management is not final, but is subject to change after a preliminary period of observation.

Usually, after four to six hours, and almost invariably within the first twelve hours, the patient with an acute perforated peptic ulcer shows definite signs of improvement. Progression of symptoms or failure to improve during the early period of observation is considered an indication for surgical intervention.

Results of Treatment

The twenty-three patients classified as "proved" acute perforated peptic ulcer all recovered on a non-operative regime. There was only one complication in this group: a localized pelvic abscess which responded promptly to transrectal drainage.

One patient, placed on a non-operative regime by the attending physician, was operated on by the author after an initial observation period of seven hours. Operation was elected for the following reasons:

1. Lack of adequate response during a seven-hour period of observation.
2. The presence of barium in the stomach.
3. The presence of a known high-grade pyloric obstruction.

At operation a large pre-pyloric perforated ulcer was found. The perforation, which was 1 cm. in diameter, was completely sealed by omentum and there was little or no soiling of the peritoneum. A subtotal gastric resection was elected as the procedure of choice. The patient's recovery was entirely uneventful (Case 1).

Case 1.—P. C., male, aged sixty-five, was admitted to the hospital because of severe pain following stomach x-ray which showed pyloric obstruction.

Course.—A Levin tube was inserted on admission by the attending physician. The tube was irrigated "several times" with soda solution, but it did not work well. Sudden, severe abdominal pain developed fourteen hours after admission. The surgical consultant was called seven hours after onset of the pain.

Treatment.—An operation was elected because of: (1) pyloric obstruction, (2) presence of barium in the stomach, (3) difficulty in emptying the stomach with the Levin tube.

Operation.—A chronic pre-pyloric gastric ulcer with perforation 1 cm. in diameter was found. Perforation was sealed by omentum. Subtotal gastric resection was done. The recovery was uneventful.

Twelve of the fourteen patients diagnosed clinically as acute perforated peptic ulcer, and classified by us on review as "probable" cases, recovered without complications when treated non-operatively. *Two patients in this group died.* One of these patients was an eighty-five-year-old woman who died in the hospital after complete recovery from peritonitis which was presumably due to acute perforated peptic ulcer. Death was due to cerebral arteriosclerosis and arteriosclerotic heart disease with cardiac decompensation (Case 2).

Case 2.—L. S., female, aged eighty-five, had a history of a duodenal ulcer for many years. She had had an inactive ulcer with complete pyloric obstruction operated on five years previously (pylorplasty). She was admitted to the hospital with an acute epigastric pain of three days' duration.

Findings.—Generalized peritonitis, free gas beneath diaphragm, pneumonia; arteriosclerotic heart disease, and glaucoma were found.

Treatment.—A non-operative treatment was decided upon.

Course.—The patient recovered from peritonitis. She tolerated soft foods and had no abdominal distress for five weeks prior to death from arteriosclerotic heart disease with cardiac decompensation.

The patient expired forty-seven days after admission. No autopsy was performed.

The second death was that of a seventy-nine-year-old man who was admitted to the hospital with a massive hemorrhage from a chronic duodenal ulcer. Probably perforation occurred as a terminal event and was a definite factor in the patient's death (Case 3).

Case 3.—H. S., male, aged seventy-nine, had a history of duodenal ulcer for many years. He had epigastric pain and tarry stools for two weeks. The hemoglobin was 46 per cent. He had arteriosclerotic heart disease and auricular fibrillation.

Treatment.—He received transfusions and a special diet for seven days. Improvement was noted. The hemoglobin was 100 per cent.

Course.—Vomiting of undigested food and blood, with increased pain, started the eighth hospital day. A Levin tube was inserted on the tenth day for "gastric dilatation." Fecal impaction was noted. There was tenderness and rigidity in upper abdomen.

The patient expired on the thirteenth hospital day from anuria, cardiac failure and peritonitis. An autopsy was not obtained.

It is necessary to arrtibute this death statistically to the non-operative method of management, although there is a question as to the accuracy of the diagnosis. It should also be noted that the non-operative management was not ideal; that there were serious associated diseases; and that two surgical consultants felt that the patient would not tolerate even the simplest operative procedure.

The two patients operated on initially and the ten patients, treated non-operatively as acute perforated peptic ulcer but reclassified by us as "penetrating ulcers" or "other diseases," all recovered without serious complications.

Hazards of Non-Operative Treatment

The deliberate non-operative management of acute perforated peptic ulcer has certain potential hazards that should be forcefully stressed.

1. *Errors of diagnosis.*—An erroneous diagnosis of acute perforated peptic ulcer, when accompanied by a decision to treat the patient non-operatively, is a very real danger of this method of therapy. Some surgeons argue that this danger is so great that they will not consider the use of the non-operative regime under any circumstances.

We feel that an accurate diagnosis is possible in most instances, and that with certain safeguards, a brief delay for observation is not detrimental. On many occasions during the period covered by this report, we have considered the possibility of an acute perforated peptic ulcer in the differential diagnosis of an acute surgical abdomen. When operation was deliberately delayed for a few hours of observation, the correct diagnosis and proper course of treatment often became amazingly clear, and the patient benefited thereby. A necessary operation was delayed unduly because of the erroneous diagnosis of acute perforated peptic ulcer on only one occasion. This patient's history is summarized briefly in Case 4.

Case 4.—I. H., male, aged fifty-six, was known to have a duodenal ulcer with recent active symptoms. He was admitted to the hospital with severe abdominal pain of four hours' duration, shock and board-like regidity. White blood count was 20,000. An x-ray of the abdomen showed no abnormalties.

Course.—In six hours the patient was not improved. After twelve hours the pain was more severe, his condition was worse, the board-like rigidity were unchanged. After forty-eight hours the patient was toxic; the abdomen was tense and distended. On the third to seventh day he was improved; an LLQ mass developed.

Operation.—Abdomen was explored on the seventh day after I & D revealed old bloody fluid. Volvulus of ileum with seventeen feet of gangrenous bowel, resection of ileum.

The patient expired two and one-half months after the operation with a fecal fistula, toxic hepatitis, and uremia.

This one incident forcefully emphasizes, in the breach thereof, a firm rule that we have established in the non-operative management of acute perforated peptic ulcer: *Re-evaluation after two to four hours of non-operative management is mandatory. If the patient is not improved, or if the diagnosis is in doubt after four hours, operation is indicated.*

The initial election of non-operative therapy is not final, but is subject to change after a preliminary period of observation. We failed to follow this rule in the management of the patient discussed above. On two other occasions, we have evoked this rule when we operated on a known perforated pre-pyloric ulcer that was not improving, as cited above, and once when we operated on a suspected acute perforated peptic ulcer, after a preliminary period of observation, because of persisting pain and a change in the physical findings.

Case 5.—R. G., male, aged fifty-seven, was admitted to the hospital with severe abdominal pain of six hours' duration with board-like abdominal rigidity. The hemoglobin was 40 per cent. The patient had a history of indigestion and increasing weakness for one year. X-ray examination of the abdomen gave negative findings.

Treatment.—Gastric suction; transfusions and narcotics. General condition improved, but pain was severe, abdomen rigid and LLQ mass suspected after six hours' observation. An operation was advised.

Operation.—Leiomyosarcoma of jejunum 5 inches in diameter, with gross perforation and hemorrhage, was found. Resection was performed. The patient recovered.

2. *Improper management.*—Another potential hazard in the non-operative management of acute perforated peptic ulcer is the danger of poor supervision of the treatment. The simplicity of the method is likely to create a false sense of security until the physician learns, too late, that things are not going well. It must be understood that the non-operative regime is an active, dynamic method of treatment. It should *not* be entrusted to anyone who is not thoroughly familiar with the diagnosis and treatment of the acute surgical abdomen. It should *not* be elected as the treatment of choice unless the surgeon is willing to give the patient the same careful supervision that he would give a critically ill postoperative patient. The surgeon must know that the stomach is empty, and he must know that it will be *kept emptied* by the surgical personnel under his supervision. He must observe the patient carefully, and he must be willing to change his course of therapy if the indications arise.

Contraindications to Non-Operative Management

The following may be considered as *absolute* contraindications to the non-operative management of acute perforated peptic ulcer:

1. *An uncooperative patient.* An alcoholic, demented or uncooperative patient who will not leave the Levin tube in place should not be treated by this method. Complete and continuous emptying of the stomach is absolutely essential to the success of the treatment.

2. Perforation when the stomach is filled with barium should not be treated non-operatively for fear of chemical peritonitis due to barium, which is difficult to remove from the stomach with gastric suction.

(Continued on Page 622)

In Memoriam

GUSTAF EDLUND

Dr. Gustaf Edlund of Saint Paul, widely known as a physician and golfer, collapsed and died suddenly at his home, 3122 Owasso Boulevard, Saint Paul, June 10. He was sixty-two years old and, according to his family, had never been ill before in his life.

Dr. Edlund had practiced medicine and surgery for thirty-three years in Saint Paul with offices at 120 North Snelling Avenue. He was a former president of the Midland Hills Country Club and was a member in recent years of the North Oaks Country Club. He was said to have shot holes-in-one at least three times in a long golfing career which began in boyhood.

He was born in Saint Paul and received his advanced education at the University of Minnesota and Northwestern University in Chicago from which he received his medical degree in 1920. He served his internship at Miller Hospital and was a member of the professional staffs at Gillette State Hospital and Midway Hospital in Saint Paul.

Dr. Edlund was a member of the Ramsey County Medical Society, the Minnesota State Medical Association and the American Medical Association. He was also a member of Osman Temple of the Shrine and the Reformation Lutheran Church in Saint Paul.

Survivors are his wife, Helen; a son, Gustaf, Jr.; three brothers, John and Edward of Saint Paul and Fred of San Francisco; two sisters, Esther of Saint Paul and Elsie of Minneapolis.

FREDERICK WILLIAM ENGDAHL

Dr. Frederick W. Engdahl died unexpectedly of a heart attack at his home in Dallas, Texas, June 11. Until about twelve years ago, Dr. Engdahl had practiced at Ortonville with Doctors Charles Bolsta, B. R. Karn and D. M. O'Donnell. He was sixty-three years old.

Son of the Rev. and Mrs. A. Engdahl of Ortonville, Dr. Engdahl was born in Milbank, South Dakota, but went to school in Ortonville and then graduated in civil engineering from the Cincinnati, Ohio, University. He served for some years as highway engineer for Big Stone County before going to the University of Minnesota to study medicine. He served his internship at St. Mary's Hospital in Duluth and practiced at Mankato and Grand Rapids before going to Ortonville. He was a veteran of World War I and a member of the Minnesota State Medical Association and the American Medical Association as well as the West Central Minnesota Medical Society.

Surviving are his wife, the former Ruth Rishoff; a daughter, Mary Elizabeth, and son, Frederick W., Jr., both of Dallas; one brother, Theodore M. of Los Gatos, California; and two sisters, Mrs. C. A. Zweiner, Saint Paul, and Mrs. Luther Youngdahl, wife of Judge Luther Youngdahl, Washington, D. C.

CHARLES GEORGE FORREST

Dr. Charles G. Forrest died June 17 at the age of eighty-eight in his home at Clearbrook. He had been in virtual retirement for some years after more than forty years of active practice spanning the "horse and buggy days" at Clearbrook.

Dr. Forrest was born at Lake City and attended Pillsbury Academy in Owatonna before going to the University of Minnesota from which he was graduated in medicine in 1899. He practiced for a short time in Bagley before moving to Clearbrook in 1910 and had been honored as an outstanding citizen and civic leader, as well as beloved family doctor, by the community.

In 1949, Dr. and Mrs. Forrest had been chosen by the Minneapolis Aquatennial committee from hundreds of nominees as the "happiest couple in Minnesota." They were feted guests of the Aquatennial that year and received a trip to California as part of the celebration. At that time it was estimated that Dr. Forrest had delivered more than 3,000 babies during his long and eventful career as a country doctor and he had found time, also, to serve for forty years on the Clearbrook school board, and to act as local health officer and as a director of the First State Bank of Clearbrook. He maintained an active interest in church work, too, and kept an eye on the nearby Red Lake Indian reservation where he had many Indian friends.

In 1901, Dr. Forrest married Miss Rosetta Nichols, a teacher in the Bagley school, who survives him. Other survivors are a daughter, Mrs. George Hoppe of Seattle; and three foster daughters, Mrs. Henry Dahlberg of Clearbrook, Mrs. Frank Gambucci of Hibbing and Mrs. Nicholas Faymoville of Minneapolis. There are eight grandchildren.

ARNOLD PHILLIP GRUENHAGEN

Arnold Phillip Gruenhagen, Saint Paul surgeon, died after a long illness, July 6, at St. Luke's Hospital in Saint Paul. He was fifty-nine years old.

Dr. Gruenhagen was born at Shell Lake, Wisconsin, but came to Saint Paul as a child and received his preliminary education there. He took a B.S. degree from Hamline and the University of Minnesota and was graduated in medicine from the University of Illinois. After an internship and residency at Ancker Hospital in Saint Paul, he became associated in practice with the late Dr. A. E. Comstock and joined the staffs of Miller, Ancker and St. Luke's hospitals, becoming chief of staff at St. Luke's Hospital later. He served as a member of the teaching staff of the University of Minnesota medical school for fifteen years, and for twenty years he was a teaching staff member of Ancker Hospital.

Among his professional affiliations was membership in the American College of Surgeons of which he was a fellow, the American Medical Association, the Minnesota State Medical Association and the Ramsey County Medical Society. Also, he was a member of the Saint Paul Surgical Society, Alpha Kappa Kappa medical fraternity, Rotary and the Town and Country Club.

Dr. Gruenhagen had been ill and retired from practice for more than seven years. He is survived by his wife, the former Floy Brown; his mother, Mrs. Charles Zieman of Saint Paul; and one sister, Mrs. Langdon D. Strong of Milwaukee.

JAMES FELTON HAMMOND

Dr. J. Felton Hammond of Saint Paul, died August 10, 1954, at the age of seventy-two. He had practiced at the same location in the Lowry Medical Arts Building since 1910 and was a life member of the Minnesota State Medical Association and the Ramsey County Medical Society. He was found dead of a heart attack in his car in the garage at the rear of his home.

Dr. Hammond was born at Ironside, Quebec, Canada, and attended Ottawa Collegiate Institute and McGill University, from which he received his medical degree in 1906. After serving an internship at Montreal Maternity Hospital, he spent two years in postgraduate work at the Montreal General Hospital and the Alexandria Hospital in Montreal before coming to Saint Paul to specialize in obstetrics and gynecology in 1910. He had been an instructor in obstetrics and gynecology at the University of Minnesota and was a staff member at Ancker, St. Luke's and Miller Hospitals in Saint Paul. He was a member of the Minnesota Academy of Medicine as well as of Ramsey County Medical Society, the Minnesota State Medical Association and the American Medical Association.

Dr. Hammond is survived by his wife, Eleanor Jane, and four sisters and two brothers who live in Canada.

MELVIN S. HENDERSON

Dr. Melvin S. Henderson, founder of the section of orthopedic surgery at the Mayo Clinic, president in 1932 of the Minnesota State Medical Association and former associate of Drs. W. J. and C. H. Mayo, died at his home in Rochester, Thursday, June 17. He had retired from active association with the Clinic in 1948 and was seventy-one years old at the time of his death.

When Dr. Henderson went to Rochester as a young man just out of medical school and the University of Toronto, there were only three special sessions in the Mayo Clinic; one was for diseases of the eyes, one for diseases of the ear, nose and throat and one for genitourinary diseases. Following a year's internship at Ancker Hospital in Saint Paul (then the City and County Hospital) in 1907, he went to the Clinic and was given charge, at first, of the outpatient post-operative department. Soon he became first assistant to Dr. W. J. Mayo and, in 1910, he formed the first section devoted exclusively to orthopedic surgery. There were few such specialized sections anywhere at the time and Dr. Henderson was his own brace-maker until he could pick and train a man to undertake this exacting auxiliary to orthopedic surgery.

Subsequently he spent considerable time in Liverpool with the celebrated Sir Robert Jones and at Edinburgh and London studying the new specialty and became, himself, one of the most celebrated orthopedic surgeons in the world.

Dr. Henderson was born in Saint Paul, son of Mr. and Mrs. Melvin Brooks Henderson, and it was Dr. Archibald MacLaren of Saint Paul who gave him the letter of introduction which brought him to the attention of the Doctors Mayo.

He was an associate and then professor of orthopedic surgery in the Mayo Foundation Graduate School of the University of Minnesota until 1951. He was a member of the Board of Governors of the Mayo Clinic from 1924 to 1947 and vice chairman of the board from 1937 to 1946. He served as chairman of the executive committee of the Mayo Clinic from 1918 to 1920, and from 1937 to 1950 he was a member of the board of the Mayo Association.

Among many professional affiliations and honors, he was president of the Board of Orthopedic Surgery in 1934 and president of the American Orthopedic Association in the same year. Later he was elected president of the American Academy of Orthopedic Surgeons. Besides serving as president of the state medical association he was also president of the Southern Minnesota Medical Association, a member of the Olmsted-Houston-Fillmore-Dodge County Medical Society, the American Medical Association and a large number of other national and international organizations.

Dr. Henderson is survived by his wife, the former Mabel Christensen and two sons, Dr. Edward D. Henderson, a member of the section at the Mayo Clinic which his father founded, and Melvin Starkey Henderson, also of Rochester.

IRVING WILLIAM STEINER

Dr. I. W. Steiner of Winona, age sixty-six, died at Winona General Hospital after a short illness May 4, 1954. He was a former Winona County coroner, the former secretary for many years of the Winona County Medical Society and he had practiced medicine and surgery in Winona for thirty-five years.

Dr. Steiner was born at Independence, Wisconsin, and had his secondary education there and at Arcadia, Wisconsin. After two years at the Armour Institute in Chicago he entered pre-medical training at the University of Chicago and was graduated in medicine from Rush Medical School. After a year's internship at Presbyterian Hospital in Chicago he entered practice at Harvey, Illinois, and, a year later, joined the armed forces for service in World War I. He went to Winona immediately after the war to engage in general practice there.

Dr. Steiner served as coroner from 1923 to 1927 and joined the State Guard as major in the Ambulance Corps during World War II. He was vice president of the Minnesota Chapter of the American Academy of General Practice, a member of the American Medical Association, the Minnesota State Medical Association and the Southern Minnesota Medical Association. He was also a member of Rotary and a 32nd degree Mason as well as a member of the Elks, the American Legion, the Winona Athletic and Country

Clubs and St. Paul's Episcopal Church in Winona. He was a staff member of Winona General Hospital.

Surviving are his wife; a daughter, Susan of Winona; and one sister, Mrs. Donald Ruhnke of Canton, Ohio.

JOSEPH J. STRATTE

Dr. Joseph J. Stratte, formerly of Hallock, died suddenly in Minneapolis, April 9, while attending a continuation study course on urology at the Center for Continuation Study on the University campus. He was sixty-seven years old.

Dr. Stratte had practiced in Grand Forks, North Dakota, since 1932. He was born in Dawson, Minnesota, received his medical education at the University of Minnesota and served his internship at the University Hospital. He became assistant to the chief of surgery at the University in 1918 and opened his practice in Hallock in 1922. He remained a staff member at the Kittson Memorial Hospital at Hallock and he had just completed a term as chief of surgery at Deaconess Hospital in Grand Forks.

He was a member of the Minnesota State Medical Association until 1932 when he transferred his membership to the Grand Forks District Medical Society and the North Dakota State Medical Association. He was also a member of the North Dakota-Manitoba Urological Society.

Survivors include his wife, the former Irene Dille; four children by an earlier marriage, Dr. John Stratte, Enumclaw, Washington, Dr. Paul Stratte, Redding, California, Mrs. B. M. Griffin, Mauston, Wisconsin, and Mrs. E. L. Billing, San Francisco; a brother, Henry Stratte, Dawson; a sister, Mina Stratte, Glendale, California, and ten grandchildren.

MERRITT WHITACRE WHEELER

Dr. Merritt W. Wheeler, sixty-seven years old, died at his home in Stillwater, June 21. He had practiced medicine in Saint Paul for twenty-eight years until his retirement in 1946.

Dr. Wheeler was born at Albion, Iowa, and received his B.A. and B.S. degrees from Iowa Teachers College and the University of Iowa, respectively. His medical education was obtained at the University of Minnesota. After an internship of three years at St. Joseph's and St. Luke's Hospitals in Saint Paul he took one year's post-graduate training at Tulane University and practiced for six years at Glencoe. Following service as captain in the Army Medical Corps in World War I he moved to Saint Paul and, after a few years, limited his practice to eye, ear, nose and throat work.

Dr. Wheeler was a member of the Ramsey County Medical Society, the Minnesota State Medical Association and the American Medical Association. He was also a member of the American College of Surgeons, the American Legion, Veterans of Foreign Wars, Rotary, Hard-of-Hearing League and Masonic order.

Surviving are his wife, Ruth: a son, Merritt W., Jr., of Lake Elmo; a daughter, Mrs. Henrietta Glenn of Saint Paul, wife of Municipal Judge Andrew Glenn; a sister Mrs. Myrl Gardner of Saint Paul; a brother, Forrest of Minneapolis, and four grandchildren.

Letters to the Editor

Dear Editor:

Because of the great demand on the part of physicians to have a complete set of the Society's monograph series for the practicing physician, National has reprinted copies of all of them.

We are now in a position to fill requests from physicians who may not have received these publications when they were originally distributed.

Is there any possibility of including this announcement in one of the publications of the MINNESOTA MEDICINE?

The complete list of monographs now available is as follows:

1. THE CANCER PROBLEM, by Dr. Shields Warren, 1948.
2. CANCER OF THE HEAD AND NECK, by Dr. Hayes Martin, 1949.
3. THE DIAGNOSIS OF GENITO-URINARY NEOPLASMS, by Dr. Victor F. Marshall, 1949.
4. CANCER OF THE LUNG, by Dr. Richard H. Overholt and Dr. Sherman W. Atwell, 1950.
5. CARCINOMA OF THE BREAST, by Dr. C. D. HAAGENSEN, 1950.
6. CANCER OF THE ESOPHAGUS AND THE STOMACH, by Dr. Owen H. Wangensteen, 1951.
7. VALUE OF EARLY DIAGNOSES OF MALIGNANT LYMPHOMAS AND LEUKEMIAS, by Dr. Lloyd F. Craver, 1952.
8. CANCER OF THE FEMALE GENITAL TRACT, by Dr. Herbert F. Traut and Dr. Ralph C. Benson, 1954.

These monographs are available without charge. Requests can be addressed to: Minnesota Division, American Cancer Society, 295 North Snelling Avenue, Saint Paul 4, Minnesota.

Sincerely yours,
GLORIA FERNANDEZ
Education Director, Minnesota Division
American Cancer Society

May 23, 1954

When I came to consider local government, I began to see how it was in essence the first-line defense thrown up by the community against our common enemies: poverty, sickness, ignorance, isolation, mental derangement and social maladjustment. The battle is not faultlessly conducted, nor are the motives of those who take part all righteous or disinterested, but the war is, I believe, worth fighting, and this corporate action is at least based upon the recognition of one fundamental truth about human nature: we are not only single individuals, face to face with eternity and our separate spirits; we are members one of another.—WINIFRED HOLTBY, "South Riding," quoted in European Conference on Health Education of the Public, London, England, April 10-18, 1953.

◆ Reports and Announcements ◆

MEDICAL MEETINGS

National

American Cancer Society, Board of Directors and special committee meetings, Hotel Radisson, Minneapolis, Minnesota, June 2, 1955.

American Heart Association, Washington, D. C., September 12-17, 1954.

Annual Assembly in Otolaryngology, University of Illinois, Chicago, Illinois, September 6-11, 1954.

Annual Convention of the National Society for Crippled Children and Adults, Hotel Statler, Boston, Massachusetts, November 3-5, 1954.

Industrial Health Conference Seventh Annual, Shamrock Hotel, Houston, Texas, September 23-25, 1954.

International

Conference of International Union Against Tuberculosis, Madrid, Spain, September 26-October 2, 1954.

Congress of International Association for the Prevention of Blindness, New York, New York, September 12-17, 1954.

International Anesthesia Research Society, Los Angeles, California, October 10-14, 1954.

International Congress of Clinical Pathology, Washington, D. C., September 6-10, 1954.

International Congress of Diseases of the Chest, Barcelona, Spain, October 4-8, 1954.

International Poliomyelitis Congress, University of Rome, Orthopedic Clinic, Rome, Italy, September 6-10, 1954.

International Congress of Hematology, Paris, September 6-11, 1954.

International Society of Blood Transfusion, Paris, France, September 12-19, 1954.

International Congress of Industrial Medicine, Naples, Italy, September 13-19, 1954.

International Congress of Internal Medicine, September 15-18, 1954.

International Congress of Ophthalmology, University of Montreal and McGill University, Montreal, Canada, September 9-11, 1954.

International Congress of Orthopedic Surgery and Traumatology, Berne, Switzerland, August 30-September 3, 1954.

International Society of Geographical Pathology, Washington, D. C., September 6-10, 1954.

World Congress of Cardiology, Washington, D. C., September 12-18, 1954.

World Medical Association, Rome, Italy, September 26-October 2, 1954.

NORTHERN MINNESOTA MEDICAL ASSOCIATION

The annual meeting of the Northern Minnesota Medical Association will be held at Willmar, Minnesota, Friday and Saturday, September 10 and 11, 1954. The program will include the following features:

Friday, September 10

Morning Session—10:00 A.M.

"Fractures of the Elbow"—Wesley Burnham, M.D., Minneapolis
Discussion

"Pruritus Ani"—H. M. Frykman, M.D., Minneapolis
Discussion

"Premature Separation of the Placenta"—Bruce Williams, M.D., Duluth
Discussion

Business Meeting—11:50 A.M.

Luncheon—12:30 P.M.

Hosts—Kandiyohi-Swift-Meeker County Medical Society

Afternoon Session—2:00 A.M.

"Rheumatic Fever"—Robert A. Good, M.D., Minneapolis
Discussion

"The Modern Trends in the Management of Hypertension"—Arthur C. Kerkhof, M.D., Minneapolis
Discussion

Panel Discussion:

"The Management of the Bleeding and Recurrent Peptic Ulcers"—Owen H. Wangensteen, M.D., Minneapolis; George A. Hallenbeck, M. D., Rochester; J. J. Coll, M.D., Duluth

Annual Banquet—7:00 P.M.

Address: Paul de Kruif, Holland, Michigan

Saturday, September 11

Morning Session—9:00 A.M.

Clinico-Roentgen-Pathological Conference—Conducted by H. G. Moehring, M.D., Duluth

MINNESOTA ACADEMY OF OCCUPATIONAL MEDICINE AND SURGERY

The Minnesota Academy of Occupational Medicine and Surgery will hold its next meeting at the Kitchi Gammi Club in Duluth, Friday evening, August 27, 1954. Dinner will be at 7:00 p.m. preceded by a social hour.

The scientific session will include a discussion of "Hand Injuries" by Dr. John C. Feuling of Duluth and a description of "The Taconite Research and Development in Minnesota" by Mr. Edward Schmid, Public Relations Chief for the Reserve Mining Company. The participants have been invited by the Reserve Mining Company to tour its plant, particularly the Health Service, on Saturday morning.

SOUTHERN MINNESOTA MEDICAL ASSOCIATION

The annual meeting of the Southern Minnesota Medical Association will be held this year at Winona as an all-day session on Monday, September 13. The tentative program is as follows:

Morning Session—9:00-12:00

"Carcinoma of the Common Duct." Report of two cases. William A. Finkelnburg, M.D., Winona.

"Indications and Contraindications for Tonsillectomy." J. D. Sjoding, M.D., Mankato.

"The Problem of Massive Gastric Hemorrhage with Cause Unknown Pre-operatively." C. E. Rea, M.D., Saint Paul.

"Some Fundamental Considerations in Diagnosis and Treatment of Tumors of the Colon and Rectum." W. A. Fansler, M.D., and William T. Smith, M.D., Minneapolis.

"Psychoses Associated with the Post-Partum Period." E. M. Hammes, Jr., M.D., Saint Paul.

"Current Status of Radio-isotopes in Diagnosis and Treatment of Blood Dyscrasias." C. F. Stroebel, M.D., Rochester.

"Precautions in the Use of Cortisone." Emerson Ward, M.D., Rochester.

"Hearing Loss in Children: Detection, Treatment, and Rehabilitation." K. M. Simonton, M.D., Rochester.

Afternoon Session—2:00-5:00

"Cardiac Surgery."
(To be announced).

"Tumors of the Brain and Spinal Cord as They Concern the General Surgeon." H. J. Svien, M.D., Rochester.

"The Nursing Education Problem in Minnesota." Thelma Dodd, R.N., Saint Paul.

"Teratoma of the Pelvic Colon Presenting in the Lumen."—Case report. S. T. Kucera, M.D., Northfield.

"Pulmonary Hypertension Due to Thrombo-Embolic Pulmonary Arterial Disease Following Pregnancy."—Case report. Ben Sommers, M.D., Saint Paul.

"Meconium Peritonitis."—Case report. G. R. McNear, Jr., M.D., Mankato.

"Case Report: Giant Condyloma (verruca) simulating Carcinoma of the Penis."—Case report. I. Fisher, M.D., Minneapolis.

"Lipoid Pneumonia: The Problems of Differential Diagnosis of Lung Shadows." J. Gordon Beaton, M.D., Northfield.

AMERICAN BOARD OF OBSTETRICS AND GYNECOLOGY

Applications for certification (American Board of Obstetrics and Gynecology) for the 1955 Part I Examinations are now being accepted. Candidates are urged to make such application sometime in July or August.

All candidates for admission to the Examinations are required to submit with their application, a plain typewritten list of all patients admitted to the hospitals where they practice, for the year preceding their application or the year prior to their request for reopening of their application, with the diagnosis, pathological diagnosis, nature of treatment, and end result.

Under a change of requirements for the Part I Examination, candidates must submit twenty case abstracts rather than twenty-five as formerly. Five of these may be from one's residency service.

Application for examination or re-examination, as well as requests for resubmission of case abstracts, must be made to the Secretary prior to October 1, 1954, and should be addressed as follows: Robert L. Faulkner, M.D., Secretary, 2105 Adelbert Road, Cleveland 6, Ohio.

PEDIATRIC REUNION TO HONOR DOCTOR McQUARRIE

In honor of twenty-five years of distinguished service to the University of Minnesota, Dr. Irvine McQuarrie, head of the department of pediatrics, will be honored at a pediatrics grand reunion September 23-25 at the University.

Colleagues and students of Dr. McQuarrie have planned the reunion in conjunction with the annual Northwestern Pediatric Society meeting.

Lectures will be delivered at the meeting by twenty-two academic leaders in the field of pediatrics. Most of them received their pediatric training under Dr. McQuarrie.

As an additional tribute to the University pediatric chief, a McQuarrie Pediarics Fund has been established with a goal of $50,000. An annual source of income from it will be earmarked: to provide a lectureship in pediatrics, as a scientific travel fund for pediatrics staff members to attend special conferences, to provide fellowships in unusual circumstances, to provide necessary equipment that may be needed from time to time by the department and for any other purpose within the department as approved by the committee.

Dr. McQuarrie, a graduate of the University of Utah who received his doctor of philosophy degree from the University of California and his doctor of medicine degree from Johns Hopkins university, joined the University of Minnesota staff in 1929.

Before coming to Minnesota, he had served on staffs of the University of California, Henry Ford Hospital, Yale university and the University of Rochester. And in addition to his duties at the University during the past twenty-five years he was visiting professor of pediatrics at Peking Union Medical college in China and served on the China Medical Board of Rockefeller Foundation.

Lecturing assignments on pediatrics have taken him to the far corners of the world, including Japan, Brazil, Uruguay, Argentina, Chile, Peru, Canada, Mexico, Italy, Cuba and China.

Dr. McQuarrie is editor-in-chief of Brennemann-

McQuarrie System Practice in Pediatrics and is an associate editor of ten medical publications.

In addition to his personal contributions to the field of pediatrics, men trained by him are now spreading his educational theories on pediatrics in such places as the Universities of Texas, Louisiana, California, Iowa, Utah, Colorado and Oregon as well as Tulane University, Meharry College, Stanford University and Peking Union Medical College.

CONTINUATION COURSES

The University of Minnesota will join the Minnesota Tuberculosis and Health Association in sponsoring a continuation course in *Tuberculosis Control for Lay Persons* October 11 to 13, 1954, at the Center for Continuation Study on the University Campus. Public health aspects of tuberculosis and other chest diseases will be stressed.

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A continuation course in *Dermatology for Specialists* will be held at the Center for Continuation Study October 28 to 30, 1954. The program will deal with many topics of current interest to practicing dermatologists; sarcoidosis will receive special emphasis. The program will be presented under the direction of Dr. Henry E. Michelson, Professor, Department of Medicine, and Director, Division of Dermatology, University of Minnesota.

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A continuation course in *Clinical Microscopy and Parasitology for Medical Technologists* will be held at the Center for Continuation Study November 15 to 17, 1954. Half of the three-day program will be devoted to the examination of various body fluids such as urine, spinal fluid, and gastric secretion; the remainder to parasitology. Guest faculty will include Dr. Donald B. McMullen, Chief, Department of Medical Zoology, Army Medical Service Graduate School, Washington, D.C.

UROLOGY AWARD

The American Urological Association offers an annual award of $1000 (first prize of $500, second prize $300 and third prize $200) for essays on the result of some clinical or laboratory research in urology. Competition shall be limited to urologists who have been graduated not more than ten years, and to men in training to become urologists.

The first prize essay will appear on the program of the forthcoming meeting of the American Urological Association, to be held at the Biltmore Hotel, Los Angeles, California, May 16-19, 1955.

For full particulars, write the Executive Secretary, William P. Didusch, 1120 North Charles Street, Baltimore, Maryland. Essays must be in his hands before January 1, 1955.

Woman's Auxiliary

DATE CHOSEN FOR AUXILIARY PROGRAM

October 1 is the date set for the annual School of Instruction of the Woman's Auxiliary of the Minnesota State Medical Association, according to Mrs. Peter S. Rudie of Duluth, president of the organization.

The day's events will begin with registration and a Board meeting at 9 a.m., to be followed by a series of talks and panel discussions plus a luncheon and a special luncheon program. The place is the Hotel Radisson in Minneapolis, and Auxiliary members from all over the state will be invited to attend.

Several nationally known speakers are expected to take part in the program, including the new president of the Woman's Auxiliary to the American Medical Association, Mrs. George Turner of El Paso, Texas.

RAMSEY COUNTY REPORTS ACTIVITIES

Current activities of the Woman's Auxiliary to the Ramsey County Medical Society are chiefly directed toward raising funds for local and national projects of interest to doctors' wives, according to Mrs. L. T. Simons, chairman for MINNESOTA MEDICINE.

Neighborhood House and the Hallie Q. Brown Neighborhood House benefited chiefly by 1953-54 fund-raising activities with Mrs. Philip K. Arzt, president of the Auxiliary, in charge. The purpose was to help send children to summer camps. A gift was also sent during this period for use of the University of Minnesota to the American Medical Association Research and Scholarship Fund.

Mrs. W. P. Gardner, who was elected to succeed Mrs. Arzt for 1955, called a meeting of the Auxiliary Board at her home, June 21, to plan other fund-raising activities to benefit the American Medical Education Fund. A "Kick-Off Fall Fashion Show and Luncheon" is the first event planned for this purpose. It will be held September 23 at the Saint Paul Hotel with Mrs. M. Dudley Hilker as general chairman and Mrs. W. A. Carley as co-chairman. Other committee chairmen appointed at the meeting are: Mrs. Rodney F. Sturley and Mrs. C. T. Eginton, tickets; Mrs. Duane R. Ausman, hostesses; Mrs. Douglas R. Kusske, decorations and Mrs. G. E. Knutson, door prizes.

An exhibit showing work of the Auxiliary and presented in connection with the Middle States Public Health meeting, June 4, at the Saint Paul Hotel, was also arranged and manned by the Ramsey county group with Mrs. R. K. Grau in charge.

◆ Of General Interest ◆

Dr. John J. Hochfilzer, Saint Paul, will deliver a lecture on "Endotrachael Anesthesia in Children" before the Austrian Otolaryngological Society in Bregenz, Austria, September 11, 1954. Dr. and Mrs. Hochfilzer let for Austria by plane on August 10.

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Dr. Henry W. Goehrs, Saint Cloud, was elected President of the Great Northern Railway Surgeons' Association at its annual meeting in Great Falls, Montana, June 29, 1954. Dr. Goehrs presented a paper on "The Silicosis Problem" with a number of films taken over a period of several years.

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Dr. William C. Bernstein, Saint Paul, has been promoted to Clinical Associate Professor of Surgery and Proctology, according to an announcement by the Board of Regents of the University of Minnesota following their June meeting. Dr. Bernstein has been active in the teaching programs at the University Hospitals, U. S. Veterans Hospital and Ancker Hospital.

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Dr. Gordon R. Kamman, Saint Paul, was Moderator at a round-table discussion at the Center for Continuation Study, University of Minnesota, on the subject "Continuation Course in New Developments in Nursing Home Care." On the panel were Dr. Jeanette Baker, Mr. Stuart Atkinson and Mrs. Mary Swanson, all of Fergus Falls. Dr. Baker is the Medical Director of the Geriatric Program at the Fergus Falls State Hospital; Mr. Atkinson is the Clinical Psychologist in charge of this project and Mrs. Swanson is the Chief Social Service Worker at Fergus Falls. More than 150 people attended the meeting.

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Dr. Edward C. Maeder, Minneapolis, announces the association of Dr. William E. Taylor of Saint Paul in the practice of obstetrics and gynecology at 1051 Medical Arts Building, Minneapolis. Dr. Taylor has just completed a two-year residency in obstetrics and gynecology at St. Mary's Hospital, Minneapolis.

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Dr. David D. Norman, who has just completed his postgraduate Fellowship in Neurology and Psychiatry at the Medical School of the University of Minnesota and the Veterans Hospital, Minneapolis, has become associated in the practice of neurology and psychiatry with Doctors Hammes and Hammes at 1124 Lowry Medical Arts Building, Saint Paul.

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Dr. Harold Basinger, after a year of study under a fellowship at Williamsport Hospital, Williamsport, Pennsylvania, has returned to Windom to be associated in practice with his father, Dr. Homer Basinger, and with Dr. Harvey Basinger and Dr. Elmer Schutz of Mountain Lake. Dr. Basinger holds the rank of Lieutenant (jg) in the Navy.

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Dr. F. W. Behmler, Morris, has filed as a candidate for the State Senate from the 48th District, which comprises the counties of Stevens, Traverse, Big Stone and Grant. Dr. Behmler will seek the office now held by Senator A. R. Johanson of Wheaton, who has announced that he will not be a candidate for re-election this year. Dr. Behmler, who is first vice president of the Minnesota State Medical Association and is serving his second year as president of the Minnesota State Board of Health, was among the first to be mentioned as a possible candidate from his district, after Senator Johanson's announcement. He has practiced medicine in Morris for the past eighteen years.

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Sympathy is extended to **Dr. and Mrs. Lawrence R. Boies,** of Hopkins, in the loss of their son, William M. Boies, a student at the University of Wisconsin, who was accidentally drowned while picnicking with a group on the Baraboo Bluffs near Sauk City, Sunday, July 11, 1954.

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Dr. and Mrs. N. W. Barker, of Rochester, have returned from a trip to Europe, where Dr. Barker attended the International Congress on Thrombosis and Embolism in Basle, Switzerland. Dr. and Mrs. Barker enjoyed a six weeks' tour including Paris, Geneva, Lucerne, Basle, Innsbruck, Salzburg, Heidelberg, Cologne, Amsterdam, London and rural southern England. Included was a trip down the Rhine River by boat.

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Dr. J. S. Blumenthal, of Minneapolis, has been promoted to Associate Professor of Internal Medicine and Allergy at the University of Minnesota and was made chief of the Allergy Clinic.

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Dr. J. A. Cosgriff, Sr., of Olivia, was elected first vice president of the Minnesota Heart Association at the annual meeting held in July. Dr. Cosgriff served as a member of the Board of Directors the past year.

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Dr. James S. Cole, who recently completed a residency at Ancker Hospital, has become associated with the Oxboro Clinic, Minneapolis, and will confine his practice to internal medicine. Dr. Cole is a graduate of Indiana University School of Medicine, 1947.

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Dr. Eleanor Colle, of Minneapolis, and **Dr. Lloyd D. MacLean,** of Calgary, Alberta, Canada, were united in marriage at St. John's Episcopal Church, Linden Hills, June 30, 1954. The bride is a staff member of the pediatrics department of the University Hospitals; the groom is serving his residency in surgery at University Hospitals, having received his M. D. degree from the University of Alberta. Dr. and Mrs. MacLean are now at home at 132 Orlin Avenue S.E., Minneapolis.

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Dr. Lorin D. Dawson, who recently completed a surgery residency at Veterans Hospital, Minneapolis, has been appointed staff surgeon of the Worthington Clinic, Worthington. Dr. Dawson is a graduate of the University of Wisconsin Medical School and interned at the University of Pennsylvania before. He practiced for a time in Milbank, South Dakota, before coming to the Twin Cities for special training in surgery.

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Dr. and Mrs. E. C. Elkins, of Rochester, have returned from a two months' sojourn in Europe, where Dr. Elkins attended the meeting of the International Federation of Physical Medicine in Ostend, Belgium. While traveling in Europe, Dr. Elkins visited medical centers in Rotterdam, London, Stockholm, Copenhagen and Zurich. He and Mrs. Elkins toured England, Scotland, The Netherlands, Italy, France and Belgium.

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Dr. George B. Eusterman, of Rochester, has returned to his home there after spending eighteen months in Detroit, Michigan, helping to organize the medical department of the new Sinai Hospital there. He served as chief of the medical staff and director of Medical Education and was instrumental in having the hospital accrediting for intern and resident training. At present, Dr. Eusterman is completing a history of the department of medicine of the Mayo Clinic for the Clinic archives.

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Dr. and Mrs. T. R. Fritsche, of New Ulm, have returned from Ulm, Germany, where Dr. Fritsche, as Mayor of New Ulm, acted as official delegate from his home town at the 1100th birthday celebration of the ancient city for which New Ulm was named. During Dr. Fritsche's absence, Dr. R. O. Leavenworth, Jr., of Saint Paul, took care of his practice which is devoted to diseases of the eye, ear, nose and throat.

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Dr. Darwin K. Holian, who has been a fellow at Minneapolis General Hospital since 1951, has been named to fill a newly created post with the Medical and Surgical Center in Albert Lea. He will limit his practice to general surgery and orthopedics. Dr. Holian, a native of Maynard, Minnesota, is a graduate of the University of Minnesota School of Medicine and served in the U. S. Army Medical Corps for two years.

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Dr. Stuart W. Harrington, head of the section of surgery in the Mayo Clinic since 1920 and professor of surgery in the Mayo Foundation since 1936, retired from Clinic practice in July. Dr. Harrington was honored by his assistants who came from all parts of the country to participate in a dinner party at which they presented the honor guest with a silver tray engraved with their names.

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Dr. William H. Henney, of McIntosh, was guest of honor on "Dr. Henney Appreciation Day," June 19, 1954, when his townsmen gathered to pay their respects to the physician who has served them as physician and mentor for the past forty-six years.

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Dr. Arthur J. Myers, Minneapolis, received the honorary degree of Doctor of Laws at Ohio University on June 13, 1954.

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Dr. E. J. Engberg, Faribault, was among those certified as a competent mental hospital administrator by the Committee on Certification of Mental Hospital Administrators of the American Psychiatric Association at the meeting held in St. Louis in May.

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Dr. J. V. Wallinga, who has been practicing child psychiatry in Minneapolis and serving as psychiatric consultant to the Child Services Division, State Department of Publc Welfare, has been recalled into the Navy. He will report to Oakland Naval Hospital, Oakland, California. Dr. Wallinga received certification by the American Board of Psychiatry and Neurology in May, 1954.

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Dr. Paul Hedenstrom, son of the late Dr. L. H. Hedenstrom, has joined the staff of the Cambridge Clinic. Dr. Hedenstrom received his medical degree from the University of Minnesota and interned at a Duluth hospital.

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Dr. E. H. Haberman, Osakis, was the recipient of an ovation in June when members of the Osakis community gathered to pay tribute to his long years of service to the Osakis residents and to commemorate the golden anniversary of his graduation from medical school. Dr. Haberman was presented with a gold medal in honor of the event.

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Dr. and Mrs. Edward N. Peterson, Virginia, attended the University of Minnesota commencement exercises June 12, when two of their sons received degrees from the University of Minnesota School of Medicine. Edward, who received the degree of doctor of medicine, is now in Buffalo, New York, where he will intern at the Edward J. Meyer Memorial Hospital. Charles, who received the bachelor of arts degree, cum laude, will be a sophomore in medical school next year.

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Dr. S. A. Slater, Worthington, recently attended the meeting of the National Tuberculosis Association at Atlantic City, New Jersey, where he was re-elected to the Board of Directors. He has served on the board for twenty-four years, longer than any other member. He also attended the forty-fifth reunion of his medical school class at the Medical College of Virginia.

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Dr. F. W. Behmler, Morris, attended a meeting of the State Board of Health in Minneapolis on June 1. He also attended the Middle States Public Health conference in Saint Paul, June 2, and presented the welcoming address to the visiting public health officials from a number of states.

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Dr. and Mrs. D. R. Navratil, Montgomery, are now in Baltimore, Maryland, where the doctor is taking a three-year postgraduate course in surgery at Johns Hopkins University and the University of Maryland Medical School.

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Dr. Karl R. Lundberg, former Army specialist in preventive medicine, took over his duties as Minneapolis commissioner of health on June 15.

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Dr. Louis J. Wilson, who has just completed a tour of military service in the Navy Medical Corps, has returned to Winona and is resuming practice with his father, **Dr. R. H. Wilson.**

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United States Public Health Service fellowships have been awarded to two residents in surgery at Ancker Hospital, Saint Paul, **Dr. Joseph L. Sprafka, Jr.,** was named a "national heart trainee" and **Dr. Truman J. Newberry,** "a national cancer trainee."

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Dr. Howard K. Gray, Mayo Clinic surgeon, was awarded the honorary degree of Doctor of Science by the University of Nebraska College of Medicine in Omaha, at the June graduation exercises

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Dr. T. C. Chisholm, Minneapolis, spoke to the Range Medical Association on June 22 at the Itasca Memorial Hospital. His topic was "Latest Developments in the Field of Children's Surgery."

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Dr. and Mrs. H. W. Meyerding, Rochester, recently returned from a two-month vacation in South America. While in Sao Paulo, Dr. Meyerding spoke before the International Surgical Society.

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Dr. Gilman Goehrs, St. Cloud, was recently re-elected state surgeon of the Veterans of Foreign Wars at the encampment of the service organization at Hibbing.

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Dr. Donald W. Mulder has returned to the Mayo Clinic after fourteen months of active duty in the Navy in the Pacific theater. Dr. Mulder, who is a consultant in the section of neurology and psychiatry, held the rank of lieutenant commander. He was recalled into service in March, 1953, to carry out a study of head injuries incident to the Korean war. After the cease fire, he took part in a study of amytrophic lateral sclerosis, an assignment that took him to Guam and other South Pacific islands.

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Dr. Viktor O. Wilson, Rochester, has been re-named city-county health officer of Rochester and Olmsted County.

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A new children's clinic has opened in Wayzata. Three pediatricians, **Dr. E. J. Huennekens, Dr. R. D. Semsch** and **Dr. D. H. Petersen** make up the staff of the clinic.

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Dr. John Carroll, Winsted's new physician, has joined the staff of the Glencoe Hospital.

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Twenty-one Minnesota doctors celebrating their fifty years in the practice of medicine were honored by the State Medical association at its annual banquet in Duluth, June 8. Those so honored were: **Drs. Albert E. Ahrens,** Saint Paul; **Jacob Biedermann,** Thief River Falls; **William A. Brand,** Redwood Falls; **Hermanus DeBoer,** Edgerton; **Herbert Z. Giffin,** Rochester; **Emil Haberman,** Osakis; **John G. W. Havens,** Austin; **Andrew D. Hoidale,** Tracy; **LeRoy J. Holmberg,** Canby; **Alva E. Holmes,** Rush City; **John E. Hymes,** Minneapolis; **Claude B. Lewis,** St. Cloud; **George R. Melzer,** Lyle; **R. P. Pearsall,** Virginia; **Joseph W. Preisinger,** Dallas, Texas; **Albert G. Schulze,** Saint Paul; **Ivar Sivertsen,** Minneapolis; **George E. Thomas,** Minneapolis; **Leon A. Williams,** Minneapolis; **Rasmus V. Williams,** Rushford; **Andrew Gullixson,** Longmont, Colorado.

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Dr. Frederick A. Figi, head of the section of oral and plastic surgery, Mayo Clinic, received an honorary Doctor of Science degree from the University of Nebraska, at the 83rd commencement exercises in June. He was also one of the speakers at the American Medical association meeting in San Francisco, June 21-25.

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The Damon Runyon Memorial fund has awarded $15,000 to the University of Minnesota to permit **Dr. Owen H. Wangensteen,** chief of the university's surgery department, to continue with his "second look" project for patients with gastric, colic or rectal cancer.

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Dr. M. W. Comfort, Mayo Clinic staff member and professor of medicine in the Mayo foundation graduate school since 1946, was awarded the honorary degree of Doctor of Laws by Austin College, Sherman, Texas, at the commencement exercises in June.

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Dr. Howard O. Mortenson of Menahga and Miss Carol Jean Jarvinen, also of Menahga, were united in marriage June 21, 1954. Dr. and Mrs. Mortenson are now at home in Menahga following a trip to Banff and Lake Louise, Canada.

Dr. Charles P. Robbins of Winona this year completed fifty-nine years of practice in Winona, where he came a year following his graduation from Jefferson Medical College in 1894. Although Dr. Robbins discontinued office practice about three years ago, owing to a back injury, he still maintains an office at his residence.

NEW LOCATIONS

Dr. Kristofer Hagen, formerly of Cokato, is now located in Richfield.

Dr. Henry B. Blumberg, Fairmont physician, has accepted a position as chief resident of the San Joaquin County Hospital in Stockton, California, where he will head a staff of twelve doctors. He intends to obtain further specialized training in internal medicine for one year and then return to Fairmont.

Dr. E. Duane Sommerness is the new clinical director at the Fergus Falls State Hospital. Dr. Sommerness formerly was clinical director at the Willmar Hospital.

Dr. Leighton W. Larson, formerly associated with Dr. J. W. Bretholdt at Watertown, is now engaged in practice at Waconia.

Dr. Joseph W. Burnett, formerly of Fergus Falls, is the new consultant at Union Hospital and Loretta Hospital in New Ulm, and at the Sleepy Eye Hospital.

Dr. Philip J. Parker, who has been associated with the Bratrud Clinic in Thief River Falls for the past three years, has opened an office in Hallock.

Dr. Ernest Ager is now located in Le Center. He formerly practiced in Minneapolis.

Dr. Carl O. Bretzke has moved from Greenbush to Hutchinson, where he has opened an office for the practice of medicine.

Dr. John Bouma, who has been associated in practice with Dr. Gerrit Beckering at Edgerton, has been awarded a three-year fellowship in psychiatry at the Menninger Foundation in Topeka, Kansas. Dr. Bouma started his work under the fellowship July 1.

Dr. William Alden, at present a lieutenant colonel in the Air Force, plans to open a medical practice in Kimball, upon his release from the Air Force.

Drs. M. J. Bernier and **R. W. Reif** of Saint Paul are now located in their new offices at 721 Margaret Street.

Dr. Calvin Elrod, who has been associated in practice with Dr. William I. Davis at Mound the past two years, has moved with his wife and two children to Long Lake, where he started his practice in July.

Dr. Bradley W. Kusske, who was associated with Dr. D. Howard Vogel in the Vogel Clinic at New Ulm for three years, is now practicing medicine in Saint Paul with Dr. A. W. Hilger, Dr. Jerome A. Hilger, and his brother, Dr. Douglas R. Kusske.

Dr. E. R. Crow has left Walker, where he maintained an office with Dr. O. F. Ringle, to take a position with the Healthwin Hospital for tuberculosis patients at South Bend, Indiana.

MINNESOTA BLUE SHIELD-BLUE CROSS

During the recent annual meeting of the Minnesota State Medical Association in Duluth, many doctors and their office personnel visited the Blue Cross and Blue Shield booth. Compared with previous years, the number of visitors at the booth and the interest in Blue Shield matters made it a banner year. Not only were practicing physicians and their office nurses, receptionists and secretaries among the callers, but also doctors in residencies, internships and advanced study visited the booth.

In order to provide any requested information to members of the medical profession and other interested visitors, representatives of the Blue Cross-Blue Shield staff were constantly on hand. Interest of the physicians embraced a wide range of subjects such as enrollment, the different services covered by the contract, comparison of Plan A and Plan B contracts and fees, and identification of Plan A and Plan B subscribers. Discussion with the members of the doctors' offices primarily involved matters relating to the filing and payment of claims.

The fact that a representative of Blue Shield was in attendance at the booth furnished an excellent opportunity for the exchange of suggestions and information between the Blue Shield representatives and the visiting doctors. Many of the questions disclosed a need for a more intensive professional relations program directed toward keeping the profession informed about Blue Shield matters and assisting the personnel of doctors' offices in promptly and properly filing claims. Many recent graduates, interns and resident physicians were most interested in how to become a participating Blue Shield physician.

Another feature attracting attention was the Blue Shield plaque for display in the doctor's office. This plaque consists of a Blue Shield with a central gray caduceus placed upon a gray background. On the face are printed the words, "Participating Doctor of Medicine, Minnesota Medical Service, Inc." These plaques are useful in encouraging the patients to show their Blue Shield identification cards so that claims may be filed early and payment made promptly. Any participating physician who wishes to have a Blue Shield plaque may obtain one by writing Minnesota Medical Service, Inc., 2610 University Avenue, Saint Paul 14, Minnesota.

During the month of May, 4,624 contracts representing 10,649 participant subscribers were enrolled in Blue Cross, bringing the net enrollment as of May 31, 1954 to a total of 378,019 Blue Cross contracts covering 1,003,140 participant subscribers.

Enrollment in Blue Shield during the month of May, 1954, was increased by 1,423 contracts representing 9,029 participant subscribers bringing the net enrollment as of May 31, 1954, to 241,798 Blue Shield contracts covering 635,130 participant subscribers. As of May 31, 1954, there were 240,762 Plan A contracts in effect covering 633,424 participant subscribers and 1,036 Plan B contracts covering 1,706 participant subscribers.

BOOK REVIEWS

Books listed here become the property of the Ramsey, Hennepin and St. Louis County Medical Libraries when reviewed. Members, however, are urged to write reviews of any or every recent book which may be of interest to physicians.

BOOKS RECEIVED FOR REVIEW

ILLUSTRATED REVIEW OF FRACTURE TREATMENT. Frederick Lee Liebolt, A.B., M.D., Sc.D., LL.D. Attending Surgeon in Charge of Orthopedics, New York Hospital; Attending Orthopedic Surgeon, Hospital for Special Surgery; Associate Professor of Clinical Surgery (Orthopedics), Cornell University Medical College, 1954.

PRIMER OF ALLERGY. Warren T. Vaughan, M.S., M.D., Richmond, Virginia. Fourth Edition, Revised by J. Harvey Black, M.D., Dallas, Texas. 191 pages. Illus. Price $4.25, cloth. St. Louis: C. V. Mosby Co., 1954.

THE PHYSICIAN AND HIS PRACTICE. By Eighteen Authorities. Edited by Joseph Garland, M.D., editor of The New England Journal of Medicine. 270 pages. Price $5.00, cloth. Boston: Little, Brown & Co., 1954.

ANNUAL REPORT ON THE RESULTS OF TREATMENT IN CARCINOMA OF THE UTERUS. J. Hayman, M.D., Editor, and Committee. 256 pages. Illus. Paper cover. Stockholm, Sweden: Radiumhemmet, 1954.

BCG AND VOLE VACCINATION. A practical handbook. By K. Neville Irvine, MA, DM, BCh, MRCS, LRCP, Adviser in BCG Vaccination to Oxford Regional Hospital Board. 96 pages. Illus. 10 colored plates. Price 12s. 6d. London: The National Association for the Prevention of Tuberculosis, 1954.

This pocket-size book contains a comprehensive index, a number of black and white illustrations and ten colored plates. It gives a concise summary of what is known about the BCG and vole vaccines, and outlines the theory of vaccination. Practical instructions for tuberculin testing by a variety of methods are followed by instructions on the technique of intracutaneous vaccination, the multiple-puncture method, scarification and oral vaccination. Further chapters deal with the complications of vaccination and the organization of vaccination procedure.

The four pages of colored plates illustrate clearly the results obtained by a positive tuberculin test, using four different methods, and show the results of intracutaneous BCG vaccination (in four progressive stages) and of multiple-puncture vaccination.

In a Foreword, Frederick Heaf, MA, MD, FRCP, David Davies Professor of Tuberculosis, The Welsh National School of Medicine, says, "There can be no doubt that this small book on a complicated and intricate subject will, by its clarity and lucidity, help many who are already using the vaccines. It will help many more who are as yet only vaguely acquainted with the subject to understand and take a greater interest in this work of national importance."

ACUTE PERFORATED PEPTIC ULCER

(Continued from Page 611)

3. Inability to completely empty the stomach for any reason should be considered a contraindication to the non-operative regime. We have not considered a recent meal as an absolute contraindication to this method of treatment, although the initial emptying of the stomach is more difficult and time-consuming.

4. Theoretically, a huge perforation, such as those occasionally seen from a large carcinomatous ulcer on the anterior aspect of the stomach, might not respond to non-operative treatment. However, benign peptic ulcer perforations of apparently quite large size were treated non-operatively by us with no unusual difficulties.

Conclusions

We feel that the non-operative management of acute perforated peptic ulcer, if properly conducted, is a safe and effectual means of handling this acute abdominal catastrophe. The non-operative regime has many advantages over the operative treatment in that the complications of the operation itself, few as these may be, are avoided, and the recovery period is easier and shorter. There are definite contraindications to the use of this method of treatment. There are also very definite hazards which can be avoided only by strict adherence to rigid rules of management.

* * *

Dr. Ray Hedin, of Red Wing, Minnesota, then read his inaugural thesis on "Parotid Tumers." Lantern slides were shown. (Published in June, 1954, issue.)

The meeting was adjourned.

Robert E. Priest, M.D., *Secretary*

TRUDEAU SANATORIUM PAMPHLET AVAILABLE

A new twenty-page illustrated folder on Trudeau Sanatorium has just been issued by that Institution.

Printed in two colors, the pamphlet describes the facilities of Trudeau and is designed primarily to give prospective patients general information about the Sanatorium. Copies were mailed in mid-May to 14,500 physicians east of the Mississippi.

Additional copies are available from Dr. Roger S. Mitchell, Clinical Director, Trudeau Sanatorium, Trudeau, New York.

NEW EXHIBIT ON SINUS TROUBLE

Persons suffering from clogged heads or draining noses will be especially interested in the new exhibit—"Sinus Trouble"—which the AMA Bureau of Exhibits now is offering to state and county medical societies for local showings at fairs and similar public gatherings. Depicting the location of the sinuses, diagnostic procedures and latest treatments now followed by physicians, this exhibit is available for immediate bookings through the Bureau.

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PROBLEMS ARISING IN THE TREATMENT OF ADRENAL INSUFFICIENCY WITH CORTISONE AND HYDROCORTISONE

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ALTHOUGH there are some problems in the use of cortisone and hydrocortisone in the treatment of adrenal insufficiency, the benefits from use of these compounds far outweigh any problems. Since the benefits are really very great, I shall spend a few minutes describing them and outlining a satisfactory regime for treatment of Addison's disease.

Adrenal insufficiency causes a loss of sodium and chloride in the urine decrease in renal excretion of urea and of potassium, faulty handling of water with tendency to water intoxication, very poor tolerance for stress of any kind, decreased gluconeogenesis with resulting spontaneous hypoglycemia on fasting more than twelve to sixteen hours, and marked decrease in muscle strength. These phenomena make untreated adrenal insufficiency a very serious situation. The use of cortisone and hydrocortisone have greatly improved the well-being of patients with Addison's disease and has practically eliminated the occurrence of crises. Treatment with desoxycorticosterone and adrenal cortical extract prolonged the lives of many patients and permitted many to be fairly active. Cortisone treatment has improved vigor and work tolerance to or toward normal, improved muscle strength, corrected abnormal water metabolism, promoted gluconeogenesis, corrected electroencephalographic abnormalities, and prevented development of crises during infections and other stress-producing situations.

One of the best indices of the efficacy of present treatment is the decrease in the need for hospitalization. The number of admissions for treatment of Addison's disease was obtained by analyzing the records of thirteen addisonian patients who were treated chiefly with desoxycorticosterone before 1950 or 1951 and who are being treated with cortisone with or without other supplements to the present time. There were forty-eight admissions to the hospital (often prolonged and complicated illnesses) in 43.2 patient years. Since cortisone treatment was instituted, there have been only four admissions in 35 patient years in this same group of patients. If the length of hospital stay under these circumstances were also taken into consideration, the results would be even more striking. Reciting the case histories of most of these patients would reveal a most remarkable improvement—often from a life of chronic invalidism to a really full normal life. Sorkin and Soffer[5] do not agree that cortisone is as beneficial as indicated above. On the other hand, Thorn and his collaborators[6,7] have had a large experience with the use of these compounds and report very favorable results.

Analysis of the records of twenty patients has revealed the following facts about treatment which is effective in controlling symptoms very satisfactorily:

1. Average daily dose of cortisone (oral)	*25 mg.*	*18.5 mg.*	*12.5 mg.*
Number of patients	6	2	12
2. Requirement for Desoxycorticosterone	Oral	Parenteral	None
Number of patients	5	5	10

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Presented at the annual meeting of the Minnesota State Medical Association, Duluth, June 8, 1954.

The dose of cortisone taken orally should be divided into at least two doses (and three doses are often desirable). It has been found to be practical and desirable to give doses as follows: ¼ tablet on arising and another ¼ tablet at 1:00 to 2:00 P.M. If a dose of 25 mg. is needed, 10 to 12.5 mg. can be given in the morning, 5 to 6.25 mg. at noon, and 5 to 6.25 mg. in the afternoon. It has become apparent that it is usually unnecessary and even unwise to give an evening dose. Patients complain of insomnia if the dose is split into two equal parts and given at twelve-hour intervals. By giving the doses as described above, one simulates what actually happens in health. The adrenal cortex in normal healthy individuals is most active in early morning hours, and conversely secretes very little steroid material during the evening and night hours.

Whenever a patient has a respiratory infection or when he is undergoing any unusual stress or is injured, the dose of cortisone should be doubled or tripled. If the patient vomits, it is necessary to administer the cortisone intramuscularly and a dose of 50 to 100 mg. is indicated. It should be remembered that absorption of cortisone from a muscle depot is much slower than from an oral dose. This has been known from clinical evidence for some time, and it has been conclusively proven by measuring the plasma 17-hydroxycorticosteroid content after oral and intramuscular routes of administration.[2] After a dose of 100 mg. orally, there is a very high peak in two hours but with no 17-hydroxycorticosteroid demonstrable at twenty-four hours. Conversely, after only 50 mg. given intramuscularly an appreciable concentration of these compounds is measurable at twenty-four hours.

As has been indicated above, at least half the patients will need to receive some form of desoxycorticosterone acetate. Oral linguets (2.0 mg.) once or twice a day usually suffice. Daily intramuscular injections (0.5-2.0 mg/d.) afford exact and yet flexible therapy but are inconvenient. Intramuscular injection of a new preparation, a micro-crystalline suspension of the trimethyl-acetate ester of desoxycorticosterone, lasts thirty to forty days so the dose should be from 30 mg. to 60 mg. or even more once every four to six weeks. Pellet implantation also is a good method but is unnecessary under most circumstances now. Unless there is specific contra-indication to DCA, it is probably better to administer some form of it to all patients with Addison's disease.

Cortisone or hydrocortisone is also very useful in the treatment of the adrenal cortical insufficiency secondary to hypopituitarism. The results in the latter condition are often as striking as in primary adrenal insufficiency; but because there are multiple glandular deficiencies, replacement therapy often is not successful in completely correcting the symptoms.

The treatment of addisonian crisis will be discussed in general terms to indicate the order of magnitude of therapy. Cortisone or hydrocortisone should be administered orally if the patient is not vomiting. An initial dose should be 50 to 100 mg. and a total dose for the first day should be from 100 to 300 mg. When vomiting occurs or the patient is unconscious, the intramuscular dose should be from 100 to 200 mg. initially with a total dose of 150 to 300 mg. on the first day. Usually, the condition is so much better the second day that the total dose by either route can be reduced to 100 mg. If hydrocortisone is available for intravenous use, an initial dose of 50 mg. followed by an infusion of 5 to 10 mg. per hour for the first twelve to twenty-four hours is the most effective therapy thus far available.

In the treatment of patients with severe collapse where the hydrocortisone preparation for intravenous use is not available, adrenal cortical extract is indicated. An initial dose of 50 cc. of aqueous adrenal cortical extract intravenously immediately and 50 cc. in the first infusion flask will be effective until enough cortisone is absorbed from deposits in the muscle to be effective.

Desoxycorticosterone acetate (DCA) in oil (10 mg.) should be given intramuscularly unless there is evidence of recent toxicity from this drug. On subsequent days, the dose can be quickly lowered to maintenance amounts (v.s.).

The need for salt and fluids is variable. If the patient has not been treated previously, there is need for 2 to 3 liters of saline with glucose. Usually, in the previously treated patient, who has developed a crisis because of infection or trauma, the amount of sodium chloride should be restricted to 9.0 Gm. (or 1,000 cc. of physiological saline) and a total amount of water to 2,000 to 3,000 cc. a day.

After devoting a major portion of the time to discussing advantages and doses of cortisone, I shall devote a short time to some problems. Some patients are quite sensitive to cortisone or hydrocortisone on initial administration. The patients may become mentally stimulated during the initial period of treatment. If this occurs, it is imperative that the dose be reduced and spread out over the whole day and then gradually increased to a maintenance dose of 12.5 mg. or more per day. When a patient exhibits such sensitivity, it is very important to give the drug in 3 or more divided doses, since there is a high peak in the plasma and then a rapid decline from any single oral dose.

There is no evidence that the use of moderate doses (12.5 to 25 mg.) has interfered with the healing of tuberculosis—in fact, addisonian patients with tuberculosis get along better with than without cortisone.[1]

The maintenance dose of cortisone is not large enough to cause difficulty with sodium retention or potassium depletion. In fact, it is usually necessary to administer extra salt, and in about half the patients desoxycorticosterone is also needed.

Knowlton[3] has described patients who developed hypertension for the first time when given cortisone in relatively small doses. This occurred in one patient who had been maintained on desoxycorticosterone without hypertension for seven years. I have not made any similar observations.

One patient was admitted to the University Hospital and shortly afterward another patient was admitted to the Veterans Hospital with identical situations. Each patient had well-established Addison's disease, and both had developed acute respiratory infections. On admission, there was considerable confusion as to the needs of these patients because the serum electrolyte concentrations were entirely normal, but in spite of this the blood pressure was low and the patients were very ill. The immediate need for saline infusion was not recognized though it was given later. In both instances, there was a failure of recognition of the presence of hemoconcentration. The hemoglobin and hematocrit concentrations were significantly higher than they had been in the recent past. It is therefore important to remember that hemoconcentration can and does occur without abnormality in electrolyte concentrations, especially in patients who have been getting along well on ordinary maintenance doses of cortisone until onset of an infection. This difficulty can be quickly remedied. On the other hand, when most patients who are maintained on cortisone develop an infection or injury, the need is for more cortisone and not for large amounts of salt and water. Patients who have had total adrenalectomy are particularly prone to develop acute and rapidly progressive symptoms on cortisone withdrawal. This is not due to fluid loss and sodium loss but more likely due to an internal shift of water[4] with consequent decrease in concentration of sodium. Cortisone corrects this difficulty promptly.

Although corticotropin (ACTH) cannot be used in the treatment of primary adrenal insufficiency for obvious reasons, it is very useful in the diagnosis of adrenal cortical insufficiency. In the first place, the eosinophil response following the intramuscular injection of 25 U. of ACTH is fairly good as a screening procedure, but is likely to give false positive tests. A false negative test also occurs occasionally.[3] It is my impression that the incidence of false positive reactions (failure to produce a 50 per cent decrease in eosinophils in four hours) occurs in about 10 per cent of patients with various diseases not affecting the adrenal glands. Therefore the intravenous administration of 20 to 25 units of ACTH in 500 cc. saline solution over an eight-hour period is far superior. The determination of decrease of eosinophils offers a reliable qualitative appraisal of adrenal cortical function. There is a decrease of 75 to 100 per cent at the end of eight hours. Determination of the increase in excretion of 17-hydroxycorticosteroids or of 17-ketosteroids affords a better quantitative appraisal than the eosinophil counts.[7] When an eight-hour infusion is given on two successive days, the average normal rise of 17-hydroxycorticosteroids is 10 mg. for the first twenty-four hours and 20 mg. for the second twenty-four hours, and about half this much increase in 17-ketosteroids at the same time. In patients with adrenal cortical insufficiency secondary to pituitary failure or secondary to prolonged administration of cortisone or hydrocortisone, it is common to see little or no response to the first and even the second intravenous injection of ACTH. Maximal stimulation is obtained usually by the fourth day. ACTH gel in a dose of 30 to 40 units b.i.d. produces

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UNTOWARD EFFECTS OF CORTISONE AND CORTICOTROPIN ON THE GASTROINTESTINAL TRACT

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IN RECENT years, an increasing number of reports have appeared in the medical literature concerning serious gastrointestinal complications among patients receiving cortisone or corticotropin (ACTH). Some of these patients have had no previous symptoms or signs of gastrointestinal disease; others who were known to have gastrointestinal disease before treatment experienced exacerbations after treatment was begun. By far, the greatest number of reports are concerned with the development of new or recurrent gastric or duodenal ulcers.

In a careful, but by no means exhaustive, search of the medical literature, I was able to find reports of fifty-five cases in which either new peptic ulcer or serious exacerbations of peptic ulcer developed while the patients were taking cortisone or corticotropin or shortly after they discontinued its use (Table I). Of these, seventeen patients had gastric ulcers, twenty-five duodenal and two had both gastric and duodenal ulcers. Nine patients had "peptic ulcers" but the exact site of the lesions was not indicated. Two had jejunal ulcers. Almost half of the patients in the series (twenty-five of the fifty-five) had never had previous signs or symptoms of ulcer. The incidence of serious complications in these reported cases is high since either massive hemorrhage or free perforation occurred in about 45 per cent.

Because of the high incidence of peptic ulcer in the general population, and the great frequency with which cortisone and corticotropin are administered, the possibility must be considered that development of these ulcers may be the result of mere chance and that administration of the steroids has no causative role. This possibility seems unlikely, however. The great frequency of major complications (hemorrhage and perforation) among these patients in whom new and recurrent ulcers developed while being treated with cortisone or corticotropin would in itself seem to indicate some cause-and-effect relationship. Although my search of the literature disclosed only fifty-five cases of ulcer associated with this type of medication, it seems probable that many other cases of this type have gone unrecognized or unreported. If the association of the development of peptic ulcers with administration of these hormones is purely a matter of coincidence, it seems strange that no such association has been noted for other commonly used drugs, such as salicylates, laxatives and vitamin capsules, which the American public consumes in great quantities. Furthermore, there is good theoretic basis for the development of ulcers while patients are taking cortisone or corticotropin. Gray and his colleagues[9] showed that these hormones increase the fasting basal secretion of hydrochloric acid and pepsin into the stomach, and the degree of increase is comparable to that found in patients with active duodenal ulcers. From the evidence now available, we, therefore, must strongly suspect that there is a causative connection between the administration of corticotropin and cortisone and the development of peptic ulcers in some patients.

TABLE I. PEPTIC ULCERS DEVELOPING OR RECURRING IN COURSE OF CORTISONE OR CORTICOTROPIN THERAPY*

	Ulcer					
	Gastric	Duodenal	Gastric and Duodenal	Peptic (site not recorded)	Jejunal	Total
Cases	17	25	2	9	2	55
No previous ulcer	5	12	2	6	0	25
Reactivation of previous ulcer	12	13	0	3	2	30
Massive hemorrhage	3	3	2	3	2	13
Perforation	1	9	0	2	0	12

*Cases comprising this table were recorded in references 1-13; 16-20; 23; 26-29.

The percentage of patients who will experience symptoms from newly developed or reactivated peptic ulcers while taking these hormones is not definitely known. Halsted[12] estimated about 4

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per cent because symptoms and signs of ulcer occurred in sixteen of 360 patients who were receiving cortisone or corticotropin in his hospital. Freyberg and his associates[8] observed massive hemorrhage from the upper part of the gastrointestinal tract in two of forty-four patients being treated for rheumatoid arthritis. One of these patients had a duodenal ulcer; the source of the bleeding in the other patient was not discovered. Hollander[16] reported only four active peptic ulcers among 324 patients, most of whom had been taking one or the other hormone for short periods. The incidence of active ulcer was a little more than 1 per cent in this series.

Not all patients with known ulcers are made worse by corticotropin or cortisone therapy. Freyberg and associates[8] mentioned several such patients who had no flareup of ulcer symptoms, even with prolonged cortisone therapy. Sandweiss and his colleagues[26] cited the cases of two patients with active duodenal ulcer who became asymptomatic while taking these hormones. Of a group of eighteen patients with active gastric ulcers treated by Forbes,[7] three received corticotropin and eight cortisone. Seven patients received no special treatment and served as controls. All eighteen patients received the same diet which was free of irritating foods and which provided between-meal feedings. The ulcers of three patients treated with corticotropin failed to show improvement radiologically, and one of them became worse symptomatically. On the other hand, six of the eight patients treated with cortisone became free of symptoms of ulcer, and their ulcers healed completely. Healing of the ulcers also occurred in six of the seven patients who received no special treatment.

Just why there should be such a great difference in reaction to these drugs among patients receiving them, particularly among those with known ulcers, is a baffling question, indeed. It seems clear, however, that the effect of these steroids is only one of a number of factors operating to produce or to prevent the occurrence of peptic ulcers. Under certain circumstances and in certain individuals the hormones appear to play a determining role in producing ulcers. There seems to be little correlation between the dose of hormone administered and the occurrence of ulcers, although in most of the reported cases the patients have received more than 50 to 75 mg. per day. The duration of treatment also seems of little significance since some ulcers have flared up and perforated after only three to five days of treatment, whereas others have appeared after the drugs have been taken for many months.

Peptic ulcers have not been the only problem encountered. Perforation of involved segments of bowel while cortisone or corticotropin was being administered has been reported as occurring in at least eight cases of chronic ulcerative colitis and in two cases of regional enteritis.[13,14,21,27,28,31] Texter and his colleagues[24,30] have called attention to the occurrence of giant ulcers of the colon or rectum in patients with chronic ulcerative colitis. These large ulcers were found in five of fifteen patients receiving steroid therapy and were quite different in appearance and in structure from the ulcers usually seen in this disease when these drugs are not being administered. Sauer and his associates[27] encountered similar punched-out ulcers of the rectum in two patients being treated for rheumatoid arthritis who had never had previous rectal symptoms. A third patient observed by them had diffuse proctitis, while under treatment for rheumatoid spondylitis. Other intestinal complications recorded in the literature concern two patients who had perforations of the ileum and jejunum within one month after undergoing ileostomy for severe ulcerative colitis. The perforation was not closely associated with hormone therapy in one of these patients[13] since it occurred ten weeks after treatment with corticotropin was discontinued. The perforation occurred in the other patient[28] while corticotropin was being administered. A few other miscellaneous gastrointestinal complications of steroid treatment have been reported. These include a ruptured gall bladder,[22] a ruptured Meckel's diverticulum,[15] an exacerbation of ulcerative esophagitis[27] and the occurrence of phlegmonous gastritis.[25]

Ulcerations and perforations of the bowel in segments which are not exposed to gastric secretions suggest that mechanisms other than hypersecretion of gastric juice are operating to produce these lesions. Whether these mechanisms are related to the inhibition of inflammatory reactions or to the so-called antifibroplastic action which these hormones are thought to produce is at present a matter of conjecture. It seems possible that factors which promote resistance to ulceration in the normal gastrointestinal tract may be suppressed in some manner by these hormones.

Several authors have called attention to an absence of typical clinical signs of peritonitis in patients with free perforation of the gastrointestinal tract which occurred while they were taking these steroids. They mentioned particularly the absence of fever, abdominal rigidity and severe pain.[1,5,18,28] By no means have all perforations been characterized by such atypical clinical findings, however.[31]

Summary

Gastrointestinal complications occur in a small percentage of patients being treated with cortisone or corticotropin. The most common of these complications has been the development of or recrudescence of gastric or duodenal ulcers. Massive hemorrhage or free perforation has been associated in a high percentage of cases. Other gastrointestinal complications of steroid therapy include perforations of inflammatory lesions of the small and large intestine and the development of ulcers of the colon.

Cortisone and corticotropin should be administered cautiously, if at all, to patients with known peptic ulcers, and treatment for the ulcer should be carried out from the onset of steroid therapy, even if there are no symptoms or signs of ulcer activity.

The possibility of serious gastrointestinal complications should be kept in mind with respect to all patients who are receiving cortisone or corticotropin.

References

1. Beck, J. C., Browne, J. S. L., Johnson, L. G., Kennedy, B. J. and MacKenzie, D. W.: Occurrence of peritonitis during ACTH administration. Canad. M.A.J., 62:423-426 (May) 1950.
2. Bloomfield, A. L.: Untoward effects on the stomach of corticotropin and cortisone. (Editorial) A.M.A. Arch. Int.. Med., 90:281-283 (Sept.) 1952.
3. Bosien, W. R. and Tyson, M. D.: Spontaneous perforation of a benign gastric ulcer into the transverse colon: report of a case. Gastroenterology, 24:113-117 (May) 1953.
4. Bulova, P. N., Becker, F. P. and Maslon, Morris: Gastroduodenal ulcer following cortisone therapy: report of a case with fatal hemorrhage. New York State J. Med., 52:1181-1182 (May) 1952.
5. Cleghorn, R. A.: Discussion. In Mote, J. R.: Proceedings of the Second Clinical ACTH Conference. Vol. 2. Therapeutics, p. 503, Philadelphia: The Blakiston Company, 1951.
6. Davis, T. A. and Zeller, Michael: Multiple peptic ulcers with massive hemorrhage during oral cortisone therapy: report of a case. J.A.M.A., 150:31 (Sept. 6) 1952.
7. Forbes, John: Inadvisability of giving ACTH to patients with gastric ulcer. Lancet, 2:555-557 (Sept. 20) 1952.
8. Freyberg, R. H., Traeger, C. H., Patterson, M., Squires, W., Adams, C. H. and Stevenson, C.: Problems of prolonged cortisone treatment for rheumatoid arthritis; further investigations. J.A.-M.A., 147:1538-1543 (Dec. 15) 1951.
9. Gray, S. J., Benson, J. A., Jr., Spiro, H. M. and Reifenstein, R. W.: Effects of ACTH and cortisone upon the stomach: its significance in the normal and in peptic ulcer. Gastroenterology, 19:658-673 (Dec.) 1951.
10. Habif, D. V., Hare, C. C. and Glaser, G. H.: Perforated duodenal ulcer associated with pituitary adrenocorticotropic hormone (ACTH) therapy. J.A.M.A., 144:996 (Nov. 18) 1950.
11. Hall, B. D.: Gastric ulcer associated with cortisone therapy. Gastroenterology, 25:80-83 (Sept.) 1953.
12. Halsted, J. A.: The effects of ACTH and cortisone in diseases of the digestive system. M. Clin. North America, 32:1069-1081 (July) 1952.
13. Halsted, J. A., Adams, W. S., Sloan, Sol, Walters, R. L. and Bassett, S. H.: Clinical effects of ACTH in ulcerative colitis. Gastroenterology, 19:698-721 (Dec.) 1951.
14. Halsted, J. A., Adams, W. S., Sloan, Sol, Walters, R. L. and Bassett, S. H.: Clinical effects of ACTH in ulcerative colitis. In Mote, J. R.: Proceedings of the Second Clinical ACTH Conference. Vol. 2. Therapeutics. pp. 489-500. Philadelphia: The Blakiston Company, 1951.
15. Hanlon, C. R.: Discussion. Surgery, 28:180-181 (Aug.) 1950.
16. Hollander, J. L.: Discussion. In Mote, J. R.: Proceedings of the Second Clinical ACTH Conference. Vol. 2. Therapeutics. pp. 503-504. Philadelphia: The Blakiston Company, 1951.
17. Kirsner, J. B., Klotz, A. P. and Palmer, W. L.: Unfavorable course of gastric ulcer during administration of ACTH and cortisone. Gastroenterology, 20:27-29 (Jan.) 1952.
18. Lubin, R. I., Misbach, W. D., Zemke, E. M., Hammarsten, J. F. and Heller, B. I.: Acute perforation of duodenal ulcer during ACTH and cortisone therapy. Gastroenterology, 18:308-312 (June) 1951.
19. Mandel, William, Singer, M. J., Gudmundson, H. R., Meister, Lester and Modern, F. W. S.: Intravenous use of pituitary adrenocorticotropic hormone (ACTH): a report of its administration in twenty-five patients. J.A.M.A., 146:546-551 (June 9) 1951.
20. Moe, A. E.: Gastric ulcer appearing during cortisone administration: report of a case. Gastroenterology, 20:343-347 (Feb.) 1952.
21. Palmer, W. L.: Discussion. Gastroenterology, 19:-733-734 (Dec.) 1951.
22. Reiff, Sidney: Acute suppurative cholecystitis, with rupture of gall-bladder and liver abscess formation, during administration of cortisone. Ann. Int. Med., 38:1313-1315 (June) 1953.
23. Reynell, P. C., Martin, E. A. and Beard, A. W.: Monilia peritonitis. Brit. M. J., 1:919 (Apr. 25) 1953.
24. Ruffin, J. M.: Corticotropin (ACTH) and cortisone in the treatment of ulcerative colitis. (Editorial) Gastroenterology, 26:113 (Jan.) 1954.
25. Rutherford, P. S. and Berkeley, J. A.: Phlegmonous gastritis in a cortisone-treated patient. Canad. M.A.J., 69:68-69 (July) 1953.
26. Sandweiss, D. J., Saltzstein, H. C., Scheinberg, S. R. and Parks, A.: Hormone studies in peptic ulcer: pituitary adrenocorticotropic hormone (ACTH) and cortisone. J.A.M.A., 144:1436-1442 (Dec. 23) 1950.

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STEROID THERAPY IN DERMATOLOGY

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FOLLOWING the introduction of hydrocortisone not only the initial claims for its usefulness but also the early observed results have been so encouraging that some have inquired whether there will any longer be need for dermatologic training, suggesting that the general practitioner may now be able to cure all dermatoses with this drug. This, of course, is a gross exaggeration: there is, however, no doubt that the application of hydrocortisone is extremely effective in providing relief from itching. In so doing, it prevents scratching, and in many instances an eruption then heals. Such a sequence is particularly likely in dry, localized neurodermatitis (lichen simplex chronicus) where the persistent eruption has been largely due to habit scratching. Improvement also occurs in other forms of atopic dermatitis, if the cause has been removed. If eczema were all "allergic" and if one could depend upon allergic study and the subsequent elimination of causes, then it would be very simple, indeed, to cure. Topical use of hydrocortisone is less satisfactory in non-allergic dermatoses and is of practically no value in chronic discoid lupus erythematosus, psoriasis, and many other disorders. It is not useful when infection is present, unless one combines an antibiotic agent as is done by one manufacturer in Neo-cortef, which contains neomycin and hydrocortisone. Even such a combination will not prove satisfactory if the infection is mycotic.

Topical Therapy

Perhaps the best indication for topical hydrocortisone is a dry itching localized eczematous eruption on the lids, at the nape, in the axillas, or in the anogenital region. Even in these instances, such treatment is obviously not going to be lastingly effective if, for example, the cause of the eruption on the lids is a continued exposure to a contact allergen (either direct or airborne): or in the axillas if the process is primarily, or has become secondarily, infected with dermatomycosis, or is seborrheic dermatitis; or in the anogenital area if a local cause is allowed to persist, if diabetes is present and uncontrolled, or if infection has not been recognized and properly treated. In any case, it is quite evident that the topical use of hydrocortisone does not lessen the initial need for accurate dermatologic diagnosis; for the ability to recognize the presence of, and the type of, cutaneous infection which may occur primarily or as a complication; or for the knowledge concerning when to stop a treatment which has fulfilled its purpose and what to add when improvement has ceased.

For topical dermatologic application, the choice seems to be a 1 per cent ointment (the 2.5 per cent preparation being more expensive and little, if any, more effective). The useful preparation is hydrocortisone and not cortisone, which is completely ineffective on the skin. One argument against topical use of hydrocortisone is the high cost; another is its failure to control infection.

Systemic Administration

With systemic administration of steroids in dermatology, the usual preparation is cortisone, though hydrocortisone can be used and smaller dosage is effective, and ACTH is available for use by injection in acute emergencies or in instances where the oral drugs have not proved effective. Given systemically, these drugs are effectively anti-pruritic; however, their use is justified only if the physician also takes steps to cure the disease. The drugs do not relieve all types of itching. Their effect probably occurs chiefly by minimizing or preventing the inflammatory reaction which causes the itching, and by preventing the inflammation which results from excoriation.

A. Allergic Dermatoses.—

1. Systemic administration is very effective in relieving acute contact dermatitis when too extensive for topical treatment to be practical and too severe to allow complete dependence on older methods.

2. In chronic contact dermatitis which has gone on to eczematization and is persisting after

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removal of the initial cause, one often gets symptomatic relief from steroid therapy and the older methods, topical or radiotherapeutic, then become more effective in healing.

3. In atopic dermatitis (disseminated neurodermatitis), steroids are helpful as a temporary expedient, but the tendency has been to use the drugs too frequently and in too great dosage. The dermatologist frequently sees patients whose eruptions are worse after the treatment has been discontinued than they were before it was instituted. (In localized atopic dermatitis—localized neurodermatitis—topical administration is preferable to systemic administration and has already been discussed.)

4. Steroid therapy is very useful in relieving the symptoms of exfoliative dermatitis (erythroderma), but during this initial period of improvement one should make every possible attempt to find and eliminate or treat the cause of this disabling condition. In the majority of instances, exfoliative dermatitis is a form of atopic eczema and therefore subject to the suggestions made above. Other forms of eczema are less responsive to steroid therapy, which should not be used in infectious eczematoid dermatitis of either bacterial or mycotic origin. It is of doubtful value in seborrheic dermatitis, at least partly because of the infectious factors which are so often present. It is of little or no value in stasic dermatitis, unless there has been acute generalization of the eruption; such a reaction will subside on steroid therapy, but at the same time one must treat the localized stasic dermatitis and search for and treat its causes.

5. Steroid therapy is often very useful in the allergic reactions to drugs, particularly when these are acutely urticarial or angioneurotic, but also in eczematous reactions to drugs.

6. Urticaria in any form is usually relieved by steroids, if the dosage is sufficiently high. This treatment is indicated only when urticaria is acute and severe. In chronic urticaria, steroids may be worth a short trial, but there are numerous failures to obtain lasting help without undue continuation of therapy.

B. In Certain "Possibly Allergic" Reactions to Infections.—

1. Steroid therapy relieves erythema multiforme, but such treatment is indicated only when the reaction is particularly acute and severe, as in the more toxic, bullous condition which is known as Stevens-Johnson syndrome.

2. Treatment also is quickly effective in erythema nodosum in many instances. However, there is question as to the wisdom of such treatment because one has not treated the cause of the eruption, and the patient thus has false encouragement, not realizing the serious nature of the disease once the dermatologic symptoms have been relieved. It is perhaps better to treat these patients with rest and salicylates while searching for the cause in the usual manner, suspecting drugs and infections of various types, particularly tuberculosis and the rheumatic group.

C. Lupus Erythematosus.—

Neither topical nor systemic administration of steroids is of help in chronic lupus erythematosus. In the otherwise fatal acute disseminated form of lupus erythematosus, we have one of the best indications for steroid therapy in the entire field of medicine. Systemic administration does not lead to cure, but one frequently observes prolonged remissions; only rarely is improvement maintained without continuous or intermittent prolonged administration of the steroids. Subacute lupus erythematosus is difficult to define but, generally, the term is used when the patient is able to be ambulant but manifesting clinical and laboratory evidence of visceral or systemic involvement. In these patients, one hesitates to start steroid therapy because, once started it is extremely difficult to stop without allowing the patient to go rapidly downhill in an acute exacerbation of the disease.

D. Pemphigus.—

Before starting treatment for pemphigus, it is essential that accurate diagnosis be established because other dermatoses can mimic this serious systemic and bullous disease. Steroid therapy is not useful for dermatitis herpetiformis, which may mimic pemphigus, and gives variable results in a bullous condition which is a variant of pemphigus and for which the term "pemphigoid" has recently been gaining popularity. In true pemphigus, the steroids are distinctly not curative but are very helpful and can produce prolonged remissions which in some patients will be main-

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THE PATHOLOGIST AS A PRACTITIONER OF MEDICINE

Fifty Years of Progress

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DR. ARTHUR H. SANFORD has been for years a prominent influence in my life, as he has been in yours. As a medical student, and later as a resident in pathology, I used a clinical pathology textbook co-authored by Dr. Sanford. When I received my certificate as a Diplomate of the American Board of Pathology, Dr. Sanford's signature appeared on it as the president of the Board, incidentally its first president. I am deeply honored to be chosen to further immortalize his illustrious name, but do not find it easy to rise to this occasion.

The title which I have chosen is in part fact and in part fiction. The fact has to do with the progress of the clinical pathologist as a practitioner of medicine, and the fiction has to do with the reference to the fifty years. This reference is purely journalistic license, but in general, fifty years ago marks the time when the pathologic anatomist emerged from the static atmosphere of the dead-house and from the formalinized interpretation of sections of tissue which in his mind were both physically and philosophically detached from the patient. The studies made by the pathologic anatomist were necessary as a basic foundation, but a superstructure was needed if the living patient was to benefit from these observations; hence, the clinical pathologist was born. These pioneers in clinical pathology realized that there were many discoveries in the allied basic science fields, such as chemistry, anatomy, bacteriology, and physiology, and that a co-ordinator was needed to interpret them and harness their usefulness in order that physicians might practice better medicine. The clinical pathologist, if he is worthy of his name, must have the basic attitude that he is a practitioner of medicine; a physician who uses laboratory tools instead of surgical instruments; a physician who, when he examines the tissue section, thinks of a living patient instead of a histologic problem which he must solve by attaching an appropriate name; a physician who must be able to analyze a patient's history and the significant physical findings in order that he may help his colleagues arrive at a proper diagnosis and treatment by a helpful interpretation of the laboratory findings; a physician who, when he reports the findings at necropsy, must be able to interpret them in the light of chronological development of the disease process and the possible effects, good or bad, produced by the therapy. The static pathologist, the detached recorder of anatomic alteration, is not useful in the daily practice of medicine.

To you, Dr. Sanford, and a few hardy pioneers of your day of approximately fifty years ago, goes the credit for creating this indispensable "tool" in the practice of medicine.

I shall attempt to give you a panoramic sketch of a few of the everyday clinical-pathologic procedures with illustrations of their usefulness. These will be discussed under seven headings, for the purpose of presentation, and not with the idea of defining the scope of the field of clinical pathology.

Pathologic Anatomy

The establishment of hospital standards by the American College of Surgeons included the two most significant steps which were taken in this field to improve patient care—a minimal autopsy percentage of 18 per cent, recently elevated to 25 per cent, and a routine examination of all surgically removed tissues. Out of this grew the widespread use of the biopsy as a diagnostic method, an important weapon in the hands of all physicians in the daily practice of medicine. There are still occasional flurries of controversy about the necessity for routine examination of tissues, but to be convinced one needs only to see a few examples—such as a fibroid uterus which microscopically showed an unsuspected carcinoma of the cervix; a labial ulcer which was removed without being examined histologically and was followed by metastatic squamous cell carcinoma in the inguinal lymph nodes a year later; a "benign" mole which was removed by electro-coagulation without tissue examination and subsequently appeared in the regional lymph nodes as a metastatic melanoma. These and numerous other

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examples adequately demonstrate the need for routine microscopic examination of all surgically removed tissues. Patients are becoming more aware of the necessity of this procedure.

With the advent of the Tissue Committee, created by the Joint Commission on Accreditation of Hospitals, new controversies have arisen. These are based largely upon the attitude of many surgeons who feel that they are the objects of discriminatory surveillance, whereas their clinical colleagues, such as internists and pediatricians, go relatively scot-free. The controversy, of course, is understandable, but a properly functioning Medical Records Committee should go far in minimizing this difference.

The smear technique for the examination of body fluids and secretions, re-emphasized by Dr. Papanicolaou, has become a very useful tool, but its scope of usefulness is narrower than was hoped for. In general, the Papanicolaou technique is most useful in the early diagnosis of carcinoma of the cervix, carcinoma of the bronchus, and the examination of fluids from serous cavities. One is occasionally forced to treat a suspected carcinoma of the bronchus without either a positive Papanicolaou smear or a positive biopsy. This should never be done in the case of the cervical disease. A positive cervical Papanicolaou smear should always be followed by a biopsy before therapy is commenced. The cell block technique is more valuable than the Papanicolaou method for the examination of fluid from serous cavities. This consists of the formalin fixation of the centrifuged concentrated sediment followed by the preparation of the microscopic slide by the ordinary paraffin technique. More accurate results are obtained than by the smear method. We have had similar experience with bronchial secretion, when a sufficient quantity is present.

Aspiration biopsy by a Silverman type of needle has become an important diagnostic method, particularly applicable to areas where direct biopsy is difficult to obtain. Liver biopsies by this method are not at all difficult. They are usually done, however, only in those cases where there is an enlargement of the liver and no evidence of bleeding tendency. Successful biopsies of this type have also been made of deep-seated bone lesions, the spleen, and the lung. Usually the biopsy of a breast lesion by this method is not indicated, especially when the entire lesion may be excised.

Hematology

Of the numerous discoveries that have been made and have eventually been adopted as useful in this field, perhaps the bone marrow examination is the most important. One can hardly think of a disease problem involving the hematopoietic or the reticulo-endothelial system without also thinking in terms of the bone marrow findings. All leukemias, except chronic granulocytic leukemias, can be diagnosed much earlier and more accurately by bone marrow examination. This is particularly true in children, in whom many begin as leukopenias and present a clinical picture of some type of infectious disease. A marrow examination may be absolutely essential in some cases of infectious mononucleosis in order to avoid the horrible mistake of making an incorrect diagnosis of leukemia. All of the lipid and non-lipid reticulo-endothelial diseases, such as Hand-Schuller-Christian disease, Niemann-Pick Letterer-Siwe, and Gaucher's disease, are often first diagnosed by marrow examination.

The technique is relatively simple. Although some people prefer the iliac crest or the spinous process of the vertebra as a site, I still prefer the sternum, even in children. It requires a sternal needle and a large syringe, preferably 20 cc., and a sudden, sharp aspiration which creates sufficient vacuum to break down the marrow trabeculae in order to obtain marrow tissue; otherwise, venous blood is obtained. Local anesthesia is all that is necessary, even in most children.

In recent years, a very important new concept of hematology has been advanced, that is, the concept of hypersplenism. It has been known for many years that some patients suffering from thrombocytopenic purpura were benefited by splenectomy, but there was no method of determining which patients would benefit and which might not benefit by this procedure. In the case of thrombocytopenic purpura, the bone marrow examination is essential in the differentiation of the primary from the secondary type. In primary thrombocytopenic purpura the spleen is not enlarged.

Other types of hypersplenism are generally associated with the secondary effects of splenomegaly. In this condition, any or all of the formed marrow-derived elements of the peripheral blood may be decreased in numbers. It is not known whether the reduction is the result of the hyperactive destructive action of the spleen, the production of some hormone which depresses the ability of the bone marrow to release a sufficient number of elements, or an increased antibody formation in the spleen, which in turn may destroy platelets, red cells, or even white cells. Nevertheless, in each patient in whom this problem exists—that is, an enlarged spleen and peripheral blood evidence of a partial or a pancytopenia . . . a marrow examination is essential to determine whether the disease is or is not primarily one of marrow origin. If marrow elements are present in adequate numbers, many of these cases are benefited by splenectomy; if not, splenectomy might be fatal because the spleen may be the remaining major manufacturing plant for hematopoietic elements.

The hemophiliac can now live a much more comfortable existence; although his disease cannot be cured, it at least may be controlled. The discovery of the lack of hemophiliac globulin, that is the anti-thromboplastinogen, and its correction by the administration of freshly drawn blood has saved many a hemophiliac's life. This hemophiliac factor is a highly labile factor which soon disappears from bank blood. The same result may be accomplished by the administration of this specific globulin fraction of frozen plasma but fresh blood is often more readily available.

The L. E. cell, the diagnostic cell of disseminated lupus erythematosus, was first found in incubated, heparinized bone marrow. The demonstration of this phenomenon is now simplified by the thirty-minute incubation of whole venous blood, straining the cloth through a 40-mesh screen, centrifugalization of the fibrin free blood suspension, and staining the smears made from the buffy coat. This method is just as accurate as the marrow method.

Parasitology

Many advances have been made in this field, but compared with others, their application is limited in the daily practice of medicine in the temperate zone. Perhaps the most important is amebiasis. This was forcibly called to our attention as a non-tropical disease when faulty plumbing got the sewage mixed up with the drinking water in a rather well-known hotel and in addition, a few carriers contaminated the salads. This incident made us all aware that amebiasis may be seen anywhere, temperate or tropical, and we must be prepared to detect it in the laboratory. Unfortunately, the methods are not as good as desirable. The fact that parasitologists agree that at least three stools must be examined for cysts and vegetative forms before they can be called negative, is an indication that our present methods are far from good. The value of the complement fixation test has not been established.

During World War II, we in the temperate zones observed some hookworm and malarial infections that had little chance for survival because the natural surroundings are not conducive to the propagation of these diseases. The little, old, itchy pinworm, however, is a perennial problem. Among children between the ages of five and fifteen, the infection rate is between 25 and 50 per cent. Stool examinations, of course, are of no value because the female deposits her ova on the perianal perineum. The scotch tape method of picking up the ova from the early morning, unwashed perianal region is still the best method.

Clinical Chemistry

No vast improvements have been made in the routine examination of gastric juice, urine, or feces. Perhaps the most useful is the development of the test tablets for the detection of sugar and acetone in the urine and for blood in the stools. These go far to simplify the procedures in the doctor's office. The tablet for the detection of blood in the stool is of great value because it usually does not require that the patient be on a meat-free diet prior to the test.

Physiological Chemistry

For practical purposes, the field of applied physiological chemistry was born and raised during the last fifty years. It probably has not reached puberty. I shall only mention a few procedures, most of them are so well established that they need no comment.

The prothrombin time at first found its application in hemorrhagic disease of the newborn, in which it is prolonged during the first ten days of life. It has become a necessary daily procedure in all laboratories because dicumarol is now widely used in an attempt to prevent thrombo-embolic processes, particularly in coronary heart diseases. It is also extremely useful in the bleeding problem of obstructive jaundice.

The chemical tests for the determination of liver function are still in their infancy. A battery of tests, such as the direct and indirect VandenBergh, the thymol turbidity, the cephalin flocculation, the bromsulphalein excretion tests, the prothrombin time, the test for bile in the urine, urobilinogen studies, the total serum protein and A-G ratio, alkaline phosphatase, and blood cholesterol are collectively often referred to as a liver profile. When properly interpreted in the light of the clinical findings, this has taken much of the guesswork out of the cause of the individual case of jaundice. Many people whose jaundice is due to an infectious hepatitis may have an accurate diagnosis and consequently not be subjected to an unnecessary, hazardous, exploratory operation.

A total serum protein determination is also extremely helpful in older patients who are subjected to extensive surgery. A low level of total serum protein, as well as the abnormal ratio of albumin to globulin, reflects a lack of adequate general resistance of the body because we know that the formation of immune bodies and other defense mechanisms are intimately associated with adequate available protein.

In cardiac, renal, intestinal, pulmonary, and adrenocortical diseases and in pre- and postoperative surgical problems, especially of the gastrointestinal and biliary tracts, the electrolyte, acid-base, and water balance can now be properly assayed through the use of the carbon dioxide combining power, the plasma chlorides, the blood sodium and potassium. The designation of the quantitative level of these chemicals in milliequivalents instead of milligrams per cent aids materially in the understanding and the solution of an acid-base problem. This is based upon the fact that the chemical substances of the blood which maintain a normal acid-base balance do not react with each other gram for gram, but in proportion to their equivalent atomic weights.

The determination of urinary ketosteroids has been important in removing the mystery surrounding many of the endocrine problems, particularly as they relate to the hypophysial, adrenal, and gonadal disturbances.

Serology

About the turn of the century, it was discovered that serum from immune animals destroyed the bacteria to which they were immune. The application of this principle as an aid in the prevention or diagnosis and/or treatment of such diseases as typhoid fever, shigellosis, tularemia, brucellosis, syphilis, and many others, has made it possible virtually to eliminate them as public hazards. Syphilis, a disease in which the only carrier is the human, has also been virtually conquered in this country by vigorous widespread application of case-finding methods and through the use of serological tests. It seems a tragedy that another widespread, largely human-borne disease, tuberculosis, has not also been eliminated since it may be easily diagnosed through a combination of an immunologic principle, the tuberculin test, and the x-ray.

Many viral diseases can now be detected through the medium of serologic tests, but these tests are not yet generally available.

Lansteiner's discovery, about fifty years ago, of the non-bacterial antibody, the iso-agglutinin, was the next important milestone in this field of applied immunology. This led directly to the discovery of the naturally occurring blood group and antibody systems which are now known as the ABO system, the M & N system, and the Rh and Hr system. Evidence has recently been discovered that we also have a platelet group system. From a practical standpoint, of course, this discovery permitted the use of blood transfusions as proper therapy for many diseases characterized by blood loss, and it also permitted extensive surgical procedures because sufficient quantities of blood could be safely administered to a patient. The discovery that a pregnant Rh negative mother could be iso-immunized by her Rh positive fetus explained a complicated, baffling disease which was known then as erythroblastosis fetalis, now called hemolytic disease of the newborn. In fact, the hemolytic action of the maternal anti-Rh positive antibodies upon the red cells of the fetus was found to be responsible for four apparently different disease processes: erythroblastosis fetalis, icterus gravis neonatorum, fetal hydrops, and purpura of the newborn. A prenatal work-up is no longer complete unless Rh blood grouping of the pregnant woman and her spouse is done. Detection of the Rh antibodies in the maternal circulation during the pregnancy is a warning that preparation should be made for a replacement transfusion at the time of birth. Fortunately, the Rh antigen is a very poor antigen and only iso-immunizes the Rh negative mother sufficiently to cause the fetal disease in about one of fifty pregnancies.

Soon after the Rh iso-immunization phenomenon was discovered, it was found that there were two types of antibodies in the serum—an agglutinating bivalent antibody which was easily detected *in vitro* because it clumped the Rh positive red blood cells in saline dilution, and another one, the univalent blocking antibody, which coated the red blood cells but did not cause them to clump in a saline dilution. A simple method had to be developed to detect the presence of this blocking antibody.

Based upon the fact than an antibody is basically globulin, Coombs and his associates developed an anti-globulin immune serum which caused clumping of those red cells which were coated with blocking antibodies. This principle is being applied to many other iso-immunization problems, particularly in acquired hemolytic anemias in which it has been discovered that many such patients show red cells coated with blocking antibodies. This is not true of the congenital spherocytic form of hemolytic anemia.

The principle of the Coombs' test will probably be used in the development of laboratory methods for the detection of auto-immunization which is the production of immune bodies in response to an antigen that normally exists within the human body. This is in contrast to iso-immunization when a foreign antigen is introduced as in the case of an Rh positive antigen into an Rh negative recipient. The hemolytic process where cold agglutinins clump the patient's own red cells is an example of auto-immunization. It is felt by several investigators that there are immune bodies in some cases of thrombocytopenic purpura which actively destroy the blood platelets. A practical test has not been developed to separate those purpuras in which an immune body is operative.

An important auto-immunization mechanism probably is involved in the so-called collagen diseases: disseminated lupus erythematosus, periarteritis nodosa, rheumatoid arthritis, rheumatic fever and glomerulonephritis. The L. E. phenomenon in lupus erythematosus, with nuclear substances phagocytosed by the patient's leukocytes, may be an example of auto-immunization. A disease resembling human glomerulonephritis has been produced experimentally in rabbits by injecting anti-rabbit kidney immune serum.

Medical Microbiology

No clinical pathology laboratory today can exist without a vigorous medical microbiology division. Dr. Alexander Fleming's accidental discovery in his microbiologic laboratory resulted in the greatest medical benefit to mankind during the last fifty years. Following his notation that some bacteria would not grow on artificial media in the immediate zone about the colony of a common contaminating mold, *Penicillium notatum,* the story of the development of antibiotics is unbelievably fantastic. The clinical pathology laboratory, by the use of sensitivity tests, aids the physician in the selection of the proper antibiotic. These have proved very helpful despite the fact that for some time there was doubt whether the *in vitro* demonstration of sensitivity was applicable *in vivo.*

Improved routine laboratory methods have helped immeasurably in the study of fungus diseases. Perhaps the most practical help can be obtained by the isolation of the fungus responsible for a given case of dermatomycosis. The discovery that there is a vast reservoir of *Coccidiodes immitis* organisms on natural vegetation and a *Histoplasma capsulatum* reservoir in animals was highly significant. Skin sensitivity tests for both of these diseases have revealed that there are many non-fatal subclinical cases similar, in that respect, to tuberculosis.

The diagnosis of viral disease by routine laboratory methods is not yet at hand. The need for extensive animal innoculations, ultra and refrigerated centrifuges, electronic microscopy, and highly technical filtrations makes it impossible for the laboratory of the average clinical pathologist to be useful in this field.

Conclusion

If I have succeeded in creating in you a feeling that the laboratory is another facet in the practice of medicine and not merely some remote, busy, isolated group of rooms where urine is being boiled, blood is being examined, and tissues are being cut, all in an air of total detachment from the problems of the patient, then the purpose of this talk has been accomplished. That would be a most fitting tribute to Dr. Arthur H. Sanford, physician, scholar, scientist, investigator, author, and gentleman.

CANCER OF THE CERVIX

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A BASIC concept in cancer is that the key to better results is earlier diagnosis, and more prompt and adequate treatments. Programs of lay and medical education have made significant gains upon the objective of diagnosis despite the fact we have only a few tools that aid in early recognition. For cervical cancer the Shiller test, in use for a period of approximately thirty years, was the first important development. More recent is the use of smears for examining vaginal secretions microscopically. As a result, there is now improvement in quality of clinical material. There is also advance in technique of treatment. If to some the speed of advance seems slow, they need be reminded that our predecessors included competent surgeons and diagnosticians, and that empiricism derived from trial and error in radiotherapy forms the basis for greater precision in modern treatment.

We have not equally improved estimation of prognosis. To be sure, the early case presents a better outlook than more advanced forms, but knowledge remains vague for explaining failure in a favorable lesion, or success in a late growth. Error in estimating stage of advance might be suspected as a significant factor. Accessibility of normal pathways of growth to palpation makes practical the classification of cervical cancer into different stages of advance, but error is inescapable due to difficulty in recognizing small extensions or dispersions, and to confusion in distinguishing induration of tumor from that due to infection. Henriksen estimates error to be on the order of 25 per cent. That magnitude is of significance, but in considering the broad spectrum of induration in question, it does not explain adequately the inaccuracies in estimating prognosis.

In pursuit of the question, it is pertinent to examine results in two independent series of patients treated by a medical staff that is essentially overlapping. During the years 1935-1947, a total of 452 ward and private patients with cervical cancer were treated at the Barnes Hospital, and 990 patients at the Barnard Free Skin and Cancer Hospital. Results are given in Table I. It can be seen that the total five-year survival free of recurrence is 30 per cent, but the value for Barnes is 40.5 per cent, and that for Barnard only 25.1 per cent. Since the majority of individuals responsible for treatment are staff members at both hospitals, there must be intrinsic reasons for the wide difference in end results.

TABLE I. FIVE-YEAR RESULTS IN CERVICAL CANCER TREATED AT THE BARNES AND AT THE BARNARD HOSPITALS

Hospital	Number of Patients	Alive and well at 5 years Number	Per Cent
Barnes	452	183	40.5
Barnard	990	249	25.1
Total	1442	432	30.0

The International Classification is employed for both series. Results according to stage of advance for the Barnes patients are given in Table II. Four patients with intra-epithelial cancer are included in Stage I. Five-year values for each stage are in good agreement with those generally reported, with the exception of Stage IV in which no survivals are shown. That is believed largely due to conservative classification. Patients have not regularly been cystoscoped or proctoscoped. Qualification for Stage IV has been evidence of extension outside the birth canal as noted biman-

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ually, radiographically, or on general physical examination.

TABLE II. FIVE-YEAR RESULTS IN THE BARNES SERIES ACCORDING TO STAGE OF ADVANCE

(International Classification)

League of Nations Classification	Number of Patients	Alive and well at 5 years	
		Number	Per Cent
I (20 per cent)	90	64	71.1
II (46.3 per cent)	209	98	47.3
III (26.2 per cent)	119	21	17.6
IV (7.5 per cent)	34	0	0
	452	183	40.5

TABLE III. FIVE-YEAR RESULTS IN THE BARNES SERIES ACCORDING TO INFECTION

Patients	Per Cent Alive and Well at 5 Years League of Nations Classification							
	I		II		III		IV	
Total Series 183/452	64/90	71.1	98/209	47.3	21/119	17.6	0/34	0
Infected in Total Series 35/170	7/13	53.8	22/71	31	6/62	9.7	0/24	0

Infection is known to affect adversely the response to irradiation. The attempt has been made to evaluate the effects of infection upon results in the Barnes patients, and those data are given in Table III. Lesions were classed as infected if mention of that complication was made in the clinical record, or if there was an appreciable amount of fever during hospitalization for radium treatment. For the entire series only thirty-five (20.6 per cent) of the 170 infected patients survived five years free of recurrence. For each stage the values fall below the average for the group in question. Thus, it appears evident that infection affects prognosis. A substantial segment of the patients were treated during a period antedating modern antibiotics. It is possible that future statistics will show a lesser effect for infection.

Relationship has been demonstrated for gross architecture of tumor and end results. Primary lesions can be classified into everting, infiltrating, and cratered forms. The everting, or exophytic type, is the typical cauliflower growth composed of numerous papillary excresences with marked friability and a rich blood supply. Infiltrating forms expand the cervix by growth beneath a more or less intact covering epithelium, and produce a firm, hard, nodular type of lesion. Those often begin as endocervical growths, and in more advanced stages present a hard collar of tumor replacing the cervix and filling all vaginal fornices. Catered lesions are formed as the result of tissue loss due to slough following necrosis. They are severely infected, and tend usually to be the more advanced forms of everting types. Results according to clinical type of growth are given for the Barnes series in Table IV. In recalling that the average five-year result for the entire series is 40.5 per cent, it can be seen that values for specified gross forms are 54.4 per cent for everting lesions, 39.1 per cent for infiltrating tumors, and 21.1 per cent for cratered varieties. In the same table, values are given for each stage. In each instance, the everting tumors present a result better than average for the stage in question. Other forms tend to fall below average.

There is evidence, therefore, that character of growth pursued by the tumor has certain relationship to end results. It is possible that variation in clinical response may be due in part to differences in tendency toward spread by direct extension or by dispersion. Marked infection of cratered forms must have some bearing upon lesser results in these lesions.

TABLE IV. FIVE-YEAR RESULTS IN THE BARNES SERIES ACCORDING TO CLINICAL TYPE OF GROWTH

Clinical Type	Per Cent Alive and Well at 5 Years League of Nations Classification									
	I		II		III		IV		Total	
Everting	27/37	73	37/69	53.6	4/17	23.5	0/2	0	68/125	54.4
Infiltrating	23/34	67.6	38/87	43.7	13/54	24.0	0/14	0	74/189	39.1
Cratered	4/9	44.4	13/38	34.2	2/31	6.4	0/12	0	19/90	21.1
Unclassified	10/10	100	10/15	66.6	2/17	11.8	0/6	0	22/48	46.0
Total	64/90	71.1	98/209	47.3	21/119	17.6	0/34	0	183/452	40.5

TABLE V. AVERAGE FIVE-YEAR RESULTS IN BARNES AND BARNARD SERIES ACCORDING TO CLINICAL TYPE OF GROWTH AND INFECTION

Per Cent Alive and Well at 5 Years

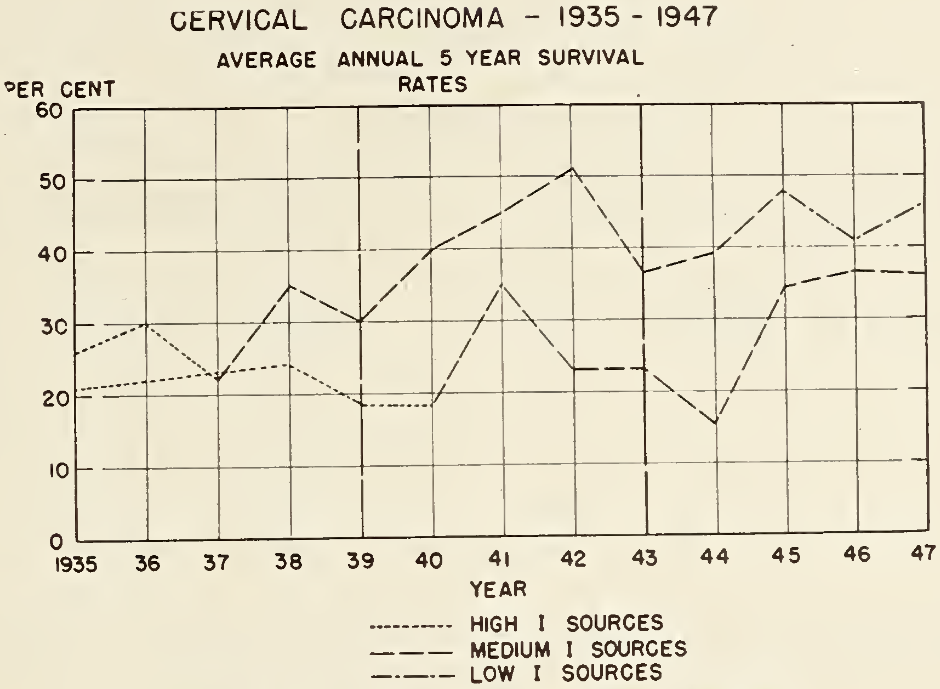
Hospital	Everting	Infiltrating	Cratered	Unclassified	Infected
Barnes	54.4	39.1	21.1	46.0	20.6
Barnard	22.5	20.0	22.0	48.0	9.0

Upon accumulation of data given above for the Barnes series showing the better average result, it becomes pertinent to make comparison with the Barnard patients. Those data are given in Table V. It can be seen that effect of gross architecture is obliterated in the Barnard series. Values for everting, infiltrating, and cratered forms are not significantly different from the average survival rate of 25.1 per cent for the entire series. That is not found true for infection. Fall in survival for infected lesions is greater in the Barnard patients, the value being only about one-third that for the total group. In the Barnes series, the value for infected lesions is one-half the average. Explanation for those phenomena is vague, but it seems probable that factors were present that decreased resistance to infection in the Barnard patients, and that those, or other factors, obliterated significance of gross architecture.

It now becomes important to compare over the period in question any fluctuations in yearly survival, differences in treatment, or alterations in type of material for the two series of patients. Those data can best be presented by the graph shown in Figure 1. Yearly survival curves are drawn in dots, dashes, and a combination of the two. Progression in that order respresents successive decreases in intensity of radium treatment. At the outset, strengths were usually 25 mg. and 50 mg. In more recent years, tubes of about 5 mg. and 10 mg. have been used.

The yearly survival curve for each series shows gradual improvement. In the early years, the curves are in close agreement. The first important rise for the Barnes curve occurs after 1937, which coincides with the first decrease in intensity of radium tubes. After 1945, there was another decrease in intensity of radium treatment, but that is not accompanied by any significant change in results during the particular years in study. The Barnard curve shows improvement after 1940, which coincides with a change in method of radium treatment at that institution. Both curves show a temporary fall during the war years.

The final comparison to be attempted here is upon the basis of clinical material. Distribution into the various stages of advance is shown for each four-year period. The Barnes material has become progressively more favorable, but quality of the Barnard material has remained almost constant. It may be that there has been tendency toward "over-classification" at Barnard due to infection, and it is possible that educational programs have been less effective among indigent classes of peoples.



PERIODIC SUMMARY OF CLINICAL STAGE (PER CENT)

	I	II	III	IV	I	II	III	IV	I	II	III	IV
BARNES	9	36	34	21	18	50	27	5	27^{+}	50	20	3
BARNARD	25	54	20	1	26	57	15	2	23	57	19	1

$^{+}$4 CASES IN SITU

Fig. 1. Curves showing yearly survival rate in Barnes and Barnard series. Intensity of radium sources used in treatment is shown for different periods. Distribution of patients into different stages of advance is shown for each four-year period in question.

No definite explanations can be made for differences in clinical results at the two institutions. It should be noted that a more favorable material has prevailed in the Barnes series. At that institution, there was an earlier attempt to modify treatment by use of lower intensity sources of radium. It is possible that nutritional and hygienic factors are important. Detrimental effects of infection are more marked in the Barnard patients. For some reason, it has not been possible to demonstrate in those patients a relationship between gross architecture and end results, which is so clearly shown in the Barnes series. For individual prognosis, one must evaluate stage of advance, gross architecture, infection, and method of treatment. To those should be added nutritional state. The patient to be treated surgically is adequately prepared before operation and supported during recovery. In most instances, nutrition is given little attention during irradiation. Adequate support for those patients should result in further advance in survival rates. It is not unsound to postulate that tumor control by irradiation is accomplished through effects upon normal tissues supporting the neoplasm, as well as by more direct effects upon the lesion. In that instance, the health of host tissues must be important.

In final analysis, we are still without detailed knowledge upon tumor regression and prognosis. The most reliable criterion for estimating curability is spread of tumor. That has been illustrated many times in survival statistics given in relation to proven lymph node involvement. Those data are available only in patients treated by radical hysterectomy, or in those subjected to iliac lymphadenectomy in conjunction with irradiation. The latter procedure has been performed in 176 patients, and radical hysterectomy in seventy-seven individuals. Most of those patients are in the Barnard series. Results from those procedures are given in Table VI. Among the 253 patients in whom lymph node data are available, there are fifty-five with positive nodes, and 198 with negative nodes. In the positive group, five-year survival is 10.9 per cent, but for the negative group, the value is 52 per cent. Thus, the presence or absence of lymphatic dispersion becomes the most significant factor in determining prognosis. Those data are not available in patients treated by irradiation alone.

TABLE VI. INCIDENCE OF POSITIVE NODES AND SURVIVAL RATE IN 253 PATIENTS TREATED ENTIRELY OR IN PART BY SURGERY

LYMPH NODE INVOLVEMENT

Iliac Lymphadenectomy 176 patients*
Radical Hysterectomy 77 patients*

International Classification
Stages I and II

Lymph Nodes	Number of Patients	Per Cent Total	Five-Year Survival Number	Five-Year Survival Percent
Positive	55	21.7	6	10.9
Negative	198	78.3	103	52
Total	253	100.0	109	42.8

*Various amounts of pre-operative irradiation were employed in a large percentage of patients.

The question as to whether or not lymph-node dispersion can be controlled by irradiation has been extensively discussed without final decision. Presumably, an occasional patient may gain control, but only infrequently will the quantity of radiation arriving at the lateral pelvis be adequate for that effect. It should be noted also that with surgical removal the survival is low among patients with proven node dispersions.

Control of lymphatic spread is the focal point of interest in the treatment of cervical cancer. The paracervical and parametrial injection of radioactive gold presents a new technique with that aim. At the Washington University, the work has been performed by Dr. Allen and Dr. Sherman. They have demonstrated that the material has the affinity to be picked up by lymphatics and transported to regional nodes with promptness that permits arrival at those points with considerable residual radioactivity. They have shown that metastases will not necessarily block passage of the material into involved nodes. They have demonstrated histologic alterations in lymph node cancer that appear microscopically identical to changes following more conventional forms of irradiation. More recently, in collaboration with Dr. Ter Pergossian, they have attempted to arrive at specification of dose comparable to expressions that can be given for x-rays and radium. It cannot be stated that lymph node dispersions are controlled by radioactive gold, but the clinical results noted so far are impressive. During the four years that gold has been employed in selected cases upon the ward service at Barnes Hospital, the current survival free of recognized recurrence in the total ward service is greater than upon the private service. In all previous experience, the private series has invariably shown the better result.

SURGICAL OPERATION IN IDIOPATHIC ULCERATIVE COLITIS

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ONE CANNOT long deal with such a disease as idiopathic ulcerative colitis without wondering whether it is not a group of diseases, at present classified as one. Several of the factors thought to be of etiological importance are usually recognizable in each individual case. The result seems to be that no one of us can provide a consistently or even occasionally specific curative therapy.

When we left Minnesota to go to New York three years ago, we reviewed those cases seen at the University of Minnesota Hospitals from 1934 to 1951 and at Minneapolis General Hospital from 1947 to 1951.[5] There were 267 such cases, most of which were either advanced or very acute. An evaluation of these was undertaken to learn if possible the fate of those handled primarily by conservative as opposed to those handled by operative means. Of that group treated without operation, or with operation deferred until undertaken as an acute emergency, only 29 per cent of those followed were alive in 1951, and only 27 per cent of these survivors were free of symptoms. Of that group treated with the aid of surgical intervention, 80 per cent of those followed were alive in 1951 and all but 9 per cent were active and well.

The factors responsible for death in these patients were varied. Surgical mortality was high early but very low later in the combined experience, which included that of W. T. Peyton, O. H. Wangensteen, J. R. Paine, and others.

Cancer as Cause of Late Death

The chief lethal factor, in addition to colitis proper and surgical misfortunes, was cancer development. As previously reported, this study indicates that those who retain colons with a ten-year history of colitis developed this complication in 10 per cent of the series. It has been the ill fortune of one of us to deal with sixteen such cases, and two more of the entire series have developed cancer of the colon since 1951. This is an experience in keeping with that of Dukes and Brooke[2] and of Cattell,[3] who found a cancer incidence above 30 per cent in cases of long duration.

Perhaps the most pathetic aspect of cancer in late colitis is that the symptoms which usually call attention to cancer of the colon, namely, alternating diarrhea and constipation, melena, weight loss, abdominal cramps, malaise, et cetera, are already familiar to the patient and of long standing. The warning symptoms are, therefore, rarely appreciated until the situation is far advanced. The diagnostic signs are similarly misleading, in particular the roentgen studies, for the fibrosis of late colitis can obscure completely the signs of carcinoma. Finally, the cancer which complicates colitis is commonly more malignant than that seen in the absence of colitic disease. Of the entire group of sixteen cancers in this category which we have seen and of the two in the study group to develop cancer since 1951, only one was alive at last report.

Types of Surgical Intervention

Vagotomy.—In 1947, Franklin Eddy and one of us undertook to evaluate the part which vagotomy might play in the management of ulcerative colitis, a maneuver suggested to us by C. J. Watson. Our initial results were most encouraging, and therefore this procedure was utilized in a series of forty-two patients who were carefully followed by an enlarged study group over several years. Careful study of the records suggests that the best responses occurred in those cases without appreciable fibrosis of the colon, without massive hemorrhage, and without extremely extensive ulceration.[4,6] The problems of gastric retention occurred in more than 10 per cent of these cases, and it seemed desirable to perform a parasympathetic block which might be removed when desired. The introduction of banthine by Grimson has accomplished this end, and only rarely is operative vagotomy employed now. It is essential apparently to give the drug every four hours day and night in dosage sufficient just slightly to impair ocular accommodation.

Ileostomy.—Ileostomy without simultaneous colectomy should be abandoned in the care of

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colitis. The thesis that the colon may heal sufficiently to permit closure of the ileostomy has been discredited, for fewer than ten patients so closed without trouble can be found in the literature. Furthermore, the mortality of ileostomy alone, except in meticulously selected cases, ranges upward from 10 per cent. Finally, the natural reluctance of patients in the series from the University of Minnesota to accept colectomy after effective ileostomy, and the early reluctance of the staff to advise it, were rewarded by a further 10 per cent loss of patients.[5]

Primary Colectomy.—Several years ago, Wangensteen and Toon[8] reported striking improvement in mortality figures pursuant to removal of the colon as well as ileostomy at the initial operative intervention. A few months later similar results were published by Miller and Ripstein.[7] Accumulation of the data of the University of Minnesota, Miller and Ripstein, and us, indicates the risk to be somewhere between 2 and 5 per cent for the larger operation, but a fraction of that of ileostomy alone. It appears that postoperative complications and fluid losses are vastly reduced by removal of this huge bed of infected, ulcerated bowel.

Characteristics of a Good Ileostomy.—There is no more distressing mishap for the colitic patient than the creation of a poor ileostomy. Cutaneous excoriation, ascending enteritis, a stenotic orifice, fistula formation, prolapse, retraction, and herniation are all nearly always outgrowths of poor surgical management, and these are precisely the developments which have led so many internists to defer operation until the last possible moment.

There are certain components to the construction of a good ileostomy which need repeated emphasis.

1. *Choice of site and incision.*—The ileostomy will of necessity be single barrelled if primary colectomy is performed. It should never be made in the main incision, for skin to skin closure tolerates the discharge poorly; the area in addition is usually depressed after a few months, making the use of an adhesive bag unsatisfactory. A circular incision an inch in diameter through all layers of the abdominal wall serves admirably.

The ileostomy is preferably placed in the beltline and close to the midline, well away from bony prominences and the crease of the groin. Loosening of the bag on exercise is thereby minimized.

2. *Use of healthy ileum.*—Approximately one-third of patients studied were found to have involvement of the small intestine to a greater or less degree. Recognition of such involvement upon gross inspection of either the serosal or the mucosal surface has proven less satisfactory than utilization of the rapid frozen section method. Use of ileum at the bottom end of normal bowel leads to a more secure ileostomy with minimal excoriation and with semi-formed stool.

3. *Attachment to abdominal wall.*—Secure attachment of the ileum and mesentery to the posterior rectus sheath and peritoneum is essential to prevention of herniation beside the ileum through the abdominal wall, without which prolapse does not occur. Use of fine silk (#0000) placed so as not to include mucosa and so as not to cause strangulation of tissue is apparently effective in avoidance of fistula formation. The authors have had no fistulas in association with use of such sutures.

4. *Formation of mesenteric diaphragm.*—Torsion of other segments of bowel about the loop attached to the anterior abdominal wall for ileostomy is a real risk, but one easily avoided by fashioning a diaphragm of the mesentery of resected terminal ileum or ascending colon to close off the space from terminal ileum to anterior, lateral, and posterior abdominal walls to the right of the ileostomy.

5. *Skin attachment.*—Attachment of the margin of the one inch circular incision to the bowel as it passes through not only hastens healing, but permits assurance of some slack ileum in the fatty layers. The latter measure forestalls the later funnelling of skin down to the muco-cutaneous junction, a complication which can make comfortable ileostomy care impossible and muco-cutaneous stenosis a virtual certainty.

6. *Coverage of projecting stoma.*—Brooke has suggested eversion of the projecting ileum, bringing the cut edge back to the circular skin margin, where it is sutured in place.[1] A well-covered ileostomy is thus fashioned at once, and a very soft and pliable stoma results. This method appears to surpass in simplicity and freedom from

complications either the use of split thickness skin grafts or mucosal grafts.

Conservative Proctectomy.—Rectal excision as for the Miles operation is commonly followed by sexual crippling through injury to the pelvic parasympathetic outflow. In colitis such radical resection leaves a huge hole which heals more slowly than when the resection is performed in the absence of colitis. For these reasons, those proctectomies performed for colitis (after proctoscopic evidence of freedom from cancer) are best accomplished by dissection immediately adjacent to the rectal wall. In the authors' series, there is no instance of impotency, and primary closure and smooth healing are usually possible.

Indications for Operation

Operative intervention should regularly consist of primary subtotal or pan-colectomy. The indications recommended are:

1. Fulminating colitis which fails to respond within days to good medical management.
2. Acute colitis which cannot be brought under control within a month of onset.
3. Colitis which advances the degree of damage and fibrosis to a stage in which malignant degeneration would be difficult to recognize.
4. Chronic colitis of more than three to five years' duration.
5. Colitis with complications of serious or uncontrollable nature:
    - (a.) Perforation
    - (b.) Carcinomatous degeneration, proved or suspected
    - (c.) Cirrhosis of the liver
    - (d.) Secondary arthritis
    - (e.) Secondary pyoderma

Prognosis After Colectomy

With primary colectomy, as indicated above, the initial risk is apparently lower than with other surgical maneuvers. In addition, the subsequent outlook is very gratifying. Of seventy-eight cases followed in Minnesota and ten in New York for periods up to ten years after dismissal from the hospital following colectomy and ileostomy, there have been three deaths from metastatic cancer of the colon and one of lymphatic leukemia. As of last record, all others are alive. Of those alive, all but seven are in good health. Several of the women have had children since operation, all without complications or mortality.

These figures are in striking contrast to the staggering mortality in the group not undergoing operation of any kind, in which over one-third had died within five years of first observation.

Conclusions

1. It is possible that many cases of idiopathic ulcerative colitis can be successfully handled on a long-term basis by non-surgical means.
2. Vagotomy appears usually to be helpful in carefully selected cases of colitis, but the use of banthine usually makes this operation unnecessary.
3. The risk of cancer development in these colons late in the disease is immensely greater than the risk of primary colectomy and ileostomy.
4. Satisfactory ileostomy results only with careful attention to precise details.

References

1. Brooke, B. N.: Ulcerative Colitis and Its Surgical Management. Baltimore: The Williams & Wilkins Co., 1954.
2. Brooke, B. N.: Personal communication.
3. Cattell, R. B.: Personal communication.
4. Dennis, C.; Eddy, F. D.; Frykman, Howard M.; McCarthy, A. M., and Westover, D.: The response to vagotomy in idiopathic ulcerative colitis and regional enteritis. Ann. Surg., 128:479, 1948.
5. Dennis, C., and Karlson, K. E.: Surgical measures as supplements to the management of idiopathic ulcerative colitis; cancer, cirrhosis, and arthritis as frequent complications. Surgery, 32:892, 1952.
6. Eddy, F. D.: Late results of vagotomy in the treatment of idiopathic ulcerative colitis and regional enteritis. Surgery, 29:11, 1951.
7. Miller, G.; Ripstein, C.B., and Tabah, E. J.: Surgical management of ulcerative colitis. Surg., Gynec., & Obst., 88:351, 1949.
8. Wangensteen, O. H., and Toon, R. W.: Primary resection of the colon and rectum with particular reference to cancer and ulcerative colitis. Am. J. Surg., 65:384, 1948.

LIVER BIOPSY: A NECROPSY STUDY

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BIOPSY of the liver as an aid in the diagnosis and study of hepatic disease has become an established procedure. Although Iversen and Roholm were not the first to utilize biopsy of the liver, it was their report in 1939 that gave the impetus to the present widespread use of this procedure. Since their report, a multitude of papers have attested to the value of this technique in the diagnosis and study of a wide variety of hepatic diseases.

At the time the present study was proposed, no detailed investigation had been reported concerning attempts to correlate histologic sections obtained at hepatic biopsy with sections of the liver made at necropsy. Consequently, it was decided to compare material obtained from the liver shortly after death by means of biopsy techniques with regular pathologic sections made at necropsy. Since this study was begun, a number of investigations were reported that were similar in some respects to this work. Waldstein and Szanto took ten specimens per liver from the livers of fifteen cadavers; four of these livers were normal, two showed toxic hepatitis and nine were affected with cirrhosis. They noted agreement in the various sections from each liver with regard to the amount of injury to hepatic cells and fatty changes. Dissimilarity appeared in 10 per cent of the 150 specimens with regard to alteration of lobular pattern, scarring in portal or central zones, inflammation of the peripheral zone and portal areas, and leukocytic reaction. Giesen and associates correlated material obtained at punch biopsy of the liver with that found at necropsy. They found the over-all correlation to be excellent in forty-five of forty-eight cases. Twenty-three of twenty-six focal lesions, all of which were metastatic malignant tumors, correlated perfectly. The correlation was perfect in all of twenty-two cases in which diffuse hepatic disease was present. Wagoner and co-workers compared results of study of ninety-seven specimens of hepatic tissue obtained by needle biopsy in eighty-six patients with findings at necropsy. Specimens were obtained prior to death in sixty-six patients and immediately postmortem in thirty-one instances. Antemortem specimens showed correlation in fifty-six of the sixty-six cases; postmortem specimens correlated in twenty-seven of the thirty-one cases. Of twenty-four livers in which no specific diagnostic changes were noted on biopsy, twenty-three showed a similar lack of changes at necropsy. In thirty-two cases of neoplastic disease, twelve biopsies failed to show the presence of tumor. Complete agreement between findings at biopsy and at necropsy was noted in twenty-one cases of cirrhosis, nine cases of obstructive jaundice, five cases of fatty vacuolization and two cases of acute yellow atrophy.

Christian, in 1952, did a "brief study to determine the effectiveness of needle biopsy in representing the histology of the entire organ." Material was obtained from five widely separate regions of the livers of ten deceased persons by means of biopsy technique; this material was assigned secret identification and was submitted to a pathologist for interpretation. General agreement was noted in these ten instances between findings of needle biopsy and those established by thorough examination of the entire liver.

Materials and Methods

By means of biopsy techniques, 282 specimens of liver were obtained from 207 cadavers prior to detailed necropsy. With the exception of stillborn infants, who were excluded, the 207 cadavers utilized in this study represented nearly consecutive deaths from all causes at the Mayo Clinic and the Rochester State Hospital.

Hepatic tissue was obtained from eighty-five bodies prior to embalming, whereas 122 bodies had been embalmed when the specimens were taken. The elapsed time after death varied from one to thirty-two hours, with an average of 3.18 hours.

The Vim-Silverman needle was used in all instances. The majority of specimens were taken via the transthoracic approach in the right an-

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Abridgment of thesis submitted by Dr. Brandenburg to the Faculty of the Graduate School of the University of Minnesota in partial fulfillment of the requirements for the degree of Master of Science in Medicine. Because of lack of space, the complete list of references used to document this work has not been used.

terior axillary line. The remainder, especially when the liver was known to be enlarged, were obtained by use of the right subcostal approach. When the initial attempt resulted in failure to obtain tissue, second and, occasionally, third attempts were made. When failures resulted from the subcostal approach, the transthoracic route was utilized. Since 1950, Stauffer[6] has used a modification of the original Vim-Silverman needle, which has decreased failures to obtain tissue in living patients to 1 or 2 per cent.

The core of tissue obtained from the liver was placed immediately in 10 per cent solution of formalin. Specimens varied from 2 mm to 2.5 cm in length and were 2 mm in diameter. After fixation in the formalin all specimens were cut longitudinally and stained with hematoxylin and eosin. In addition, selected specimens were stained with van Gieson's stain for connective tissue, with Gömöri's reticulum stain, by the Prussian-blue method for iron, and with cresyl violet for amyloid.

These sections obtained by means of biopsy techniques were then compared with the regular pathologic sections made at necropsy; at the latter time, routine sections were taken from the right and left lobes and additional sections were made as indicated by the gross findings. On the basis of microscopic examination of the tissue obtained by needle and prior to examination of the regular necropsy sections, a definitive pathologic diagnosis was made whenever possible. In addition a brief description of the histologic findings was made. If no diagnosis was possible, only the description was recorded.

On the basis of this comparison, the sections obtained by needle were graded as correct, suggestive or false. These sections were considered "correct" if they compared favorably with the regular necropsy sections and if a correct pathologic diagnosis could be made. Sections were considered "suggestive" if they suggested the correct diagnosis but if some details necessary in making this diagnosis were lacking; this might mean simply that the tissue procured by needle did not quite reflect the over-all status of the hepatic parenchyma. Sections were considered "false" if they did not furnish sufficient data for diagnosis.

The sections obtained by biopsy techniques were also classified as "adequate" or "inadequate." Sections were adequate if the amount of tissue sufficed for the purpose of diagnosis and if fragmentation and distortion were minimal or absent. Inadequate sections were those in which mechanical defects were moderate or marked. As might be expected, considerable correlation was present between the adequacy of the section obtained by needle and its diagnostic value.

TABLE I. RESULTS OF HISTOLOGIC EXAMINATION OF TISSUE REMOVED AFTER DEATH BY NEEDLE FROM 207 LIVERS

Condition	Total Cases	Diagnostic			Technical	
		Correct	Suggestive	False	Adequate	Inadequate
Normal liver	72	63	9	0	62	10
Chronic passive congestion	36	31	4*	1	34	2
Metastatic carcinoma	17	5	0	12	11	6
Fatty liver	15	14	0	1	14	1
Cirrhosis†	12	7	4‡	1	8	4
Leukemia	12	8	1	3	8	4
Focal granulomas	10	2	0	8	6	4
Hepatitis	7	3	2§	2	4	3
Obstructive jaundice	6	5	1	0	5	1
Cholangitis	5	1	4	0	1	4
Hemangioma	4	0	0	4	3	1
Amyloid	2	1	0	1	1	1
Hodgkin's disease	1	1	0	0	1	0
Miscellaneous	8	7	0	1	7	1
Totals	207	148	25	34	165	42

*Of these, three were adequate and one was inadequate.
†Includes two cases with hepatoma, two with hemochromatosis and one with biliary cirrhosis.
‡Of these, three were inadequate and one was adequate.
§Of these, one was adequate and one was inadequate.

Results

The diagnostic and technical gradings of the sections obtained by needle from the 207 livers are presented in the table. When more than one specimen was obtained in any instance only the better or best specimen was utilized. Examples of the histologic features encountered are illustrated in Figures 1 and 2.

Comment

Results in the livers affected by focal hepatic disease, such as metastatic carcinoma, hemangiomas and granulomas, require some comment. In the seventeen instances of metastatic carcinoma, the needle specimen was negative in twelve and diagnostic in five. This poor diagnostic percentage can be explained in part by the fact that only one to five metastatic nodules were found in the liver in five cases and hepatic metastasis was not suspected in any of these cases at the time of death. In the other seven instances in which the diagnosis was missed, there was moderate to extensive spread of malignant tissue that was simply missed by the biopsy needle. In a large

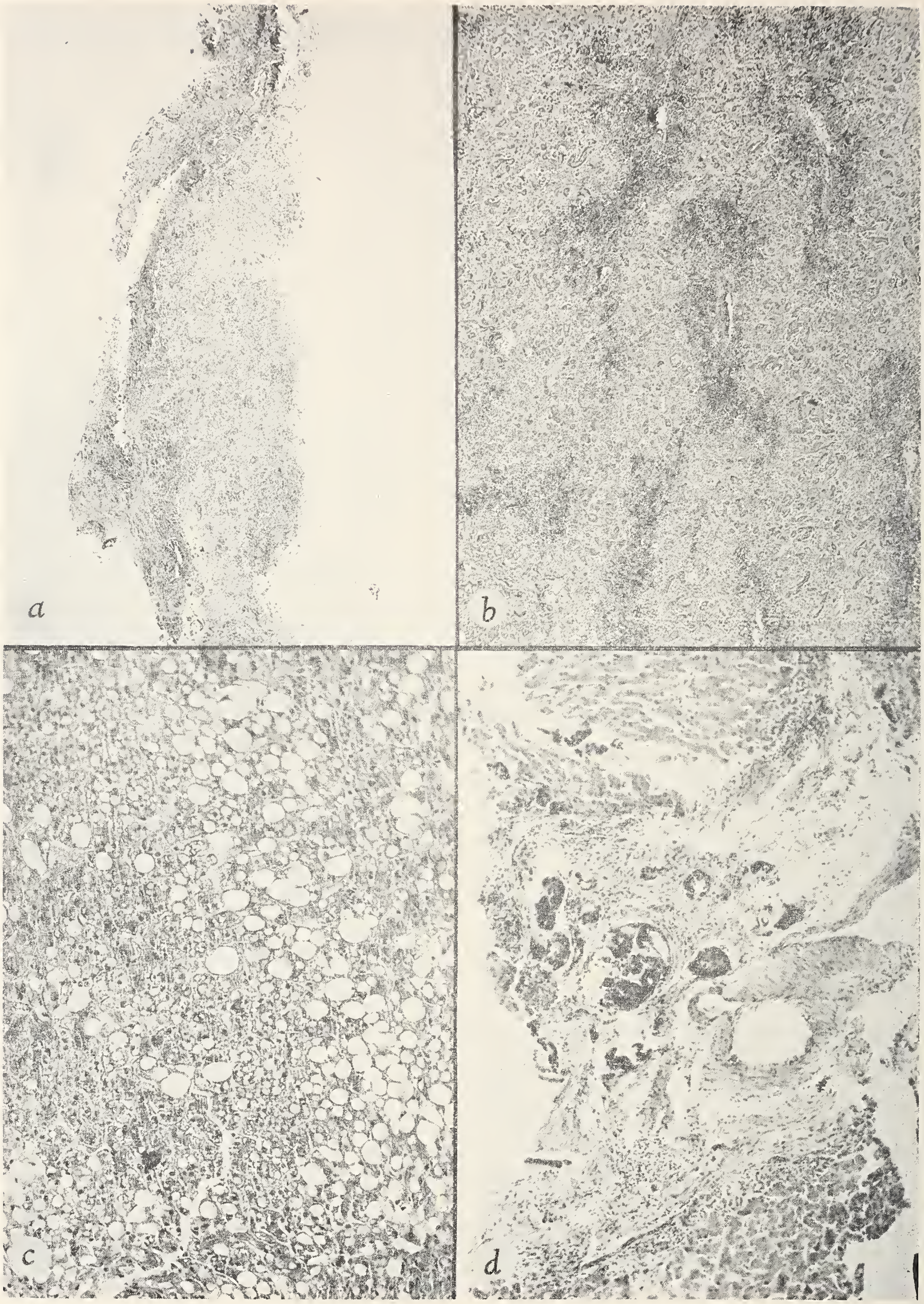

Fig. 1*a*. Needle specimen showing acute hepatitis with extensive cellular infiltration. *b*. Regular necropsy specimen in same case, showing extensive areas of hemorrhage in addition to the findings in *a*. These hemorrhagic areas were extensive throughout the entire parenchyma, but were not apparent in sections obtained by means of biopsy technique (both hematoxylin and eosin; x29). *c*. Extensive fatty infiltration in a forty-three-year-old man, an alcoholic, who died of lobar pneumonia (hematoxylin and eosin; x105). *d*. Metastatic carcinoma of the liver. The primary lesion was in the rectum (hematoxylin and eosin; x65).

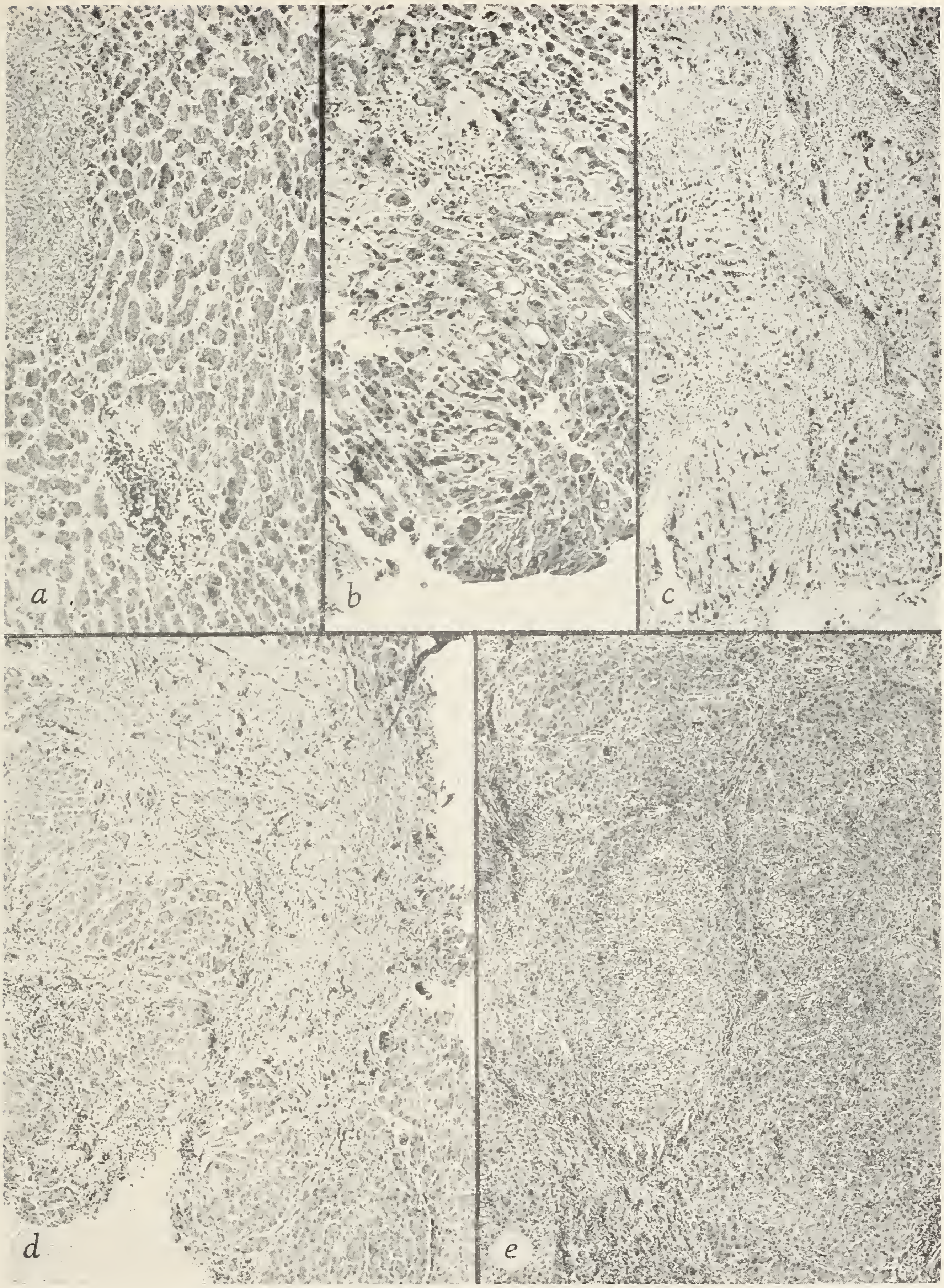

Fig. 2*a*. Acute leukemia and miliary tuberculosis. A granuloma is present at the edge of the section; note periportal collection of lymphocytes (hematoxylin and eosin; x105). *b*. Amyloidosis of the liver. Note pale, homogeneous areas of amyloid almost entirely replacing parenchymal cells in some parts. There was similar involvement of the spleen, adrenals, lymph nodes and kidney (hematoxylin and eosin; x100). *c*. Extensive deposits of iron in the parenchymal cells characteristic of hemochromatosis. There are associated fibrosis and regenerative nodules (hematoxylin and eosin; x65). *d*. Regenerative nodules, fibrosis and regeneration of bile ducts characteristic of cirrhosis. The broad band of fibrosis and lack of fat are more suggestive of postnecrotic cirrhosis than of Laennec's cirrhosis. *e*. Routine section at necropsy from same liver showing rather uniform small regenerative nodules, thin bands of fibrosis and fatty changes characteristic of Laennec's cirrhosis (both hematoxylin and eosin; x65).

series of needle biopsies, Stauffer[5] has obtained positive results in malignant neoplasms in approximately 70 per cent of the cases. This degree of accuracy has been attained in cases in which the liver was large in most instances and by taking multiple specimens from one or two sites, with the use of frozen sections until evidence of malignancy was found.

In the four instances of single hemangiomas of the liver, the lesion was missed in all.

The lesion was missed in eight of ten livers affected by focal tuberculous granulomas or granulomas of unknown cause. In two of these eight cases, only two and five small white nodules respectively were present in the entire parenchyma. In the remainder, however, the granulomas were diffusely spread throughout the hepatic parenchyma. In only one instance was the granulomatous hepatic lesion a manifestation of active tuberculosis. On examination of the microscopic sections obtained at necropsy it was noted that in most instances the granulomas were distributed sparsely enough in the hepatic parenchyma to make a "miss" at needle biopsy appear likely. In Figure 2*a* it can be noted that the specimen obtained by needle caught only a portion of a single granulomatous lesion. This would suggest that perhaps repetitive biopsy should be done to increase the percentage of positive results when granulomatous processes are suspected.

Since the selection of cadavers for this study represented nearly consecutive deaths from all causes, it would be expected that there would be a relatively large number of normal livers and livers showing only chronic passive congestion associated with cardiac disease. This is borne out since thirty-six specimens showed chronic passive congestion and seventy-two specimens were normal.

Results of study of needle specimens in the presence of diffuse hepatic disease were consistently good when the specimen was adequate. The majority of inadequate specimens were obtained in the early part of the study. As experience was gained, the incidence of poor specimens became progressively less and consequently the incidence of correct diagnoses improved. In the presence of diffuse hepatic disease it was impossible to make the correct diagnosis in only five instances in which good technical sections were obtained; these five cases included one of cirrhosis, one of hepatitis and three of chronic passive congestion.

While hepatic biopsy in diffuse hepatic disease has been shown to have great value, various shortcomings of the procedure have been reported. Womack noted that the study of lobular arrangement and relationships in such specimens was unsatisfactory. Volwiler and Jones stated that in coarsely nodular "toxic cirrhosis" a specimen procured by needle biopsy may fail to include scar tissue although the specimen is apparently sufficient in amount. Ricketts and associates reported on a patient in whom biopsy of the liver showed normal findings. At splenectomy they found extensive cirrhosis with irregular nodulation of regenerated liver separated by wide zones of scarring. The biopsy needle had entered areas of scarring separated by regions of apparently normal hepatic tissue. Stauffer[6] has noticed that nodular regeneration not found in specimens obtained by needle is often evident in larger wedge-shaped specimens obtained surgically, especially in primary biliary cirrhosis.

Results of this study would verify the opinion of some investigators who have noted that in some instances hepatic biopsy does not reflect adequately the general state of the liver in diffuse hepatic disease. Figure 1*a* and *b* is an example in that tissue obtained by needle did not show the rather pronounced hemorrhagic aspect of the hepatitis noted in regular sections. In sections from cirrhotic livers, also, it was noted that the over-all architecture of the parenchyma was not accurately portrayed in all instances. This was especially apparent in one instance of cirrhosis following homologous serum hepatitis; the needle specimen showed clear-cut evidence of cirrhosis but in these sections the individual hepatic cells appeared relatively normal. Routine sections at necropsy showed pronounced destruction of the lobules in many regions, with early regenerative changes at the periphery; these sections indicated a much severer process than was indicated by the needle specimen. It is of interest that hepatic cells often appear normal in a regenerative nodule in any type of cirrhosis.[6] In another instance the specimen obtained by needle was characteristic of postnecrotic cirrhosis; however, gross examination of the liver and study of the routine sections at necropsy showed the condition to be Laennec's cirrhosis (Fig. 2*d* and *e*).

Despite these limitations it was still possible to make a definite diagnosis in most of these cases, and this ordinarily is the most important fact to be gained from biopsy. Variations in the degree of injury to hepatic cells and connective-tissue changes in hepatitic and cirrhotic processes in various parts of the liver have been known for a long time, and it is apparent that tissue procured by needle can reflect only a small part of this process. Fortunately, in almost all instances, the degree of variation of the microscopic change in various portions of the liver in diffuse disease is not great enough to prevent accurate diagnosis at biopsy.

Summary and Conclusions

Specimens from the liver were obtained by use of the Vim-Silverman needle in 207 cases shortly after death. Results of study of such tissue were compared with results of examination of routine sections at necropsy from each lobe of the liver plus special sections through diseased portions.

From the standpoint of diagnostic accuracy the specimens obtained by means of biopsy techniques were correct or suggestive in 173 of the 207 cases. This represents an accuracy of approximately 83 per cent. In thirty-four cases, results of study of needle specimens were false; of these, twenty-one failures were for technical reasons. In this study on cadavers, continued experience with the Vim-Silverman needle increased the percentage of adequate specimens obtained by needle.

The highest degree of correlation was found in cases of fatty livers, normal livers, chronic passive congestive and obstructive jaundice. The percentage of missed diagnoses in metatastic carcinoma was high when compared to that found in clinical experience. This was due in part to focal lesions clinically unsuspected in some cases and in part to failure to repeat the biopsy. The biopsy needle missed involved tissue in seven of twelve cases in which the metastatic lesions were extensive.

Focal lesions, such as malignant lymphoma or tuberculosis, were rarely diagnosed by examination of tissue obtained by needle in this study. This suggests that multiple biopsies might be considered when such lesions are suspected. The correlation was high in diffuse hepatic diseases, with the exception of those of a granulomatous nature.

References

1. Christian, E. R.: An evaluation of needle biopsy of the liver. Am. J. Med., 13:689-703 (Dec.) 1952.
2. Giesen, C. P., Koepsell, J. E., Hastings, E. V., and Lindert, M. C. F.: Correlation of punch liver biopsy with autopsy material. Am. J. Digest. Dis., 18:304-307 (Oct.) 1951.
3. Iversen, Paol and Roholm, Kaj: On aspiration biopsy of the liver, with remarks on its diagnostic significance. Acta med. Scandinav., 102:1-16, 1939.
4. Ricketts, W. E., Kirsner, J. B., Palmer, W. L., and Sterling, Kenneth: Observations on the diagnostic value of liver biopsy, tests of hepatic function, and electrophoretic fractionation of serum proteins in asymptomatic portal cirrhosis. J. Lab. & Clin Med., 35:403-407 (Mar.) 1950.
5. Stauffer, M. H.: Needle biopsy of the liver. Thesis, Graduate School, University of Minnesota, 1950.
6. Stauffer, M. H.: Personal communication to the author.
7. Volwiler, Wade and Jones, C. M.: The diagnostic and therapeutic value of liver biopsies: With particular reference to trocar biopsy. New England J. Med., 237:651-656 (Oct. 30) 1947.
8. Wagoner, G. P., Ulevitch, Herman, Abernathy, E. L., Gall, E. A. and Schiff, Leon: Correlation of the results of needle biopsy of the liver with autopsy findings. J. Lab. & Clin. Med., 36:1000-1001 (Dec.) 1950.
9. Waldstein, S. S., and Szanto, P. B.: Accuracy of sampling by needle biopsy in diffuse liver disease. Arch. Path., 50:326-328 (Sept.) 1950.
10. Womack, N. A.: Biopsy of the liver. North Carolina M. J., 8:300-306 (May) 1947.

UNTOWARD EFFECTS OF CORTISONE, HYDROCORTISONE AND CORTICOTROPIN ON THE GASTROINTESTINAL TRACT

(Continued from Page 628)

27. Sauer, W. G., Dearing, W. H. and Wollaeger, E. E.: Serious untoward gastrointestinal manifestations possibly related to administration of cortisone and corticotropin. Proc. Staff Meet., Mayo Clin., 28:641-649 (Nov. 18) 1953.
28. Sloan, Sol, Briggs, J. D. and Halsted, J. A.: ACTH therapy for ulcerative colitis complicated by perforation of coexisting peptic ulcer. Gastroenterology, 18:438-442 (July) 1951.
29. Smyth, G. A.: Activation of peptic ulcer during pituitary adrenocorticotropic hormone therapy: report of three cases. J.A.M.A., 145:474-477 (Feb. 17) 1951.
30. Texter, E. C., Jr., Legerton, C. W., Jr., Ruffin, J. M., Boyd, J. A., Jr. and Smith, A. G.: Idiopathic ulcerative colitis: giant ulceration following corticotropin (ACTH) and cortisone therapy. A.M.A. Arch. Int. Med., 91:744-757 (June) 1953.
31. Tulin, Maurice, Kern, Fred, Jr. and Almy, T. P.: Perforation of bowel during treatment of ulcerative colitis with corticotropin. J.A.M.A., 150:559-562 (Oct. 11) 1952.

THE USE OF HEMODIALYSIS IN THE TREATMENT OF BARBITURATE INTOXICATION

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WHILE the depressant effect of large quantities of barbiturates on the central nervous system is well known, the exact manner in which it is produced remains obscure. There is general agreement that cerebral activity is depressed by interference with cellular oxidation primarily through the inhibition of cellular enzymatic systems.[3,10] Whatever the mechanism of this action, the presence of coma creates additional barriers to the successful treatment. The maintenance of adequate nutrition and skin hygiene and the prevention of pulmonary and urinary tract infections are problems which become proportionately more difficult with the prolongation of the coma. The rapid termination of coma is, therefore, necessary to the successful treatment of this condition.

The current aim of the treatment of barbiturate intoxication has been directed towards the stimulation of depressed cerebral activity. This has been primarily affected by the widespread employment of analeptic drugs and supplemented by general supportive measures. That this approach has not always been successful has been emphasized by Reed, Driggs, and Foote,[11] Mousel and Essex,[9] Schmidt,[12] and Mousel.[8]

The urinary excretion of barbiturates is quite slow although there is some variation in the rate of excretion depending upon the drug involved.[2] Only two of the current therapeutic measures are aimed at the elimination of the offending drug from the body. Of these, gastric lavage is widely used, extremely important, and most beneficial when employed immediately after ingestion of the drug. Massive intravenous infusions have been employed in an effort to promote diuresis and elimination of the drug. In view of the slow urinary excretion of the barbiturates, which may be further aggravated by coma and shock, a vicious circle results which can be interrupted by the removal of the drug as rapidly as possible.

The artificial kidney has provided an effective and rapid means of eliminating barbiturate from the body and successful treatment of two cases of barbiturate coma by hemodialysis has been reported by Kyle and associates.[5] The rate of barbiturate excretion by this method as determined by these authors was twenty-four to thirty times greater than that of the patient's own renal system.

The purpose of this communication is to report a case of barbiturate coma which did not respond to analeptics but in which consciousness was restored within twelve hours by means of artificial kidney dialysis.

Case Report

During a period of despondency, a twenty-seven-year-old housewife ingested approximately twenty one-quarter grain tablets of phenobarbital and fifty-three quarter grain capsules of nembutal. Four hours later she was found deeply comatose and was admitted to a local hospital where attempts at arousal were made with picrotoxin and electrical stimulation. Gastric lavage was not attempted. The patient remained deeply comatose and in addition developed an atelectasis as a result of the aspiration of pharyngeal secretions. This necessitated a tracheotomy. The patient was transferred to the University of Minnesota Hospitals approximately twenty-four hours after the ingestion of the drug.

Upon admission to this hospital, the patient was deeply comatose and responded minimally to painful cutaneous stimulation. The ciliospinal reflex was absent, the pupils were miotic but equal, and did not respond to light. The deep tendon reflexes were absent and no pathological toe signs were obtainable. A tracheotomy tube was present and functioning well. Respiratory movements were extremely shallow and rapid with a lag in evidence on the high side and moist râles in the right lung field. The blood pressure was 90/65, pulse 125 per minute and the body temperature was 99.6° F.

An admission urinalysis showed traces of albumin, sugar, occasional red blood corpuscles, leukocytes and casts. The blood urea nitrogen was 5 mgm per 100 ml, the hemoglobin was 15.2 grams and the leukocyte count within normal limits. The blood sugar, carbon dioxide combining power, and chlorides were normal. Chest x-ray showed a segmental patchy atelectasis of the right lung with a shift of the mediastinum to the right. Barbiturate was present in the urine. Initially the patient was given 1100 milligrams of metrazole which did not affect consciousness and which produced only transient weak knee and ankle tendon reflexes. Eighteen milligrams

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of picrotoxin were given with no appreciable change in the comatose state.

In view of the negative response to analeptics and the presence of atelectasis, prolonged coma, and mild shock, artificial kidney dialysis was instigated in the hope of eliminating sufficient barbiturate to arouse the patient. The blood barbiturate level, as determined by the ultraviolet spectro-photometric method was 2.65 milligrams per cent. The patient was given heparin prior to dialysis.

The apparatus employed was the artificial kidney designed by Kolff and modified by Merrill.[4,5,6] Dialysis was carried out for a period of six hours during which time the comatose condition gradually lessened. Three hours after the onset of dialysis the deep tendon reflexes were present in the lower extremities. Towards the latter part of the procedure the patient became quite restless, yawned frequently, and moved her arms and legs at will. The blood pressure which at the start of dialysis was 95/70 rose to 114/70 and the respiratory movements deepened. Two hours after the termination of dialysis the patient responded to verbal commands but was confused and disoriented. The gross confusion gradually lessened and disappeared during the following twelve hours. Eighteen hours after the start of dialysis, she was oriented and sitting up in bed. The tracheotomy tube was plugged and removed on the following day. At this time the patient was transferred to the psychiatric service since she was emotionally unstable and exhibited inappropriate behavior. Within a few more days the patient had recovered from all noticeable effects of the intoxication. She did continue to show a neurotic type of personality disturbance which, as far as could be assessed, was essentially the same as it had been before the suicide attempt. Neither clinical nor psychometric evidence of cerebral deficit could be found. The psychiatric aspects will be the subject of another communication.

The blood barbiturate level was 1.58 milligrams per cent at the termination of dialysis. The dialysis bath was changed during the procedure and the barbiturate content of the first bath was 162 milligrams. That of the second was 55 milligrams, making a total of 217 milligrams of barbiturate removed during the period of hemodialysis. The blood chlorides, CO2 combining power, serum sodium and potassium were normal at the end of dialysis.

Discussion

While the distribution of the barbiturates in the tissues is poorly understood, the liver and kidney are known to exert a profound effect upon the metabolism of these drugs. In the liver the barbiturates are destroyed by side chain oxidations and the degradation products appear in the urine. Those barbiturates which escape hepatic detoxification are eliminated in the urine. The renal excretion of barbiturates is very slow, especially of those that are dependent upon the kidneys for their elimination, such as dial, alurate and barbital. In the normal human being, traces of barbital are detectable in the urine nine days after the ingestion of a single therapeutic dose. Of a given dose of barbiturate, only 8 per cent is eliminated in the first twelve hours, 20 per cent in the next twenty-four hours and 35 to 65 per cent in forty-eight hours.[2]. The slow rate of excretion may be due to a slow release from tissue depots, delayed hepatic detoxification, or to reabsorption by renal tubules. The latter has been reported by Giotti and Maynert.[1] Impaired renal or hepatic function will further decrease barbiturate excretion. Barbital has a tendency to cause oliguria as large doses have an antidiuretic effect. Hypotension of any cause further reduces the excretion of barbiturates. Oliguria and hypotension are commonly seen in coma. Irrespective of the mechanism, the slow urinary excretion of the barbiturates is the basis of the cumulative effect, and in part accounts for the prolongation of coma. It is therefore apparent that the barbiturate should be eliminated as rapidly as possible. Gastric lavage is an effective means of removing recently ingested drug but is of little benefit when used twelve or more hours after ingestion.

The artifical kidney, by providing a large filtration area, effectively eliminates barbiturates from the body. Kyle and associates[5] demonstrated that human beings without renal disease excrete 4 per cent of an ingested dose of phenobarbital in two and one half hours, whereas it is possible to remove 66 per cent of the phenobarbital in the same period of time by hemodialysis. These authors reported two cases of barbiturate coma. In one of their patients 740 milligrams of barbiturate were removed in five hours by hemodialysis whereas only thirty milligrams had been excreted by the patient during the five hours preceding the dialysis. This patient was restored to consciousness in eight hours after having exhibited no improvement during the previous thirty-six hours. It is apparent from such cases and the one presented here that consciousness can be restored in a very short time by means of hemodialysis. The termination of coma over such a short period of time has distinct advantages to patient, physician and nursing personnel.

The indications for dialysis in barbiturate coma are as follows: (1) prolonged coma, (2) pulmonary distress, (3) unresponsiveness to analeptics, (4) oliguria, hypotension, (5) concurrent renal and hepatic disease.

Summary

A case of near fatal barbiturate coma treated successfully with hemodialysis is presented. The indications for the use of this procedure are listed with the primary aim directed towards the removal of ingested drug in order to shorten the period of coma.

References

1. Giotti, A., and Maynert, E. W.: The renal clearance of barbitural and the mechanism of its reabsorption. J. Pharm. Exper. Therap., 101:296, 1951.
2. Goodman, L., and Gilman, A.: The Pharmacological Basis of Therapeutics. The Macmillan Company, New York, 1941.
3. Himwich, H. E.: Brain Metabolism and Cerebral Disorders. The Williams & Williams Co., 327, 1951.
4. Ibid: The use of an artificial kidney; I. Technique. J. Clin. Investig., 29:412, 1950.
5. Kyle, L. H., Jeghers, H. Walsh, W. P., Doolan, P. D., Wishinsky, H., and Pallotta, A.: The application of hemodialysis to the treatment of barbiturate poisoning. J. Clin. Invest., 32:364, 1953.
6. Merrill, J. P.: The artificial kidney. New Eng. J. Med., 17:246, Jan. 3, 1952.
7. Merrill, J. P., Thorn, G. W., Walter C., Callahan, G. J., and Smith, S. H., Jr.: Use of artificial kidney; II. Clinical experience. J. Clin. Investig., 29:425, 1950.
8. Mousel, L. H.: Cerebral edema and its relation to barbituric acid poisoning. J.A.M.A., 153:459, 1953.
9. Mousel, L. H., and Essex, H. E.: An experimental study of the effects of respiratory stimulants in animals under pentothal sodium anaesthesia. Anaesthesiology, 2:272, 1941.
10. Quastel, J. H.: Respiration in the central nervous system. Physiol. Rev., 19:135, 1939.
11. Reed, C. E., Driggs, F. M., and Foote, C. C.: Acute barbiturate intoxication: A study of 300 cases based on a physiologic system of classification of the severity of the intoxication. Am. Int. Med., 37:290, 1952.
12. Schmidt, C. F.: Recent developments in respiratory physiology related to anaesthesia. Anaesthesiology, 6:113, 1945.

TREATMENT OF ADRENAL INSUFFICIENCY WITH CORTISONE AND HYDROCORTISONE

(Continued from Page 625)

maximal stimulation also. Measurement of steroid excretion is moderately time-consuming and exacting and is not generally available, so reliance on eosinophil response is usually a practical necessity. Very occasionally, patients with adrenal insufficiency react with fever and varying degrees of collapse to intravenously-administered ACTH. This danger is decreased somewhat by using a saline vehicle.

Summary

1. Cortisone and hydrocortisone have greatly improved the treatment of adrenal cortical insufficiency of both the primary and the secondary types.

2. Corticotropin (ACTH) has proved to be a very useful diagnostic tool in testing adrenal cortical responsiveness.

3. Problems in the use of these compounds involve chiefly adjustment of dosage under varying conditions of stress. Occasionally untoward reactions occur.

Bibliography

1. Fourman, P. and Horler, A. R.: Cortisone in treatment of chronic adrenal insufficiency. Lancet, 1:334-336 (Feb. 13) 1954.
2. Kelly, V. C., Ely, R. S., and Raile, R. B.: Hormone patterns in patients with congenital adrenal hyperplasia. Pediatrics, 12:541, 1953.
3. Knowlton, A. I.: The modern treatment of Addison's disease. M. Clin. North America, pp. 721-737 (May) 1952.
4. Randall, H. T.: An evaluation of adrenalectomy in man: Physiological changes and the effect on advanced neoplastic disease. Bull. New York Acad. Med., 30:278-302, 1954.
5. Sorkin, S. Z. and Soffer, L. J.: Maintenance management of Addison's disease with injections of "long-acting" microcrystalline esters of desoxycorticosterone. Metabolism, 2:404-410, —.
6. Thorn, G. W., Forsham, P. H., et al: Advances in diagnosis and treatment of adrenal insufficiency. Am. J. Med., 10:595-611, 1951.
7. Thorn, G. W., Jenkins, D., et al: Pharmacological aspects of adrenocortical steroids and ACTH in man. New England J. Med., 248:632-634, 1953.

Seminar

CHRONIC ULCERATIVE COLITIS

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"One swallow never makes a summer"[31]

We all pass a loose stool now and then. Down South these can be caused by all sorts of slimy things, but up here they are usually due to something we ate or drank the night before. The large epidemics of bacillary and amoebic dysentery have waned but we still have with us a few people who, for no known cause, develop a persistent purge. Webster's dictionary[175] defines normal as "according to or not deviating from an established norm," while disease is "any departure from the state of health producing marked symptoms." The present discussion is concerned with those patients whose frequent departures are caused by ulcerative colitis.

Age and Sex Incidence

"The roaring twenties"

For some unknown reason, the symptoms most often begin between the ages of twenty and thirty, but they may start at any age. Of 871 cases at the Mayo Clinic,[10] ninety-five were in children—three of these in the newborn. Banks and Klayman[9] reported a series of 200 cases in twenty of which the onset of symptoms came after the age of fifty.

Ulcerative colitis is probably as common in men as in women.[24,156,184] The only difference sex seems to make in these days of the joint income tax return is that the female is still the one who becomes pregnant. Opinions on the effect of pregnancy in ulcerative colitis range from: "Ulcerative colitis in pregnancy is extremely virulent and may be fatal"[130] to "adverse"[91,92,168] to "the effect of pregnancy is generally salutory"[53] with no authors undecided though the gentle touch of Chi Square could have softened these hard conclusions.

Symptomatology

"O wearisome condition of humanity"[69]

The history of a patient's illness is what we make it. We usually think of the symptoms of ulcerative colitis in terms of diarrhea. In the careful questioning of thirty-two patients in their first attack of ulcerative colitis, Engel[51] found that in 22 cases, the earliest symptom was bleeding and in ten of these patients, this was accompanied by constipation. Diarrhea was the presenting symptom in only 12 cases. Bleeding was also a prominent early symptom in recurrent attacks.

The number of stools may vary greatly during an attack. A sudden exacerbation can change a person with normal bowel movements into a bed patient constantly passing blood and pus.[185]

Proctoscopic Diagnosis

"The footprints upside down"

When you see a person with diarrhea, look in his rectum. That is where the diagnosis of ulcerative colitis is made, and that is the logical place to look rather than to start right off taking colon x-rays.[125] In at least one-third of the cases, the x-rays won't show anything anyway.[88]

We do not know the cause of this disease, so we can't make much use of the laboratory in diagnosis. The symptoms are too variable and can occur in other conditions so they don't help a lot and all we really have to go on is what the bowel looks like. Still, our batting average with a proctoscope is very good. The number of times you use vague terms like "proctitis" or "colitis" to describe nonspecific ulcerative colitis will depend upon how well you know the appearance of ulcerative colitis.

In the normal bowel, the mucosa is thin and transparent and you can see the blood vessels underneath. The normal velvety appearance comes from the presence of the innumerable microscopic crypts of Lieberkühn.

We seldom see the early transient stages of ulcerative colitis when the mucosa just looks red, and only the fact that it bleeds easily makes us suspect the diagnosis. Miliary abscesses may appear as tiny yellow spots which leave bleeding bases when wiped with a cotton swab.[26]

A more familiar appearance is that seen in the typical acute case. The valves of Houston look wider than normal because the mucosa gets edematous. This thick, red mucosa hides the blood vessels. The bowel lining is covered by a shaggy white scum of sloughed cells and pus.

Presented at Medical Grand Rounds, Veterans Administration Hospital, Minneapolis, Dr. E. B. Flink, Chief of Medical Service

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When you wipe it or touch it with the tip of the proctoscope, it oozes blood.

When the acute stage subsides, the mucous membrane begins to look pale, granular and glazed. The little miliary abscesses heal leaving tiny pock-like scars which Buie[26] calls "the footprints of previous activity." It is in the chronic stages that Nesselrod's[125] "three R's" appear—the valves of Houston become roughened, rounded and retracted. This, plus the narrowing and the loss of rugal folds in the rectosigmoid, gives the bowel a tubular appearance.

After the first acute attack of ulcerative colitis subsides, all stages of the disease may be seen in the intervals between exacerbations. Buie states that, "the numerous scattered pin-head ulcers and friability representing the active disease, associated with pock-like scars giving evidence of former activity, form a picture which is to me pathognomonic."

One may not see the large ulcers higher in the bowel, because it is often dangerous to run the proctoscope up very far. A good way to tell these ulcers from those of bacillary dysentery, tuberculosis or amoebiasis is that in ulcerative colitis all of the mucosa between the ulcers looks abnormal while this is seldom true in the other diseases that cause ulcerations.[125] In other words, discrete ulcerations favor bacillary dysentery, tuberculosis or amoebiasis. Of course, you should get stool cultures, aspirations for amoebae and a chest x-ray, but remember that there is no reason why a person can't have two diseases.

One diagnosis to keep in mind is a radiation proctitis following treatment of a carcinoma of the cervix—although it is hard to confuse the large anterior wall ulcer with ulcerative colitis.

In some parts of the country and depending somewhat on the recreational habits of the patient, a Frei test may be in order to rule out the proctitis of lymphogranuloma venereum.

The most important single thing to keep in mind when doing proctoscopy on a patient with ulcerative colitis is that these people have very tender rectums. Because it is a chronic recurrent disease, they often go to several doctors. If you ever want to see the patient again, be gentle with your rectal finger and try not to use air when you proctoscope.

In summary, ulcerative colitis remains one condition where the laboratory still hasn't replaced the physician who knows what the disease looks like.

X-Ray Findings

"Out, damned spot"[154]

The x-ray diagnosis of ulcerative colitis is often unreliable. It may be easy to diagnose when the entire colon is small, smooth and straight like a lead pipe, but you have to remember that the normal descending colon looks rather small and smooth.

The most common finding in a case of ulcerative colitis is tiny serrations—sometimes along the entire colon but more commonly about the sigmoid or rectum. However, retained fecal material can mimic this appearance exactly.

Always get an evacuation film. The barium-filled colon can look perfectly normal while an abnormal mucosal pattern may show up very well on the evacuation film.

If you think your patient has ulcerative colitis, do not order an air contrast barium enema. The distention may obliterate much of the mucosal pattern making diagnosis more difficult. There is also a very real danger of perforation. Results with the thick barium (Ude) technique (which is so useful in demonstrating polyps) are even less satisfactory.

Pathology and Differential Diagnosis

"The morphology of (ulcerative colitis and regional enteritis) is as different as syphilis is from gonorrhea."[174]

Some physicians have hazy ideas on the differences between regional enteritis and ulcerative colitis—particularly when regional enteritis involves the colon[104]—although the two diseases are very different pathologically.[2,18,174]

A complete study of these differences was recently made by Warren and Sommers.[174] They reviewed 300 surgical specimens of ulcerative colitis and regional enteritis. In about one-third of their cases of ulcerative colitis the terminal ileum[99] was also involved while in one-sixth of the cases of regional enteritis the colon was involved. They state that the two diseases rarely co-exist.

In regional enteritis the bowel resembles a "section of garden hose"[174] or a "boiled angleworm"[41] because of its sharp demarcation, thickening and grey to red-purple color. The thick-walled and narrow-lumened bowel produces the well-known "string sign." The mesentery is thick, stiff and corrugated and contains enlarged lymph nodes. Fistulae, adhesions, and abscesses are common. There are skip areas in less than ten per cent of the cases.[174]

On the other hand, the intestine in ulcerative colitis usually looks grossly normal from the outside although there may be some redness and spasticity found at time of surgery.

Warren and Sommers[174] microscopic description of the two diseases is as follows:

> "Microscopically, regional enteritis is a progressive, granulomatous lymphangitis. . . . It affects the lymphatics in the intestinal submucosal, muscular and subserosal layers . . . producing what is essentially an elephantiasis. . . . In its earliest stages proliferation and desquamation are noted among the endothelial lining cells of the lacteals and lymphatics, producing cellular

> masses that obstruct lymph flow. Then the plugged vessel is further altered peripherally by a proliferation of mesenchymal cells and an infiltration by lymphoid and monocytic leukocytes with formation of a tuberculoid granuloma. These tubercles may contain giant cells, usually of foreign body type. . . . However, caseous necrosis and acid fast bacilli are not seen. In the intestinal wall, (these changes) . . . lead to variable mucosal ulcerations . . . (through) interference with blood supply. . . . Gross ulceration is not a uniform characteristic of regional enteritis. The intestinal mucosa as a rule appears reddened, swollen and cobbled. However, once formed, ulcers . . . tend to persist with formation of fistulous tracts or intramural abscesses. . . . Perforation with peritonitis is unusual. . . . The (same process) is also found in the mesenteric lymphatics and lymph node sinusoids . . . . with tuberculoid granulomas in one-half to three-fourths of the cases.
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> "Microscopically . . . the damage in ulcerative colitis is surprisingly superficial—being mainly restricted to the colonic submucosa and mucosa. . . . In about half the cases studied it has not been possible to find indications of the type of lesion originally responsible for the ulceration . . . they show only shallow mucosal ulcers surrounded by acute and chronic inflammation, regeneration of mucous membrane, fibrosis and fatty infiltration of submucosa, and hypertrophy of colonic muscular tissue. . . . The inflammatory process is exudative and reparative, without any granulomatous tendencies. . . . In another 40 per cent of all nonspecific ulcerative colitis cases in which the evolution of the disease can be traced, an unusual primary localization of exudative inflammation is observed. The lesions begin with a collection of polymorphonuclear leukocytes in the bases of some of the colonic mucosal glands . . . (these) abscesses occur together and their ruptures allow pus to dissect along and separate portions of the mucosa . . . with resultant sloughing and ulcer formation. . . . When ulcerative colitis involves the ileum, there is an associated lymphangitis, as occurs with inflammations elsewhere, but no granulomas are formed."

In the remaining 10 per cent of their cases the disease was more severe. These patients had larger and deeper ulcers, and inflammatory necrosis of blood vessels with infarctions of the wall of the colon leading to perforation and peritonitis. The lesions resembled those seen in periarteritis and thromboangiitis obliterans.

Grossly, the involvement in ulcerative colitis tends to be diffuse and without sharp demarcation. The most striking change involves the mucosa with longitudinal and cross-connecting ulcers producing a network. The mucosa between the ulcerations is bright red or purple and may overhang the ulcers. Elongated tags of mucosa may hang free—these are called pseudopolyps and are not true adenomas—they are simply islands of mucosa remaining between ulcerations.

Because these two diseases are so distinct pathologically, it is logical to believe that in many cases, they can be separated clinically and roentgenologically. This has been true in our experience.

For instance, fistula-in-ano has been described as a lesion suggestive of regional enteritis.[41] Three of four patients seen at this hospital with a fistula-in-ano and a clinical diagnosis of ulcerative colitis had typical pathologic findings of regional enteritis on examination of the resected specimens. Two additional patients with fistula formation noted on barium enema also had a typical pathological picture of regional enteritis involving the colon. One patient with a "string sign" of the colon on x-ray also had regional enteritis.

The description of regional enteritis as a segmental disease and of ulcerative colitis as a diffuse, poorly demarcated process leads naturally to the question: Where do the cases of so-called "segmental colitis" and "right-sided colitis" fit in? Are they ulcerative colitis or are they regional enteritis?

The failure to attempt to separate these two diseases has led to some strange mixups: In 1950, 2,000 cases of ulcerative colitis were reviewed from the Mayo Clinic.[156] In 128 of these cases there was segmental involvement, i.e. "regional colitis." In 1954, 600 cases of regional enteritis were reported from the same institution.[172] No cases of "regional colitis" were included in the regional enteritis series although the authors referred to a "personal communication" from Newman, also of the Mayo Clinic, who had collected 201 cases of "regional colitis." In general they felt that these cases should be classed as regional enteritis. It seems probable that the reason why the cases of regional colitis were not included in the regional enteritis series was that they had already found a home in the reviews of ulcerative colitis.

In a recent article, Crohn[42] stated that in 8 per cent of his 300 office patients with ulcerative colitis, the disease was right sided. He stated that the bowel was usually thickened, but he did not discuss the pathologic findings in any detail.

Other reports of "segmental colitis" also fail to give detailed pathologic findings.[75,178] One case report of regional colitis confined to the sigmoid[60] described the involvement of a 12 cm. area by a large inflammatory mass with dense adhesions to the bladder. The mucosa was edematous and hyperemic and slightly polypoid in areas but no ulcerations were present. The wall of the colon measured 8 cm. in the narrowed portion. Gross descriptions of such lesions seem to resemble regional enteritis more than ulcerative colitis, but most writers do not include a detailed microscopic description. At present, the identity of "regional colitis" is not firmly established, but the few cases reported resemble regional enteritis grossly.

If the cases of segmental colitis are really regional enteritis, then localization of lesions in ulcerative colitis is simplified—it is a disease which invariably affects the distal colon and at times extends to involve the remainder of the colon and terminal ileum.

I could find no proctoscopic descriptions of the appearance of the lower bowel when the entire colon is involved by regional enteritis. In three such cases seen at this hospital, proctoscopy showed:

(1) "A thick walled friable mucosa, the examiner's finger could be inserted through a fistula into a rectal abscess."
(2) "Marked injection and small irregular ulcerations with a stricture at 8 cm."
(3) "Patchy areas of inflammation in the recto-sigmoid." The proctologist stated: "This is not the picture of ulcerative colitis."

Complications of Ulcerative Colitis

"Whatsoever was the father of a disease, an ill diet was the mother."[137]

"Polyposis" is often said to be the most common local complication of ulcerative colitis.[12] That is undoubtedly true if you count islands of mucosa, i.e. pseudopolyps—but, the incidence of true polyps (those with an overgrowth of glandular epithelium) has not been shown to be increased.[173]

There is, however, a real increase in the incidence of carcinoma of the colon particularly in young patients who have had their symptoms for over ten years.[37,84,105,128,171.177,179] Carcinoma complicating ulcerative colitis is typically multicentric, invasive, and rapidly fatal. Of seventy-three cases of carcinoma reported by Shands et al[155] there were only two five-year survivors and one of these died of another carcinoma of the colon two years later. The incidence of carcinoma of the colon has been estimated to be from three to 30 times more frequent in ulcerative colitis patients than in the general population.[16,176] This incidence is not great enough to justify routine prophylactic colectomy but it does represent an additional reason for removing the colon in a young patient with severe, continuing, long-standing disease. It is probably foolish to do routine barium enemas (at, say, six-month intervals) in hopes of finding an early carcinoma. The infiltrating growth would be difficult to identify and the spread is too rapid to make periodic barium enema examinations for carcinoma practical.

Patients with ulcerative colitis often have hepatic,[79,169] pancreatic[8] and renal[78] lesions. Varying degrees of fatty infiltration of the liver are reported in over half of the cases studied by needle biopsy or at autopsy.[14,79,85,93,133] There may be an increased incidence of cirrhosis[74,144,169] but this depends somewhat on the definition of cirrhosis used. Findings in the pancreas[8] are those of interstitial inflammation, fibrosis and acinar dilatation. The renal lesions are those of tubular degeneration and necrosis.[78] Do these findings mean the ulcerative colitis is a systemic disease or could the changes be related to protein deficiency? Similar hepatic, pancreatic, and renal lesions are described in South African infants on a protein deficient diet—a disease called kwashiorkor, where fatty infiltration of the liver, fibrosis of the pancreas, and renal tubular degeneration has been described.[45]

Careful nitrogen balance studies[150] have shown that patients with active ulcerative colitis are in negative nitrogen balance. The nitrogen loss was not well correlated with the number of bowel movements. (Possibly this explains the failure of Ball, Baggenstoss and Bargen[8] to correlate the pancreatic lesions with the severity of symptoms). Normal post-operative patients on intravenous feedings lose nitrogen with frequent enemas.[138] A high protein feeding will restore the positive nitrogen balance.[150,187]

Skin lesions, notably pyoderma gangrenosum [36,106,151] are thought to be secondary to the malnutrition in ulcerative colitis.

Acute arthritis (with regression following surgery) is a rare complication.[114,180]

There may be a slightly increased incidence of peptic ulcer in persons with ulcerative colitis, but the association is relatively uncommon and the reported cases may be on the basis of chance. [32,72,181] Massive dilatation of the colon can rarely occur and when it does, it is often followed by perforation.[111.112,117]

Etiology

"Science is a sacred cow."[160]

For many years it was thought that Bargen had discovered the cause of ulcerative colitis. He reported the isolation of a diplostreptococcus from 80 per cent of 1,000 patients with ulcerative colitis.[12] In a way, he fulfilled Koch's postulates. He injected suspensions of these organisms intravenously in dogs and produced hemorrhagic lesions in the gut. The organisms recovered produced "the disease" in other dogs. Agglutinin tests using the serum of patients with ulcerative colitis were positive. However, later studies with the diplostreptococcus[44] revealed that those organisms are often present in the normal colon. These organisms have a tendency to clump by themselves so agglutination tests are not reliable. The production of lesions by the intravenous injections of these clumped organisms is probably a result of the method and not proof of the etiology of ulcerative colitis.

Most reviews of ulcerative colitis[23,116] refer to a paper by Felsen[52] suggesting that ulcerative colitis may follow bacillary dysentery. Felsen, a laboratory director, and Gorenberg, a resident in obstetrics, secured records on 122 of 210 persons who had been hospitalized nine to twelve months previously with a diagnosis of bacillary dysentery. Of these, 37.7 per cent had persistent or recurrent symptoms and the authors implied that these were due to ulcerative colitis or regional enteritis although an "x-ray diagnosis" was made in only six of the patients and lesions (not described) were found on proctoscopy in only two. Even the occurrence of two cases of ulcerative colitis seems like a high incidence but Felsen and Gorenberg followed only hospitalized patients—not the mass of those with mild symptoms. If "bowel complaints" are not accepted as proof of ulcerative colitis, then their experience agrees with that of Brown and Bargen[25] who found only one instance of ulcerative colitis in a sixteen-year follow-up of 100 cases of bacillary dysentery. The presence of agglutinins against Salmonella or Shigella is not proof of infection since cross reactions with strains of coliform organisms have been demonstrated.[17] Perhaps the best argument against bacillary dysentery producing ulcerative colitis is that these infections are epidemic and tend to occur in many members of the same family while ulcerative colitis is a relatively rare disease which seems to occur at random.

There is little doubt that strong emotions can produce changes in the color and secretions of the colon.[63,64] It has never been shown that the repeated production of these changes can cause ulcerative colitis.[28] Although a great deal has been written about the psychiatric treatment of ulcerative colitis, there has never been a well-controlled psychiatric study of its etiology. The amount of work required in testing and interviews of patients and controls in such a study would be prohibitive.

Rowe[146] believes that allergy may be the cause of ulcerative colitis for such reasons as "ulcerations may arise from localized vascular allergy as do cancker sores in the mouth" and "the tendency to polyp formation as in the nasal mucosa suggests allergy as a cause." Andresen[3] is even more definite in his conclusions and states that "food allergy is the most important etiologic factor in ulcerative colitis" and "milk offends in over 80 per cent of the cases." He recommends a milk free diet. He also feels that peptic ulcers of the stomach and duodenum are probably allergic in origin[4] but has not suggested a milk free diet for peptic ulcer.

The role of the autonomic nervous system in this disease is about as clear as the anatomy of the sphenoid bone. Lium[102] in 1939 described a method of transplanting colonic mucosa to the abdomen of dogs. He noted that the application of acetylcholine to the surface of the explants would produce ulcerations if prostigmine were given intravenously to inactivate cholinesterase and intensify the effect of acetylcholine. The rationale of this experiment was that ulcerative colitis might be caused by excessive muscular contractions as a result of an overactive parasympathetic nervous system. This hypothesis was made more attractive by the fact that the author had seen two colons removed from patients with ulcerative colitis in which the longitudinal and circular ulcers seemed to parallel the muscle fibers of the colon. It was a good idea, but Warren and Sommers were unable to find a relationship between the position of the ulcers and the locations of the longitudinal and transverse muscle bands in their study of 180 cases.[173] Another correlation that failed was the demonstration in ulcerative colitis of a decreased serum cholinesterase—which has since been shown to be non-specific and likely related to the level of serum albumin.[120]

Numerous cholinergic drugs have been studied as possible etiologic agents in ulcerative colitis. Repeated injections of mecholyl in beeswax have produced ulcerations in the intestinal tract of dogs.[182] However these changes are transient[126] and not suitable for studying therapy.

Measurements of intracolonic pressures in dogs and man after vagotomy and lumbar sympathectomy have shown no change but section of the pelvic nerves in dogs reduced the pressures.[153] It is not possible to section the pelvic parasympathetic nerves in man without disturbing bladder function.

Because mucus is the chief secretion of the colon, the search began for some intestinal secretion which would lyse mucus and thus theoretically allow the normal bacteria of the colon to produce ulcers. It is hard to make any work with mucus sound scientific because anything that is done to mucus chemically will change its physical properties.[27] Therefore with little work on the effect of enzymes on mucus, efforts were directed at their other properties. Lysozyme is an enzyme (present in such places as gastric juice, tears and raw egg whites) which started one of our greatest five year wild goose hunts. Perhaps it was its beautiful name or the fact that it was easy to titre scientifically because it attacks the capsule of micrococcus lysodeicticus but at any rate, lysozyme titers were used as an index of activity[115] or as a method of checking the success or failure of various treatment regimes. The titres were invariably high in cases of active ulcerative colitis. Later it was shown that lysozyme would not dissolve mucus[109,127] and that the lysozyme was present only in the whole or lysed leukocytes.[86] Dogs with ulcers of the colonic mucosa produced by electrocautery passed large numbers of white cells and hence showed high lysozyme titres.[73] We gradually came back to something we already knew: patients with ulcerative colitis pass a lot of pus.

The tryptic digestion of the bowel by excess pancreatic enzymes was briefly suggested[43] but not proved in dog experiments.[98] The best evidence against this theory is that ulcerative colitis has been reported in patients with pancreatic insufficiency.

There has been little study of the mucus produced by mucus membranes—in contrast to the work on the mucus of connective tissue which contains hyaluronic acid. Hyaluronidase has no action on the glandular mucins. Recently, Burnet[27] has shown that Vibrio cholerae organisms produce two distinct mucinases. Sammons[147,148,149] has demonstrated a mucinase in the stools of patients with ulcerative colitis and in the ileostomy discharge, and Stoughton[161,162] has shown that filtrates of stools from ulcerative colitis patients will lyse formalin-fixed skin and intestine. At the present time this work is too incomplete to allow conclusions.

An interesting case of anaphylactic shock following a procaine penicillin injection was reported by Whipple.[183] The patient developed an ulcerative colitis and ulcerative cystitis. Pathologically, the lesions were of the "periarteritis type" seen in 10 per cent of Warren's cases.

The Course of Ulcerative Colitis

"This long disease, my life"[134]

General Andrew Jackson first developed severe diarrhea during the Creek Indian Wars. This was quite a handicap, even for an alert Indian fighter. At the battle of New Orleans, he was having up to twenty bloody stools per day. In spite of this—or perhaps because of it—he was successful in defending the American trenches against a vastly superior British Army. He suffered from recurring attacks of diarrhea until his death at the age of seventy-eight.[77]

The bulk of the literature on ulcerative colitis deals with the hospitalized and, by inference, more severe cases. We have no idea of the number or prognosis of the milder cases because reports from the fighting front, if the proctologists' office can be called that, are rare.

In 1948, Miller and Machella[119] reviewed 3,639 medically treated cases of ulcerative colitis followed from one to ten years. Of that number, only 8 per cent obtained relief for an extended period while 46 per cent were unimproved and the remaining 41 per cent showed some signs of improvement. Most of them remained more or less incapacitated.

Other follow-up studies[80,89,140,152] are in general agreement and may be summarized as follows: ulcerative colitis is a chronic recurrent disease. Attacks vary in duration and severity. If patients are followed long enough, almost all of them will have recurrences. The majority of medically treated patients continue to live restricted lives interrupted by frequent hospitalizations. You often see a patient who has been "cured" a dozen times.

It would help a great deal in planning treatment of an individual case if we knew whether or not persons with severe symptoms at the onset continue to have more trouble than those with mild symptoms. We may suspect that they do because follow-up studies have shown that the hospitalized cases usually remain ill; and while we do not know exactly what happens to the mild cases which are frequent in an office practice[186] they must not get very sick or they would be included in the hospital series. On the other hand, Bargen[13] has frequently stated "Usually when the very sick patients recover, they remain well." He has not presented statistical proof of this statement. Such proof would be desirable because when a very ill patient is carried too long on medical management (with the hope of remaining well thereafter) the surgical mortality may be increased.

Medical Treatment of Ulcerative Colitis

"Again I am beginning to live a little and feel less like a sick oyster at low tide"[1]

Ross[143] recently reviewed 100 publications (selected at random from the recent medical literature) dealing with the therapeutic use of new drugs. He found that 65 per cent of the authors used either inadequate controls or no controls. In preparing this paper, I read eighty-five articles dealing specifically with the treatment of ulcerative colitis. I was unable to find a single controlled study in spite of the fact that the disease is characterized by unpredictable remissions. The papers on ulcerative colitis report from 40 to 90 per cent "good results" using everything from raw hog's stomach to the wonder drugs. By "good results" the authors usually mean that the patient underwent a remission during the period of observation.

Machella[109] has recently reviewed the literature and prepared the following table of the results of various types of therapy:

	Cases	*Per Cent Good Results*
Sulfonamides	1275	57.7%
Antibiotics	167	61.6%
Hog Stomach	104	52-81 0%
Anti Lysozyme ...	121	65.9%
Cortisone	25	44.0%
ACTH	117	67.5%

For some strange reason, the longer a drug is on the market the poorer are the results in treatment. It is a favorable sign that the initial reports on ACTH and cortisone[68,87,110,145] have not shown quite as good results as those papers on raw hog intestines[57] or aluminum.[124] This does not necessarily mean that the steroids are any less effective but the authors are getting more cautious. The most recent publications on the use of steroids have covered the side effects and their variable effects on the suppression of acute symptoms.[48,49,67,83,107,131,167,188]

Both the narrow and broad spectrum antibiotics were tried[19,163] but apparently lost potency.[108,113]

Levine,[100] in 1951, proposed a new concept of the pathogenesis of ulcerative colitis. He had noticed a "virtual absence of the basement membrane" in specimens taken for biopsy from patients with ulcerative colitis and postulated that it might be a collagen disease. This paper prompted Sommers, Anderson and Warren[158] to make an intensive study of the basement membranes in ulcerative colitis. These authors found that the changes in the basement membrane were restricted to the areas of cellulitis and concluded that the basement membrane discontinuities were a secondary phenomonon following leukocytic infiltration.

However, the idea that ulcerative colitis was a connective tissue disease caught on and Svartz[165,166] prepared a mixture of aspirin and azo dye and sulfapyridine which she believed had an affinity for connective tissue. This drug was produced by her in collaboration with the Company Pharmacopeia in Sweden and marketed under the name of Azulfadine. She treated 387 patients with ulcerative colitis with this drug and of these, twenty-one patients had to discontinue treatment because of drug reactions—a tendency to leukopenia developed in five patients but "disappeared after liver injections." She initially reported 90 per cent "good results" and later 84 per cent good results. She stated, "In summary, I should like to stress the importance of treating ulcerative colitis with drugs having affinity for connective tissue since the main changes are localized to the submucous connective tissue."

Morrison[71,122] reported 58 per cent of his patients improved on Azulfadine and felt that this was superior to the results in his "control group" of patients treated in preceding years. Halstead[71] feels that the "results have not been striking," but Bargen[15] states that, "Azulfadine has been a tremendous help" although neither author has reported a significant number of cases.

For the sake of completeness, a word should be said about what happened to the "medical ileostomy" which consisted of tube drainage of the terminal ileum and non-residue feedings of amigen and dextri-maltose. The authors[118] later found that their results were just as good when the same feedings were used without the tube.

The literature on the treatment of ulcerative colitis contains so many articles that are controversial that it was a pleasure to review the writings on psychotherapy in this disease. All of the psychiatrists seem to agree that the "psychololgical disorder is fundamental"[157] and "the most important factor"[66] while the surgeons are in complete agreement that the "psychiatric aspect is by no means a major part of the problem."[96] A summary of articles on psychotherapy in this disease[34,50,65,70,81,101,135,136] might be: first, that probing is dangerous and unrewarding, and second, "a good doctor" invariably gets the best results.

Blood transfusions—even small amounts of blood given to patients who are not anemic are at times said to result in striking improvement.[13] Why should this be? It can't be the proteins because plasma only contains about 6 grams per 100 cc. It doesn't seem likely that it could be the red cells because they only break down at a rate of about 1 per cent per day. Either blood contains some as yet undiscovered substance which makes it a "good tonic" or else clinical impression is wrong again.

Kirsner and Palmer[86] state that in the diet of a patient with ulcerative colitis, fruits and fruit juices should be avoided while eggs and warm milk should be used for the acutely ill patient. Bockus,[121] on the other hand, says that citrus fruits and fruit juices are of great value while in many patients milk and eggs are not well tolerated. Although statements like these may confuse the physician, they make it easier for the patient—if he doesn't like his menu, he can simply get a new doctor.

The thing that determines whether or not a food is listed as "low residue" is its cellulose content.[33] The present use of cellulose compounds in the treatment of diarrhea in infants raises doubts about the rationale behind the almost universal use of the low residue diet in the treatment of ulcerative colitis.

Iconoclasts have attacked the use of absolute bed rest in such diseases as tuberculosis and hepatitis. Patients with ulcerative colitis also run a fever on the first few days after admission and this seems to subside with bed rest.

Surgical Treatment of Ulcerative Colitis

"Once . . . the diagnosis of ulcerative colitis is made, the problem is a surgical one"[153]*—vs.—"The vast majority of patients who have ulcerative colitis represent essentially medical problems."*[13]

"I want to emphasize my opinion that in every case where the patient has been shown to have ulcerative colitis . . . the gastroenterologist and the surgeon should meet and discuss treatment"[80]

In the first quarter of this century, when such things as abdominal ptosis and intestinal stasis were acceptable diagnoses, an English surgeon, Sir Arbuthnot Lane, whom Kerr[82] refers to as a "misguided enthusiast" began doing short circuiting operations and later colectomies not only for chronic constipation but also for mastitis, tuberculosis, rheumatoid arthritis, gallstones, gastric ulcer and goitre. This was the humble beginning of our present surgical treatment for ulcerative colitis.

For a more complete review of the history and techniques of surgery in ulcerative colitis, the reader is referred to the bibliography.† The best results have been reported with primary ileostomy and one-stage colectomy—removing the rectum at the same operation.[21,39,61,71,82,97,129] Even seriously ill patients seem to tolerate this better than a simple ileostomy probably because the colon, which Crile[40] states has the physiologic effect of an extensive third degree burn covered with a fecal poultice, has been removed. Ileosigmoidostomies have been almost uniformly unsuccessful. Vagotomy[46,47] has not been thoroughly evaluated but the results to date are equivocal.

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†See especially references numbered 5, 6, 20, 21, 22, 29, 35, 38, 39, 54, 55, 56, 58, 59, 61, 62, 71, 76, 90, 94, 95, 97, 129, 141, 142, 170.

Although a high mortality rate accompanied surgery a few years ago, recent reports[46,95] indicate that the mortality in good hands should be less than 4 per cent.

In contrast to the late results of medical treatment, most surgically treated patients do well.* The newer ileostomy bags have done much to minimize the discomfort of the ileostomy life. Sexual function is not disturbed if care is taken to keep close to the colonic wall in doing the abdominal-perineal operation.

However, we should remember that we do not see those milder cases who are seen in physicians' offices and large private clinics where the percentage of patients operated upon should be and is much lower.[13] The indications for surgery are difficult to define. The following direct quotations from the recent literature serve to emphasize the wide variety of existing opinions:

> "Rarely should surgical therapy be carried out for the patient who has so-called intractable disease." Bargen.[15]
>
> "Almost all patients can avoid operation but how many of them can do so and continue to carry on their normal activities?" Crile.[39]
>
> "Operation is necessary when it is considered unlikely that the patient will return to a normal or useful life." Brooke.[20]
>
> "The surgeons' position should be limited to the treatment of complications of the disease." Hutchinson.[76]
>
> "Should surgery be done on all patients with ulcerative colitis?" Lahey.[96]

And in the words of the "misguided enthusiast":

> "Many surgeons who regard the removal of a useless colon unjustifiable, knock the bottom out of a useful stomach without hesitation," (Arbuthnot Lane).[21]

A patient who undergoes a gastrectomy for peptic ulcer is often cured and is everlastingly grateful while a patient who has a colectomy for ulcerative colitis is left with a permanent disability. Perhaps it is minimal—they get married, have babies, go swimming and only change their bag once or twice a day. But, "a lifetime is a long time for second thought"[21] and the patient must be well aware of the nature and prognosis of his disease before surgery is performed.

At this hospital, a follow-up[123] of thirty-eight patients treated medically showed that twenty of them were living restricted lives requiring frequent hospital admissions whereas of twenty-five surgically treated patients, only two were doing poorly—one patient had been frequently readmitted for revision of his ileostomy and the other unfortunately had an ileosigmoidostomy and has refused abdominoperineal resection. It should also be pointed out that the surgically treated patients had all been previously treated medically with a poor response.

The initial high mortality of attacks of ulcerative colitis[140] has dropped. In recent years we have had no deaths at this hospital from medical treatment alone. Why has the mortality decreased? Rice-Oxley and Truelove were unable to show any improvement in prognosis with the use of antibiotics.[140] It seems likely that the increased awareness of the role of the potassium ion (the degree of potassium deficiency seems to be related to the fluid volume of the stools),[103] more physiologic electrolyte replacement, and the efforts directed towards correcting the plasma protein deficiencies[159] have all contributed to the declining death rate.

Our surgical mortality rate has been less than 5 per cent—perhaps this is a reflection of our tendency to operate before the patient becomes moribund. At one clinic, where surgery is reserved largely for the severe complications, the surgical mortality rate over the past ten years has been in the range of 25 per cent.[11]

The final answer on the indications for surgery is not apparent. The final decision should be an individual matter, arrived at by the co-operation of the internist and surgeon in the management of this difficult disease.

Summary

Certain aspects of the diagnosis, etiology, pathology and treatment of chronic ulcerative colitis have been discussed.

The advice of Plutarch[132] 2,000 years ago, still applies to the medical management of this disease:

> "Like a skillful physician, who in a complicated and chronic disease, as he sees occasion, at one while allows his patient the moderate use of such things as please him, at another while gives him keen pains and drugs to work the cure."

Bibliography

1. Alcott, L. M.: Life, Letters, and Journals, Chapt. 7, Thoreau's Flute.
2. Anderson, W. A. D.: Pathology. Chap. 26 in The Gastrointestinal Tract: Schenken, J. R., Burns, E. L., Chronic Ulcerative Colitis, p. 785. St. Louis: C. V. Mosby Co., 1953.
3. Andresen, A. F. R.: The ulcerative colitis problem. New York State J. Med., 49:1783-1790, 1949.
4. Andresen, A. F. R.: In Peptic Ulcer, by Sandweiss, D. J.. Philadelphia: W. B. Saunders Co., 1951.
5. Aycett, S. O.: Conservative surgery in the treatment of ulcerative colitis. Brit. M.J., 2:1348-1351, 1953.
6. Bacon, H. E., Trimpi, H. D.: The selection of an operative procedure for patients with medically intractable ulcerative colitis. Surg., Gynec., & Obst., 91:409-420, 1950.

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*See references listed in footnote, page 659.

7. Bacon, H. E., Trimpi, H. D.: Surgical management of ulcerative colitis. Brit. M.J., 153:1249-1253, 1953.
8. Ball, W. P., Bagenstoss, A. H., Bargen, J. A.: Pancreatic lesions associated with chronic ulcerative colitis. Arch. Path., 50:347-358, 1950.
9. Banks, B. M., Klayman, M. I.: Idiopathic ulcerative colitis beginning after the age of fifty. New England J. Med., 249:91-96, 1953.
10. Beranbaum, S. L., Waldron, R. J.: Chronic ulcerative colitis, case report in a newborn infant. Pediatrics, 9:773-778, 1952.
11. Bargen, J. A.: Personal communication.
12. Bargen, J. A.: Chronic ulcerative colitis. The Oxford Medicine Series, 3:197-213, New York Oxford Press, 1948.
13. Bargen, J. A.: The medical treatment of ulcerative colitis. Postgrad. Med., 10:317-321, 1951.
14. Bargen, J. A.: Disease of the liver associated with ulcerative colitis. Ann. Int. Med., 39:285-288, 1953.
15. Bargen, J. A.: Present trends in the management of ulcerative colitis and Regional enteritis. Gastroenterology, 23:673-674, 1953.
16. Bargen, J. A., Sauer, W. G., Sloan, W. P.: The development of carcinoma in chronic ulcerative colitis. Gastroenterology, 26:32-37, 1954.
17. Barksdale, W. L., Ghoda, A., Okabe, K.: Copro-agglutinins in ulcerative colitis. J. Infect. Dis., 89:47-51, 1951.
18. Bell, E. T.: A Textbook of Pathology. Philadelphia: Lea and Febiger, 1952.
19. Bercovitz, Z. T.: Sulfhydryl therapy in chronic ulcerative colitis. Ann. West. Med. & Surg., 5:546-548, 1951.
20. Brooke, B. N.: The surgery of ulcerative colitis. Ann. Royal Coll. Surg. (England) 8:440-456, 1951.
21. Brooke, B. N.: Total colectomy in one stage for ulcerative colitis. Lancet, 1:1196-1198, 1951.
22. Brooke, B. N.: The development of surgery for ulcerative colitis. Ann. Royal Coll. Surgeons, England, Lond., 12:246-258, 1953.
23. Brooke, B. N.: What is ulcerative colitis? Lancet, 1:1220-1225, 1953.
24. Brown, M. L., Kasich, A. M., Weingarten, B.: Complications of chronic ulcerative colitis. Am. J. Dig. Dis., 18:52-54, 1951.
25. Brown, P. W., Bargen, J. A.: Bacillary dysentery, late results and relationship to chronic ulcerative colitis. Am. J. Dig. Dis., 5:562, 1938.
26. Buie, L. A.: Chronic ulcerative colitis. J.A.M.A., 87:1271-1274, 1926.
27. Burnet, F. M.: The mucinase of V. cholerae. Australian J. Exp. Biol. & Med., 26:71-80, 1948.
28. Card, W. I.: Ulcerative colitis. Practitioner, Lond., 169:493-498, 1952.
29. Cattell, R. B.: Surgical aspects of chronic ulcerative colitis. Am. J. Surg., 86:613-616, 1953.
30. Cave, H. W., Winslow, S.: Earlier and more complete surgery in ulcerative colitis. Pennsylvania M. J., 55:905-909, 1952.
31. Cervantes, M. de: Don Quixote. Translated by Motteaux, P. A.
32. Chaiken, B. H., Levy, B. L., Wirts, C. W.: Coexistence of peptic ulcer and idiopathic ulcerative colitis. Gastroenterology, 24:103-108, 1953.
33. Chief Dietician (M.V.A.H.): Personal communication.
34. Clinic on Psychosomatic Problems: Anaclitic treatment in a patient with ulcerative colitis. Am. J. Med., 14:731-735, 1953.
35. Cooper, R. R.: Definitive treatment for idiopathic ulcerative colitis. Harper Hosp. Bull., 10:45-50, 1952.
36. Corbett, R.: Chronic ulcerative colitis: two specimens, Proc. Royal Soc. Med., 43:684, 1950.
37. Counsell, P. B., Dukes, C. E.: The association of chronic ulcerative colitis and carcinoma of the rectum and colon. Brit. J. Surg., 39:485-495, 1952.
38. Crile, G.: The treatment of ulcerative colitis. Gastroenterology, 23:533-541, 1953.
39. Crile, G., Jr.: Treatment of intractable ulcerative colitis. Postgrad. Med., 12:339-344, 1952.
40. Crile, G., Jr., Turnbull, R. B., Jr.: The treatment of chronic ulcerative colitis. Cleveland Clin. Quart., 18:239-245, 1951.
41. Crohn, B. B.: Regional Ileitis. Page 47. New York: Grune and Stratton, 1949.
42. Crohn, B. B., Garlock, J. H., Yarvis, H.: Right-sided (regional) colitis. J.A.M.A., 134:334-338, 1947.
43. Currie, D. J., Webster, D. R.: The effects of bile and pancreatic juice on the colon: A possible factor in the etiology of ulcerative colitis. Surg. Forum, 148-155, 1950.
44. Dack, G. M.: Bacteriologic aspects of chronic ulcerative colitis (idiopathic). J. Internat. Coll. Surgeons, 19:232-236, 1953.
45. Davies, J. N. P.: The essential pathology of Kwashiorkor. Lancet, 254:317-320, 1948.
46. Dennis, C., Karlson, K. E.: Surgical measures as supplement to the management of idiopathic ulcerative colitis, cancer, cirrhosis and arthritis as frequent complications. Surgery, 32:892-912, 1952.
47. Eddy, F. D.: Late results of vagotomy in the treatment of idiopathic ulcerative colitis and regional enteritis. Surgery, 29:11-23, 1951.
48. Elliott, J. M., Giansiracusa, J. E.: ACTH and cortisone in the treatment of ulcerative colitis. An evaluation of their prolonged administration. New England J. Med., 250:969-976, 1954.
49. Elliott, J. M., Kiefer, E. D., Hurxthal, L. M.: The treatment of chronic ulcerative colitis with ACTH. A clinical study of twenty-eight cases. New England J. Med., 245:288-292, 1951.
50. Engel, G. L.: Psychologic aspects of the management of patients with ulcerative colitis. New York State J. Med., 52:2255-2261, 1952.
51. Engel, G. L.: Studies of ulcerative colitis. II. The nature of the somatic processes and the adequacy of psychosomatic hypothesis. Am. J. Med., 16:416-433, 1954.
52. Felsen, J., Gorenberg, H.: Chronic dysentery, distal ileitis and ulcerative colitis. Am. J. M. Sci., 192:553-556, 1936.
53. Felsen, J., Wolarsky, W.: Acute and chronic bacillary dysentery and ulcerative colitis. J.A.M.A., 153:1069-1072, 1953.
54. Ferguson, L. K.: Surgical treatment of ulcerative colitis. Delaware M.J., 23:297-304, 1951.
55. Flora, W. W.: Radical surgical treatment of chronic ulcerative colitis. Am. J. Surg., 81:92-93, 1951.
56. Follis, L. S.: Barron, J.: Modified technique for total colectomy in ulcerative colitis. A.M.A. Arch. Surg., 67:363-369, 1953.
57. Friedman, M. H. F., Haskell, B. F.: The treatment of non-specific ulcerative colitis for one year with extracts of intestinal mucosa. Gastroenterology, 11:733-841, 1948.
58. Frobese, A. S.: The surgical management of regional enteritis and ulcerative colitis. Surg. Clin. North America, 1725-1744 (Dec.) 1951.
59. Gabriel, W. B.: The surgical treatment of chronic ulcerative colitis. Brit. M. J., 1:881-885, 1952.
60. Gage, A. A., MacCallum, J. D.: Regional colitis confined to sigmoid colon. A.M.A. Arch. Surg., 65:937-940, 1952.
61. Gardner, C., Miller, G. E.: Total colectomy for ulcerative colitis. A.M.A. Arch. Surg., 63:370-372, 1951.
62. Garlock, J. H., Lyons, A. S.: The role of surgery in the therapy of ulcerative colitis. Gastroenterology, 26:709-722, 1954.

63. Grace, W. J.: Life situations, emotions and chronic ulcerative colitis. A.R.M.M.D. Life Stress and Bodily Disease, pp. 679-691, Baltimore: Williams and Wilkens, 1950.
64. Grace, W. J., Wolf, S., Wolff, H. G.: The Human Colon, New York: Hoeber Inc., 1951.
65. Grace, W. J., Pinsky, R. H., Wolff, H. G.: The treatment of ulcerative colitis. II. Gastroenterology, 26:462-468, 1954.
66. Grace, W. J., Wolff, H. G.: The treatment of ulcerative colitis. Gastroenterology, 26:264-272, 1954.
67. Gray, S. J., Reifenstein, R. W., Benson, J. A., Jr., Young, J. C. G.: Treatment of ulcerative colitis with corticotropin (ACTH) and cortisone. A two-year follow-up. J.A.M.A., 148:1489-1497, 1952.
68. Gray, S. J., Reifenstein, R. W., Benson, J. A., Young, J. C. G.: Treatment of ulcerative colitis and regional enteritis with ACTH. Significance of fecal lysozyme. A.M.A. Arch. Int. Med., 87:646-662, 1951.
69. Greville, Fulke: Mustapha. Act V.
70. Groen, J., Bastiaans, J.: Psychotherapy of ulcerative colitis. Gastroenterology, 17:344-352, 1951.
71. Halsted, J. A.: The treatment of ulcerative colitis. M. Clin. North America, 1157-1174 (July) 1953.
72. Halsted, J. A., Yuhl, E. T., Stirrett, L. A., Barker, W. F.: The association of peptic ulcer with chronic ulcerative colitis. Gastroenterology, 26:65-69, 1954.
73. Hiatt, R. B., Engle, C., Flood, C., Karush, A.: The role of the granulocyte as a source of lysozyme in ulcerative colitis. J. Clin. Invest., 31:721-726, 1952.
74. Hoffbauer, F. W., McCartney, J. S., Dennis, C., Karlson, K.: The relationships of chronic ulcerative colitis and cirrhosis. Ann. Int. Med., 39:267-284, 1953.
75. Howard, M. A.: The present status of regional segmental colitis. Western J. Surg., 60:555-562, 1952.
76. Hutchinson, W. B.: Anal ileostomy in chronic ulcerative colitis. Western J. Surg., 60:121-128, 1952.
77. James, Marquis: The Life of Andrew Jackson, New York: Bobbs, 1938.
78. Jensen, E. J., Baggenstoss, A. H., Bargen, J. A.: Renal lesions associated with chronic ulcerative colitis. Am. J. M. Sci., 219:281-290, 1950.
79. Jones, G. W., Baggenstoss, A. H., Bargen, J. A.: Hepatic lesions and dysfunction associated with chronic ulcerative colitis. Am. J. M. Sci., 221:279-286, 1951.
80. Kapel, O.: Medical and modern surgical treatment of chronic ulcerative colitis. Acta. Med. Scand., 138:328-340, 1950.
81. Karush, A., Daniels, G.: Ulcerative colitis: The psychoanalysis of two cases. Psychosomatic Med., 15:140-167, 1953.
82. Kerr, A. B.: Total colectomy. Glasgow M. J., 33:71-82, 1952.
83. Kiefer, E. D., Elliott, J. M.: The effect of ACTH upon the course of chronic ulcerative colitis. Gastroenterology, 26:29-31, 1954.
84. Kiefer, E. D., Eytinge, E. J., Johnson, A. C.: Malignant degeneration in chronic ulcerative colitis. Gastroenterology, 19:51-57, 1951.
85. Kimmelstiel, P., Large, H. L., Verner, H. D.: Liver damage in ulcerative colitis. Am. J. Path., 28:259-290, 1951.
86. Kirsner, J. B., Palmer, W. L.: Ulcerative colitis, considerations of its etiology and management. J.A.M.A., 155:341-346, 1954.
87. Kirsner, J. B., Palmer, W. L., Klatz, A. P.: ACTH and cortisone in chronic ulcerative colitis, a comparison of clinical effects. J. Lab. Clin. Med., 36:-846-847, 1950.
88. Kirsner, J. B., Palmer, W. L., Klotz, A. P.: Reversibility in ulcerative colitis. Radiology, 57:1-14, 1951.
89. Kirsner, J. B., Palmer, W. L., Maimon, S. N., Ricketts, W. E.: Clinical course of chronic non-specific ulcerative colitis. J.A.M.A., 137:922-928, 1948.
90. Kleckner, M. S.: Chronic ulcerative colitis; results of treatment. J. Internat. Coll. Surgeons., 20:592-595, 1953.
91. Kleckner, M. S., Jr., Bargen, J. A., Banner, E. A.: Chronic ulcerative colitis and pregnancy. Am. J. Obst. & Gynec., 62:1234-1242, 1951.
92. Kleckner, M. S., Bargen, J. A., Banner, E. A.: The association of chronic ulcerative colitis and pregnancy. Proc. Staff Meet. Mayo Clin., 27:189-190, 1952.
93. Kleckner, M. S., Jr., Stauffer, M. H., Bargen, J. A., Dockerty, M. B.: Hepatic lesions in the living patient with chronic ulcerative colitis as demonstrated by liver biopsy. Gastroenterology, 22:13-33, 1952.
94. Kullman, H. J., Schmitt, P. E.: Chronic ulcerative colitis in veterans. J. Michigan State M. S., 50:1133-1136, 1951.
95. Lahey, F. H.: Ulcerative colitis. Rev. Gastroenterology, 17:723-736, 1950.
96. Lahey, F. H.: Indications for surgical intervention in ulcerative colitis. Ann. Surg., 133:726-742, 1951.
97. Lahey, F. H.: The place of surgery in patients with ulcerative colitis. Gastroenterology, 24:271-274, 1953.
98. Lake, M., Nickel, W. F., Andrus, W. D.: Possible role of pancreatic enzymes in the etiology of ulcerative colitis. Gastroenterology, 17:409-413, 1951.
99. Leading Articles: Ulcerative colitis with ileal involvement. Lancet, 2:624, 1951.
100. Levine, M. D., Kirsner, J. B., Klotz, A. P.: A new concept of the pathogenesis of ulcerative colitis. Science, 114:552-553, 1951.
101. Lindemann, E.: Modifications in the course of ulcerative colitis in relationship to changes in life situations and reaction patterns. Res. Pub. A. Neur. Ment. Dis., 29:679-691, 1949.
102. Lium, R., Porter, J.: Etiology of ulcerative colitis. Arch. Int. Med., 63:201-209, 1939.
103. Lubran, M., McAllen, P. M.: Potassium deficiency in ulcerative colitis. Quart. J. Med., 20:221-231, 1951.
104. Lumb, G.: Cicatrizing enterocolitis. Brit. J. Surg., 39:233-243, 1951-1952.
105. Lynn, D. H. The relationship of chronic lesions to carcinoma of the colon—chronic ulcerative colitis. Collective Review. Surg. Gynec. & Obst., 81:269-276, 1945.
106. McIver, M. A., Lucas, V.: Pyoderma gangrenosium, a complication of non-specific ulcerative colitis. Surgery, 30:573-577, 1951.
107. McKell, T. E., Tuthill, S. W., Sullivan, A. J.: The affective response of a patient with chronic ulcerative colitis to cortisone. Gastroenterology, 17:20-24, 1951.
108. Machella, T. E.: Chronic (non-specific) ulcerative colitis. A review of medical therapy. Am. J. M. Sci., 222:579-589, 1951.
109. Machella, T. E.: Problems in ulcerative colitis. Am. J. Med., 13:760-776, 1952.
110. Machella, T. E., Hollan, R. O.: The effect of cortisone on the course of chronic regional enteritis and chronic idiopathic ulcerative colitis. Am. J. M. Sci., 221:501-507, 1951.
111. Madison, M. S., Bargen, J. A.: Fulminating chronic ulcerative colitis with unusual segmental dilatation of the colon: Report of a case. Proc. Staff Meet., Mayo Clinic, 26:21-24, 1951.
112. Marshak, R. H., Lester, L. J.: Megacolon, a complication of ulcerative colitis. Gastroenterology, 16:768-773, 1950.
113. Marshall, H. C., Kirsner, J. B., Palmer, W. L.:

Chemotherapy in chronic ulcerative colitis. M. Clin. North America, 257-266 (Jan.) 1951.

114. Massachusetts General Hospital, Medical Grand Rounds: Operative management of ulcerative colitis complicated by rheumatoid arthritis. Am. Practitioner, 3:317-321, 1952.

115. Meyer, K., Gelhorn, A., Prudden, J. F., Lehman, W. L., Steinberg, A.: Lysozyme in chronic ulcerative colitis. Proc. Soc. Exper. Biol. & Med., 65:221-222, 1947.

116. Medical Times: Chronic ulcerative colitis, 79:601-615, 1951.

117. Michael, M. A.: Complications and surgical indications in chronic ulcerative colitis. Postgrad. Med., 9:340-344, 1951.

118. Miller, T. G.: The management of patients with chronic non-specific ulcerative colitis. M. Clin. North America, 1777-1784 (Nov.) 1953.

119. Miller, T. G., Machella, T. E.: Principles in the management of chronic non-specific ulcerative colitis. Am. J. M. Sci., 216:425-429, 1948.

120. Moeller, H. C., Rider, J. A., Kirsner, J. B.: Erythrocyte and serum cholinesterase in ulcerative colitis. Gastroenterology, 19:538-546, 1951.

121. Monaghan, J. F., Bockus, H. F.: Gastroenterology. vol. 2, p. 598. Philadelphia: W. B. Saunders, 1946.

122. Morrison, L. M.: Response of ulcerative colitis to therapy with salicylazosulfapyridine. J.A.M.A., 151:366-368, 1953.

123. Myhre, J.: Resume of Patients Receiving Surgical Treatment of Ulcerative Colitis. Med. Grand Rounds, Minneapolis, VAH, 2:182-187, 1949, and Brakel, F. J., Jr., O'Leary, J. B., Wilson, J. A., Reported at Meeting of the Minnesota Society of International Medicine, May, 1954.

124. Nasio, J.: Medical treatment for chronic ulcerative colitis. Am. J. Proctol., 3:34-58, 1952.

125. Nesselrod, J. P.: Proctology in General Practice. pp. 197-198. Philadelphia: W. B. Saunders Co., 1950.

126. Nickel, W. F., Gordon, G. M., Andrus, W. D.: Studies on several cholinergic drugs as possible etiologic agents in ulcerative colitis. Gastroenterology, 24:556-559, 1953.

127. Nickel, W. F., Gordon, G. M., Andrus, W. D.: Studies on lysozyme as an etiologic agent in ulcerative colitis. Gastroenterology, 17:406-408, 1951.

128. Otani, S., Snapper, I.: On the incidence of carcinoma in chronic ulcerative colitis. J. Mt. Sinai Hosp., 19:275-288, 1952.

129. Palumbo, L. T., Rutgin, G. M.: One-stage colectomy. A.M.A. Arch. Surg., 67:762-768, 1953.

130. Paterson, M., Eytinge, E. J.: Chronic ulcerative colitis and pregnancy. New England J. Med., 246:-691-694, 1952.

131. Pessel, J. F., Bearisto, E. B., Edwards, M. H., Rothmell, T. K.: Multiple perforations occurring during the treatment of ulcerative colitis with ACTH. Am. J. Dig. Dis., 20:318-322, 1953.

132. Plutarch: Translated by John Dryden, Lives. Pericles, 194.

133. Pollard, H. M., Block, M.: Association of hepatic insufficiency with chronic ulcerative colitis. Arch. Int. Med., 82:159-174, 1948.

134. Pope, Alexander: Epistle to Dr. Arbuthnot; Prologue to the Satires.

135. Prugh, D. G.: Variations in attitudes, behavior and feeling states as exhibited in the play of children during modifications in the course of ulcerative colitis. pp. 692-705, A.R.N.M.D. Baltimore: Williams & Wilkens, 1950.

136. Prugh, D. G.: The influence of emotional factors on the clinical course of ulcerative colitis in children. Gastroenterology, 18:339-354, 1951.

137. Ravenscroft, Jacula: Prudentum, 2nd Ed., 1651.

138. Reifenstein, E. C., Jr.: Conference on Metabolic Aspects of Convalescence. Tr. 15th Meeting. Report by Howard, J. E., Duncan, R., New York, March-April, 1947.

139. Reifenstein, R. W., Gray, S. J.: The effect of ACTH upon the fecal lysozyme titre in ulcerative colitis. Gastroenterology, 19:547-557, 1951.

140. Rice-Oxley, J. M., Truelove, S.: Ulcerative colitis, course and prognosis. Lancet, 1:663-669, 1950.

141. Ripstein, B.: Primary resection of the colon in acute ulcerative colitis. J.A.M.A., 152:1093-1095, 1953.

142. Ripstein, C. B., Miller, G. G., Gardner, C. McG.: Results of the surgical treatment of ulcerative colitis. Ann. Surg., 135:14-21, 1952.

143. Ross, O. B., Jr.: Use of controls in medical research. J.A.M.A., 145:72-75, 1951.

144. Ross, J. R., Swarts, J. M.: Hepatic dysfunction and cirrhosis in chronic ulcerative colitis. Gastroenterology, 10:81-95, 1948.

145. Rossmiller, H. R., Brown, C. H., Ecker, J. A.: The effect of ACTH on non-specific ulcerative colitis. Gastroenterology, 17:25-27, 1951.

146. Rowe, A. H.: Chronic ulcerative colitis—allergy in its etiology. Ann. Int. Med., 17:83-100, 1942.

147. Sammons, H. G.: Mucinases in ulcerative colitis. Gastroenterologia, 76:346-349, 1950.

148. Sammons, H. G.: Mucinases in ulcerative colitis. Lancet, 2:239-240, 1951.

149. Sammons, H. G.: The digestion of intestinal mucosa. Biochem. J., 54:9-10, 1953.

150. Sappington, T. S., Bockus, H. C.: Nitrogen metabolism in chronic ulcerative colitis and its therapeutic significance. Ann. Int. Med., 31:282-302, 1949.

151. Sawitz, M. A., Greenberg, M. S.: Skin lesions in association with ulcerative colitis. Gastroenterology, 19:476-479, 1951.

152. Schlicke, C. P., Bargen, J. A.: Fulminating ulcerative colitis. A critical analysis of twenty cases. Minnesota Med., 23:348-352, 1940.

153. Scott, H. W., Cantrell, J. R.: Colonmetrographic studies of the effects of section of the parasympathetic nerves of the colon. Bull. Johns Hopkins Hosp., 85:310-319, 1949.

154. Shakespeare, Wm.: Macbeth. Comedies and Tragedies of Shakespeare, New York: Random House, 1944.

155. Shands, W. C., Dockerty, M. B., Bargen, J. A.: Adenocarcinoma of the large intestine associated with chronic ulcerative colitis. Surg., Gynec. & Obst., 94:302-310, 1952.

156. Sloan, W. P., Bargen, J. A., Gage, R. P.: Life histories of patients with chronic ulcerative colitis: A review of 2,000 cases. Gastroenterology, 16:25-38, 1950.

157. Snorf, L. D.: Chronic ulcerative colitis. M. Clin. North America, 243-255 (Jan.) 1951.

158. Sommers, S. C., Anderson, L. M., Warren, S.: Basement membranes in chronic intestinal diseases. Lab. Invest., 2:223-226, 1953.

159. Spellberg, M. A., Mosiman, W. D., Smith, L. C.: Electrophoretic studies of plasma proteins in ulcerative colitis. J. Laborat. & Clin. Med., 36:991, 1950.

160. Standen, A.: Science is a Sacred Cow. New York: Dutton Co., 1950.

161. Stoughton, R. B.: Enzymatic cytolysis of epithelium by filtrates of feces from patients with ulcerative colitis. Science, 116:37-39, 1952.

162. Stoughton, R. B.: Enzymatic cytolysis of epithelium by filtrates from patients with ulcerative colitis. J. Invest. Derm., 20:353-356, 1953.

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Laboratory Aids to Medical Practice

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BIOPSY OF LYMPH NODES

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BIOPSY of lymph nodes for diagnostic purposes is a procedure that many physicians compare with the art of picking the most-likely-to-succeed box of crackerjack. This attitude is no doubt born of the somewhat capricious results of the enterprise.

Despite a certain degree of validity in the above simile, it is nevertheless true that discretion is rather more conducive to reward in biopsy of lymph nodes than in selection of crackerjack. Two attitudes toward the procedure are likely to subtract from its value:

1. The clinician may have a notion that the histopathologist is a wizard with a crystal ball who can look at microscopic sections of any lymph node and diagnose the disease that has baffled all.

2. The pathologist may have a notion that the first notion is correct or at least ought to be fostered in the clinician for purposes of morale.

Both clinician and pathologist should be capable of the self-confession that in order to make a histologic diagnosis the tissue must include the lesion and that, other things being equal, the largest lymph node detectable is most likely to provide the answer when the question centers around malignant lymphoma or an infectious process. When metastasis is also under consideration, the direction of lymphatic drainage in relation to a possible primary lesion justifiably may alter the importance of size alone.

Perhaps the most resistant problems in biopsy of lymph nodes occur in relation to the diagnosis of malignant lymphoma. From the pathologist's point of view most such problems can be eliminated by observance on the part of the surgeon of two basic rules:

1. In the presence of general adenopathy, the most desirable site of biopsy is usually the cervical region. Inguinal nodes are often enlarged from prior adenitis secondary to infections of the genitalia or legs, including numerous forms of dermatitis (lipomelanotic reticulosis). Axillary nodes have a habit of feigning disease, only to show their size is due to replacement by fat ("horseshoe node"). It is true that cervical adenopathy may result from infection of the upper part of the respiratory tract, but a history of such should not be distracting since many patients with cervical lymphoma relate the onset of their trouble to a sore throat, tonsillitis or "cold."

2. It is important that the pathologist receive the excised node or nodes in as nearly an intact and untraumatized state as possible. In certain forms of lymphoma, such as mature lymphocytic lymphosarcoma and follicular lymphoma, no diagnostic qualitative cellular changes are present and recognition depends largely on architectural features considered in conjunction with the cytologic population of the node. A fragmented specimen loses much of its value. Actually, in many cases of lymphadenopathy, a cluster of lymph nodes is present in the region of biopsy. One or more of these nodes often will prove nondiagnostic, while others readily provide an answer. For this reason, the surgeon should not be reluctant to remove the entire group intact, regardless of his utter faith in the ability of the pathologist to make a diagnosis from the most minute fragment of tissue.

Needle biopsy of lymph nodes for diagnosis of lymphoma has been recommended, particularly in Europe. Certain types of lymphoma, such as Hodgkin's disease, reticulum cell sarcoma and

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highly immature forms of lymphosarcoma, can be diagnosed occasionally on this basis. However, a negative result has practically no significance because the lymphoma may have involved only a portion of the node, which did not happen to be sampled by the needle, or the lymphoma may have been of a type in which qualitative cytologic features are absent. Histopathologic diagnosis of lymph nodes is difficult enough without conceding a handicap at the start. The most satisfactory and accurate approach probably is a combined histologic and cytologic examination, the latter being performed on imprints of the node stained by the Wright or the May-Grünwald-Giemsa method.

A place exists for examination of fresh frozen sections of lymph nodes if the limitations of the method are recognized. It is often possible by this means to determine the adequacy of the specimen while the patient is still on the operating table. If no significant changes are shown by rapid section, further exploration for more significant material can be carried out at once. If metastatic malignant invasion is recognized, the procedure can be concluded with the assurance that a second biopsy will be unnecessary. Also a fresh frozen section may offer guidance as to bacteriologic handling of the specimen before it has been rendered bacteriologically useless through fixation. The pathologist can be of real service to the patient by recognizing granulomatous reactions and directing animal and cultural inoculation for various bacteria and fungi. It is of equal importance to culture material that is histologically suggestive of Boeck's sarcoid, since negative results of culture and animal inoculation are of highest significance in the diagnosis of this condition.

A good routine practice is to divide all specimens of lymph nodes into two portions, one for bacteriologic examination and one for histologic study. With a sharp knife, the surgeon can cleanly transect the specimen at the operating table, placing one portion in a sterile container and submitting the remainder for histologic examination. Rarely is the specimen too small for this purpose.

An old rule that is almost an axiom states that lymphadenopathy in adults is due, with rare exceptions, to malignant lesions. In actual practice, however, the exceptions are not rare and the clinician who implies by his surprise and chagrin that the pathologist has missed the boat in returning a report of "hyperplastic lymph node" should remember that, even excepting the granulomas, there still remain many nonmalignant disorders associated with significant lymphadenopathy. These include, besides a sizable group of idiopathic lesions, such conditions as infectious mononucleosis, syphilis, lupus erythematosus and rheumatoid arthritis. Lymph nodes in the last disease have a facility for imitating malignant lymphoma, particularly follicular lymphoma and Hodgkin's disease.

Biopsy of lymph nodes perhaps has not been employed to its fullest advantage in therapeutic radiology. In malignant lymphoma proved by a single biopsy, it is natural to interpret clinically detectable nodes elsewhere as being involved by the same process and thus targets for radiation. Such assumption is by no means supportable by fact. If the radiologist desires to reserve treatment for involved areas, multiple biopsies of questionable areas can be a decided guide to his attack. Also, a change of type in the lymphoma concerned may be discovered and may serve to explain an otherwise puzzling change of course in the disease.

Possibly the most outstanding innovation in biopsy of lymph nodes is the "blind" scalene, or paratracheal, node dissection. Introduced in 1949, this approach calls for removal of clinically nonpalpable lymph nodes from the supraclavicular region in the presence of indeterminate intrathoracic disease. The method has been of greatest value when mediastinal or hilar enlargement is demonstrable roentgenologically. Positive results have been obtained in a third of the cases in one large series. The method has been most productive in Boeck's sarcoid but also has been informative in tuberculosis, histoplasmosis, silicosis, malignant lymphoma and bronchogenic carcinoma. Although this procedure has not been fully evaluated it would appear impossible not to detect a faint odor of crackerjack in the situation.

President's Letter

EARLY DETECTION — A "MUST"

For many years now, those who are vitally interested in a cure for, or at second-best, the control of the dreaded scourge of cancer, have been emphasizing the importance of early detection.

In a concerted effort to impress this importance on the public mind, and the increased rate of cure with early detection, the State Association's Cancer Committee has co-operated with the Minnesota Division of the American Cancer Society in developing the program, "Every Doctor's Office—A Cancer Detection Center."

Now, the program is being accelerated, and united action is necessary to make it succeed. In the initial stages of the plan, county societies were requested to enlist aid of individual members to implement the program. Information and forms were sent to all Association members of county societies which had approved the program.

In the near future, all Association members will receive more information and a brochure, telling of the need for their help in reducing cancer mortality, and in increasing cure by early detection. The efficient methods of early detection employed by the Cancer Detection Center at the University of Minnesota are to be utilized to their fullest extent in operating this program.

A certain amount of county society apathy has been encountered, and it is hoped that this is only because the county groups have not had a chance to meet and approve the program. I would strongly urge county societies to consider the program with renewed interest, realizing the great good individual members can do in helping to eradicate the incidence of cancer. It is emphasized that the cancer detection examination in the individual doctor's office is a relatively simple procedure, for all forms and guiding information will be supplied by the Cancer Committee through the auspices of the Minnesota Division, American Cancer Society.

Through united action, Minnesota physicians are in an excellent position to be benefactors to many persons who do not realize that cancer can actually be cured, if it is caught early enough. Physicians are urged again to become active participants in this important work—for through early detection, the goal of a high cure rate can be realized in this state. As individual physicians, and as an Association, we cannot afford to miss this opportunity to be of further service to the people of Minnesota.

President, Minnesota State Medical Association

Editorial

Arthur H. Wells, M.D., *Editor;* Henry G. Moehring, M.D., and John F. Briggs, M.D.

DRINKING DRIVER—SPORTSMAN OR MURDERER?

MINNESOTA is headed for an all-time record in the number of traffic accident fatalities. The State Highway Patrol is fighting a gallant but losing battle and is in desperate need of help from our profession.

It is generally agreed that one of the major factors in the nation's traffic accident rate is the drinking driver. In a scientifically sound study of car drivers in Illinois (Holcomb J.A.M.A., 111:1076, 1938) showed that a man under the influence of alcohol (0.15 grams or more of alcohol per 100 ml. of blood) had a fifty times greater chance of having an accident, than a sober man. Statistics and conservative estimates indicate that approximately one out of every six fatal car accidents is associated with a drinking driver. Of the 481 fatal motor vehicle traffic accidents in Minnesota during 1952, forty-nine (10 per cent) were proved to be associated with drivers under the influence of alcohol. There were at least 1290 persons injured during this period in accidents occurring under similar circumstances.

At the present time, the key to a partial solution of this problem is the enactment of legislation which relates blood alcohol levels to expected clinical manifestations. The American Medical Association and the American Society of Clinical Pathologists have long since expressed their approval of the principal features of the bill which failed to be passed by the Minnesota State Legislature during 1953. Since these matters are in the field of Medicine, one could hardly expect the layman to act without some active guidance from members of the profession in Minnesota. What is the explanation for our static inertia? Are there too many two-fisted drinkers among us, or is it the problem of someone else?

THE DOCTOR AND TELEVISION

AS PHYSICIANS, we have been derelict in our failure to accept our responsibility to instruct the public concerning medical care. This has come about partly through our heritage of the belief that it was unethical for a physician to be in the public eye. As a result, our medical societies have accentuated our poor public relations. Too often have we accepted the philosophy that "the public be damned." It has always been our responsibility to furnish the public with sound medical information. In failing to do so, we have made it possible for many unappropriate and incorrect medical philosophies to be propagated. Within recent years, organized medicine has recognized this responsibility to the public, and has emphasized that we as physicians must participate in those programs that give the public information. The first step in this direction was the development of Speakers Bureaus by the county medical societies as well as the state organizations to furnish speakers for lay groups. These speakers did and still are doing an excellent job in bringing true scientific medical knowledge to the laity. These Speakers Bureaus are increasing in number, and more and more physicians are accepting their obligation to the public by joining them. In addition, the voluntary health agencies, such as the Heart Association, the Cancer Society, the Christmas Seal Group, and the like, have brought a great fund of information to the laity concerning matters of health. A good deal of the information that they have disseminated arose originally from a small group of physicians who were interested in the problem of each voluntary health organization. Now we find that all of these organizations have their own physicians Speakers Bureau and, as a result, have brought prestige to the educational programs of these voluntary health agencies.

The result of physicians engaging in these activities has reflected itself in the public's acceptance of the medical programs. The public is vitally interested in medicine and in their own health, and the public is deeply anxious to find out what is new in medicine. A medically educated public will be the greatest supporter we have in our fight against socialized medicine. The people appreciate and recognize the gratuitous efforts by the physician in bringing them medical information. The public also recognizes the great benefit derived from such an educational program.

With the development of the Speakers Bureau, it was soon apparent that this talent could also be directed into radio. Over a period of years, both organized radio lectures on health and lectures by physicians sponsored by the voluntary health agencies became prominent. Here, too, the public responded enthusiastically to the physicians' efforts on their behalf. Now television has increased the opportunity for the medical profession to be of public service. The development of medical panels, particularly those discussing questions of timely interest in health, has proven of great value and popularity. In Minnesota, the Doctor's Round Table, a public service program, designed by WCCO-TV and conducted under the sponsorship of the Minnesota State Medical Association and the Minnesota Tuberculosis and Health Association, has been eminently successful. This panel, by inviting outstanding authorities in tuberculosis as guests, has brought to the public of Minnesota a tremendous knowledge of the problem of tuberculosis. This panel also diversifies itself sufficiently to bring information concerning other diseases to the attention of the public. The Minnesota State Medical Association and the University of Minnesota, under the leadership of Dr. James Fox, carries a program on WCCO-TV on Mondays which has proven to be of immense interest to the people. This program has done an outstanding job in medical education. During the month of February, 1954, the Minnesota Heart Association, through the courtesy of KSTP-TV and under the leadership of Bee Baxter, presented a series of four lectures over radio and television concerning heart disease. These lectures were so popular that the station, in conjunction with Miss Baxter, has invited the Minnesota Heart Association to conduct a further series of such programs. The Minnesota State Medical Association, in conjunction with the Minnesota Heart Association, is now sponsoring a series of twelve such programs. These programs will be directed not only through television media but also by radio. The title of this series is "What's New in Medicine" and brings to the public information concerning a great variety of health problems. The television programs give an excellent opportunity for the presentation of material in a graphic method. The use of x-ray films, electrocardiographic tracings, surgical instruments, and the like enhances the interest of the viewer and listener. The use of the animated films to show the function of the heart, the use of medical educational films and other motion picture material has proven very effective. Throughout all of these programs the philosophy is maintained that a person who has symptoms or who is in need of medical care should consult his family medical advisor for guidance.

The Minnesota State Medical Association and its committee on radio and television must be congratulated on their acceptance and enthusiastic development of this new means of lay education. Under the leadership of the Council of the Minnesota State Medical Association and the voluntary health organizations, radio and television programs have been developed which will better our public relations. The Minnesota State Medical Association, in conjunction with the many voluntary health agencies, is doing everything possible to facilitate this type of education. The State Medical Association, as well as all the physicians in the State of Minnesota, is indebted to the various radio and television studios who have given so freely of their time to make this public service possible. We, who are practicing in Minnesota are indeed fortunate that our leaders in the medical profession have grasped the significance of the newer type of public relations and have done so much to make these programs possible and successful.

J. F. B.

GENERAL SURGERY AS A CAREER?

If a medical student or interne asked me whether to take up general surgery today, I would not conscientiously be able to advise him to do so. The one exception to this generality is that small towns with small and limited hospital facilities still have a need for the general surgeon. Otherwise, I beieve that the general surgeon's field of activity will diminish more and more as the years go by.

Whereas, in Duluth, thirty years ago, six surgeons did about 90 per cent of all surgery, now there are over sixty surgeons, though the population has remained about the same. This indicates that formerly general surgeons had a wide field of activity, but now more specialists are operating in a particular field, and more general practitioners do the common operations.

In the old days, training in general surgery was based on the assumption that the field began with the head and ended with the feet. Training both here and abroad assumed almost unlimited individual competence to operate anywhere on the human body. Now the head is almost completely excluded from the general surgeon's field, as are the skeletal system, the skin, the lungs, heart, genito-urinary and gynecological systems. Specialization has narrowed the general surgeon's field to the gall bladder and biliary ducts, hernias, and benign and malignant lesions of the gastrointestinal tract. Even the latter is divided into several sections of specialty.

In my opinion there are today three factors that are limiting, and will continue to limit, the general surgeon's field of activity: (1) The growth and excellence of specialties and the continued education of the public to seek specialists in surgery; (2) the elimination of many surgical procedures through the use of antibiotics, radiotherapy, and other non-surgical treatments; (3) the increasing number of general practitioners doing the more common surgical operations, with or without assistance.

It was colorful and dramatic to watch the general surgeon of days gone by as he removed a clot from the brain surface one day, and in the next few days as he performed a thyroidectomy, drained an empyema, amputated a leg or an arm, resected a stomach or intestine or removed a gall bladder. The latter is about the only thing left to him today.

Therefore, when I am asked by a medical student or interne whether or not he should practice general surgery, my advice to him is to study and practice the surgical *specialty* of his choice.

A. E. Olson, M.D.

MEDICAL JOURNALISM: A NEW ADDITION TO THE COLLEGE CURRICULUM

LARGELY as the result of work by the Educational Committee of the American Medical Writers' Association, students may now pursue a four-year collegiate course in medical journalism. This course leads to a bachelor's degree and is offered by the School of Journalism and Communications, University of Illinois, the School of Journalism, University of Missouri, and the School of Journalism, University of Oklahoma.

The student who elects this course will spend approximately one-fourth of his training in professional courses in journalism; the remainder will be devoted to the humanities and sciences. The latter will include work in various biologic sciences, much of which will be provided in the medical and related departments of the university in question. As stated by Dr. R. M. Hewitt,* who is chairman of the committee that promoted this course, the aim was to find an educational institution to which we could go for graduates whose exposure to both the humanities and the sciences had been sufficient to make them of some use to us from the start of their employment as editors or . . . as medical writers, medical abstracters and medical advertising writers.

It is a rare person, indeed, who is well founded in both medicine and journalism. Yet a great demand exists for the individual who is trained to write well and who knows enough medicine to write intelligently about this particular field. It is to supply such writers that this new course is designed. Persons so trained will be able to fill positions in such places as larger medical clinics, houses publishing medical journals or books, various public health agencies, companies devoted to the manufacture of drugs and chemicals, and medical advertising agencies. Such work will be well compensated because the demand far exceeds the supply, which at present is practically nonexistent except on a fortuitous basis.

Much criticism has been leveled recently at scientific writing, especially that which pertains to medicine. There is no denying that the quantity of medical writing is too great, whereas its quality leaves much to be desired. This problem is being recognized by more and more physicians, as witnessed by the growth in membership in the American Medical Writers' Association from forty in 1947 to a total of 482 in July, 1954. The future production of well-trained medical writers should do much to improve medical writing by providing consultation from such persons, either as members of the Medical team or as free-lance writers.

The institution of this new collegiate course is of such importance that it should be made known to all physicians. Further information concerning the purposes and ultimate aims of this type of training caan be found in an article by Dr. Harold Swanberg.† Details relative to the course itself can be obtained by writing to any of the three universities already mentioned. It is hoped that physicians who know competent young people about to begin college will pass on this information to them in an effort to enlist such persons in the career of medical journalism.

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*Hewitt, R. M.: 1953 Report of the Educational Committee, American Medical Writers' Association. Mississippi Valley M. J., 76:3-6 (Jan.) 1954.

George G. Stilwell, M.D.

ROUTINE CHEST ROENTGENOGRAMS

MINNESOTA has made good progress in the acceptance by hospitals of the responsibility of doing routine radiographic studies of all patient admissions. Forty of the 200 general hospitals in Minnesota have now adopted this routine. Yet, think of the fact that 160 of our general hospitals still admit thousands of patients annually who are being denied what is now considered a very necessary health protection measure.

Most large hospitals routinely obtain chest roentgenograms of all patients admitted to their wards, principally because they have a resident radiologist and are able to give quick, efficient service. In some instances, technical costs seem to be the deterring influence. The seeming necessity of securing a photofluoric apparatus also appears to stand in the way of progress. Yet, the installation of photofluoric apparatus is not advisable unless there are more than 3,000 or 4,000 cases a year. Nearly all hospitals are equipped to give an examination on a 14 x 17 film, and in most cases the cost to the patient could be held under $2.00. Certainly, in most instances, it would not be more than that amount, provided the radiologist is willing to forego his professional fee. In some cases, the professional fee is as low as 50 cents.

Since the Minnesota Department of Health is no longer able to make mass radiographic studies, hospitals as the health centers of the community are the logical places to assume this responsibility. In some studies, the incidence of tuberculosis of patients admitted to hospitals has varied between 2 and 3 per cent. Such other pathological findings as cardiac enlargement, pulmonary neoplasm and tumors run nearly 10 per cent. From the standpoint of the control of tuberculosis, the routine x-ray examination in the hospital seems to be the best measure. The people in charge of hospitals should be aware of the other values to health through chest roentgenograms. Efforts should be made to increase as rapidly as possible present facilities and personnel to obtain a high percentage adoption of the x-ray case-finding methods in all hospitals in Minnesota.

Ray Amberg, *President*
Minnesota Tuberculosis and Health Association

STEROID THERAPY IN DERMATOLOGY

(Continued from Page 630)

tained even after discontinuation of the treatment.

In both lupus erythematosus and pemphigus, the steroid dosage is an individual matter. When cortisone fails or loses effectiveness, one may wish to use hydrocortisone or corticotropin (ACTH); in emergencies, the latter drug may be given by intravenous drip.

E. Systemic Disease.—

There are various other systemic diseases for which steroid therapy can be helpful but probably is not curative. Among these are periarteritis nodosa, sarcoidosis, leukemia and related disorders.

F. Varia.—

In various other conditions, treatment has been recommended by some observers but has not been found satisfactory by others. Though steroid therapy is probably the best available treatment for dermatomyositis, one cannot get very enthusiastic about the results. Steroids are of doubtful value in herpes zoster. They probably should not be used in scleroderma or in alopecia areata because of the risk of hypertensive renal disease, particularly in the former, and the necessity for continuous therapy in the latter if one expects to retain growth of hair. The arthropathy associated with psoriasis may respond to steroid therapy, but seldom does the psoriasis itself improve, and this treatment should not be used for psoriasis without arthropathy.

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†Swanberg, Harold: Medical journalism and writing courses now available: something new in collegiate education. Mississippi Valley M. J., 76:147-150 (July) 1954.

The Dean's Page

THE WORLD HEALTH ASSEMBLY

It was my privilege recently to serve as a member of the United States delegation to the Seventh World Health Assembly which met in Geneva, Switzerland, May 4 to 22, 1954.

The World Health Organization, with a membership of eighty-one countries, is a specialized agency of the United Nations. Of the eighty-one member nations, only sixty-five are active, as Russia and her satellites withdrew after the first World Health Assembly. Sixty-one of the sixty-five members were represented at the 1954 Assembly.

The meetings of the World Health Assembly were held in the beautiful large assembly hall and associated committee rooms at one end of the magnificent Palais des Nations (League of Nations Building). In the Council Chamber at the other end of this same building, the Asiatic conference was holding its highly secret, continually frustrating, and frequently bitter meetings. There was no intermingling of the groups whatsoever. In fact, our information as to what went on in the Asiatic conference was obtained primarily from the Paris edition of the *New York Herald Tribune.*

Most people would assume that the deliberations of the World Health Assembly would be concerned primarily with discussions of public health problems of international importance. Such, however, is not the case. Several special sessions are devoted to Technical Discussions, the subject of which at this assembly was "Rural Health." The major functions of the World Health Assembly, however, are legislative in character. Reports of health problems and health activities in different parts of the world are presented. Programs and policies, as recommended by the Director General and the Executive Board, are considered. Budget and scales of assessments are decided upon, and the relations of the World Health Organization to other international health organizations are reviewed.

To me, all of this, including budgets and scales of assessments, was most interesting. It gave one a bit of insight into the manner in which international organizations function, when the vote of a small, underdeveloped country counts as much as the vote of the United Kingdom or the United States of America. It showed what great patience, tact, and seemingly endless negotiations are necessary to get things done, particularly when national interest, national prestige, or national pride are concerned.

The accomplishments of the World Health Organization in the prevention of disease in many areas of the world are most impressive, and one cannot fail to be convinced that this is a program which should be expanded and intensified as rapidly as possible. In the judgment of all of us who made up the United States delegation, the limitation of three million dollars which Congress has placed on the United States contribution should be raised, or better still, removed. Larger expenditures in this field would not only strike at the roots of the conditions which provide fertile grounds for the empty promise of communism, but in addition they will build world-wide good will toward this country in a manner and degree that armaments can never equal.

Health is a subject in which nations, irrespective of their political philosophies, have a common interest. As such, more extensive international co-operation in the field of health may prove to be a common ground for the development of understanding and co-operation that could extend into more difficult areas. This is a field in which the United States has the ability as well as a unique opportunity to assume world leadership.

H. S. Diehl, M.D.
Dean of the Medical Sciences
University of Minnesota

Medical Economics

Edited by the Committee on Medical Economics
of the
Minnesota State Medical Association
George Earl, M.D., Chairman

HEALTH INSURANCE SHOWS "MUSHROOM GROWTH"

In a recent article on health insurance, the *Wall Street Journal* says of it: "It's Growing Like a Mushroom, though Plan for a Hothouse Fails." The lead of the story continued the simile by stating:

> "The multi-billion-dollar health insurance business has quintrupled in size in the past ten years. There is no sign this explosive growth will come to a halt, even though Democratic congressmen teamed up with conservative Republicans yesterday to defeat an Eisenhower Administration plan for a speed-up.
>
> "What the President aimed to do was build a hothouse where health insurance growth could be fertilized with Federal funds."

In spite of this, the article notes, "today some 100 million Americans have some form of protection against future hospital expenses. Over 75 million have insurance against surgical bills, and nearly 40 million have coverage on doctors' calls, usually just in the hospital." This amounts to 60 per cent of the total population of the country who carry health insurance in one, two or more forms.

Variety Noted

The article points out that the variety of coverage today is almost endless. It states:

> "In company-union negotiations, especially for huge groups like the Steel or Auto Workers, sharp-penciled actuaries will tailor a medical 'fringe benefit' to fit almost any employer's pocketbook, and dozens of insurance companies will battle for the contract. For individuals, policies are available for everything from dental cavities to 'catastrophic illness' with surgical bills as high as $10,000."

In proof of the statement that this business is one of the most rapid mushrooming phenomena this country has seen, the article quotes an official of the Health and Accident Underwriters Conference as saying, "The accident and health business is the fastest-growing of any insurance line." Figures are also quoted:

> "More than 800 insurance companies of all kinds, even some concentrating on fire insurance, are active in this business. Add to this the eighty-three regional Blue Cross plans, providing hospital coverage, and seventy-nine Blue Shield organizations, offering surgical protections; these two non-profit groups account for more than $1 billion of the present $3 billion total of annual premiums. . . . More than 300 colleges have enrolled about 900,000 students in health plans. It's estimated that several hundred doctors have arranged local systems for their patients. Just recently, a group of New York dentists organized a dental insurance plan, claimed to be the first comprehensive effort to take the pain out of dental bills."

Cites Economic Factors

The article emphasizes the fact that all plans have been bothered by rising costs. It states that hospital costs have been going up at the rate of 1 per cent a month and now average $21.09 a patient a day. This cost has doubled in the past eight years—although the average hospital stay has shortened at the same time.

Complaints on Criticism

Because some insurance companies are marketing health insurance as a "loss leader" in order to open the door for package deals for other types of coverage, such as life, auto and fire, many of the "Blues" and the labor co-operative groups are complaining that they cannot compete. The article quotes a secretary of a co-operative health plan as saying: "We have met almost insurmountable competition as the old-line commission companies have bid so desperately for group business, especially for welfare plans in labor unions. The co-operatively sponsored organizations which have tried to compete in the field have either lost out or taken heavy losses."

The over-all picture of the entire health insurance situation in America seems to indicate that this field is enjoying a great boom; and one that is largely sparked by the very thing it originally set out to prevent: too much government intervention in the field of health. For it has been found that every time legislation is proposed or threatened which would allow the government to come in on some part of this field, an increase in health insurance business is noted. At least it appears that, from the view of organized medicine and unshackled medical practice at any rate, government attempts to get into the field have done some measure of good in a round-about way.

OFFICIAL WARNS OF SOCIAL SECURITY

"The average American thinks of the Social Security plan as a low-cost program with little idea of what it is going to cost in the future directly and indirectly." This statement was made as a warning by Ray D. Murphy, president of the Equitable Life Assurance Society of the United States, who spoke at the annual meeting of the National Industrial Conference Board some time ago, reported in Insurance Economics Surveys. Accordingly, he said, it is important at this time, when the whole plan is under revision, to encourage wide discussion of the old-age benefits under the system.

Warning that the present tax rate for Social Security is not stable at that rate (2 per cent from the employer, 2 per cent from the employee), he said that necessarily, "the top figure is uncertain since the result depends on many uncertainties."

Notes Misconceptions

Mr. Murphy sounded this fear: "A dangerous thing about Social Security in the United States is that the American people have not yet come to fully realize that 'More can be given,' as Lord Beveridge said, 'only by taking more.' The nation simply does not get something for nothing in Social Security." He continued:

> "Another fundamental misunderstanding among many of our people has been that Social Security taxes are a form of savings being stored up for the future and guaranteeing their future benefits. . . . Use of the word 'insurance' is very objectionable to those in the insurance business. The words 'social insurance' slip easily from the tongues of many people, but the word 'insurance' suggests an individual equity relationship which simply does not exist in OASI. Neither is OASI based on commonly accepted insurance principles."
>
> "Moreover, the incorrect impression that OSAI taxes are insurance premiums or savings has doubtless misled many into thinking that the government has sufficient money stored up to meet the benefits becoming payable in the future from wage credits already acquired. The uninitiated, when told that the OSAI holds some $19 billion in government bonds may have such a belief strengthened. Little do they realize that some $200 billion would be needed at the present time to cover the system's accrued liabilities."

Mr. Murphy stated that essentially the OASI system is operating as a pay-as-you-go plan with a moderate contingency fund available to act as a buffer to cover any temporary excess of benefit payments over tax receipts—an excess such as might occur in a business recession. Putting it another way, he explained that present active workers and employers are paying taxes for benefits to fellow citizens now on the benefit rolls; the active workers and employers themselves must look to future active workers and employers to do the same for them.

SOCIALIZED MEDICINE "TO STAY" IN BRITAIN

Speaking on the same program, Lord Beveridge of Great Britain stated that socialized medicine in his country "has come to stay." His famous Beveridge Report led to Britain's National Insurance Act of 1946, according to a recent issue of Insurance Economics Surveys. Lord Beveridge, seventy-five-year-old economist, conceded that the National Health Service was more expensive than had been expected and that it was in need of "tuning up." But, he added, there was no thought in Britain that the service should be abandoned.

Discussing the role of government in assuring the welfare of the people, he said: "The part of the state is to establish a minimum below which no man need fall, leaving freedom and responsibility for each man to make and live his life above the minimum."

Irradiation, triethylene melamine, and nitrogen mustard are usually the most effective therapeutic measures available for Hodgkin's disease, lymphosarcoma, follicular lymphoblastoma, and reticulum cell sarcoma.

Public Health

MINNESOTA HAS EPIDEMIC OF VIRAL HEPATITIS

Infectious hepatitis has shown a definite increase all over the United States during the last few years. Minnesota has been no exception. During 1953, there were 1192 cases with fourteen deaths reported to the Minnesota Department of Health. For the first seven months of 1954, there have been 1,772 cases reported. That figure would seem to indicate a decided increase in the number of reported cases for this year over last year. The following table shows the steady increase in reported cases for Minnesota during the last twenty years:

5-year period	*Cases*	*Deaths*
1934-1938	72	6
1939-1943	380	5
1944-1948	610	33
1949-1953	2071	45

Outbreaks of this disease have reached epidemic proportions in several Minnesota communities. Reports of a serious increase in the number of cases encountered by physicians came from the Little Falls area last year. The epidemic now appears to be subsiding there, but other areas have reported exceptionally large outbreaks this year. These localities include the counties of Faribault, Freeborn, Rice, Sibley, and Waseca in the southern part of the state; the Twin Cities, Anoka, and Washington counties in the metropolitan area; also Itasca, Mahnomen, Mille Lacs, Morrison, and Otter Tail counties in central Minnesota.

Mortality from the disease has been relatively low, as indicated by the table above and by the fact that only two deaths from infectious hepatitis have been reported for the current year. Deaths are uncommon in persons under forty years of age. Of the fourteen deaths in Minnesota last year, all but two were in persons over forty, nine of them being over fifty years of age. Cases, on the other hand, are most common among children and young adults.

Nearly all cases of this disease reported to the Minnesota Department of Health have been of the infectious variety rather than the homologous serum type. The two types of hepatitis are clinically indistinguishable. The chief points of difference are the incubation period and the mode of spread. The incubation period for infectious hepatitis is from fifteen to thirty-five days, the average being about twenty-five days. In serum hepatitis, the incubation period may be as long as six months; probably a period of twelve to fourteen weeks is most common. Infectious heptatitis is spread almost entirely by the fecal-oral route. Respiratory spread and spread by contaminated blood are negligible. In serum hepatitis, the mode of spread is by inoculation of infected blood or blood products and by use of needles and syringes contaminated with traces of blood from infected persons.

Both types of hepatitis are caused by a filterable virus—Virus A in the infectious type, Virus B in serum hepatitis. Both viruses are relatively resistant to heat but are destroyed by autoclaving or by boiling for fifteen minutes. Virus A is present in the blood for a short time during the acute stage of the disease and in the stools for a longer period. This virus has not been demonstrated in nasopharyngeal secretions or urine. Virus B has been known to persist in the blood of infected persons for as long as five years.

Control Measures

As in all diseases spread by the fecal-oral route, control of infectious hepatitis depends chiefly on good personal and community hygiene. Patients should be isolated for the first week of the illness. Bowel discharges should be properly disposed of. Outdoor toilets should be kept clean and flyproof. Hands should always be washed thoroughly with soap and water after use of the toilet, before handling food and before eating. Children should be excluded from school until clinically well. Quarantine of contacts is not indicated.

Gamma globulin has proved effective at least as a temporary preventive of infectious hepatitis. It should be given to all household contacts, regardless of age. It is available to physicians from the Minnesota Department of Health for preventive treatment of contacts. During the first six months of 1954, the health department distributed 10,468 cc. of GG for the prophylaxis of hepatitis. This amount represents at least 5,000 contacts who received protective doses from the physicians. It should be borne in mind, however, that the use of GG is not indicated once symptoms have developed, since it has no therapeutic value.

To prevent any possible spread by stylets, needles, or syringes, they should be thoroughly sterlized by heat. Syringes may become contaminated by traces of blood drawn into them in giving intramuscular or intravenous injections.

Infectious hepatitis is a reportable disease. Cases should be reported to the local health officer and the State Department of Health.

J. W. Bower, M.P.H.
Director, Departmental Administration
Minnesota Department of Health

MINNESOTA STATE MEDICAL ASSOCIATION

House of Delegates—Summary of Proceedings*

Duluth Session—June 6 and 7, 1954

First Meeting, Sunday, June 6, 1954

Ballroom, Hotel Duluth
Duluth, Minnesota

The 101st Annual Session of the House of Delegates of the Minnesota State Medical Association opened in the Ballroom of the Hotel Duluth, Duluth, Minnesota, at 2:15 p.m., Sunday, June 6, 1954, Dr. C. G. Sheppard, Speaker of the House of Delegates, presiding.

After an invocation by Reverend Arthur Young, pastor of the Lakeside Presbyterian Church of Duluth, "freshman" delegates, serving for the first time were asked to identify themselves, Dr. R. G. Arveson of Frederic, Wisconsin, chairman of the Council, State Medical Society of Wisconsin, was introduced, and minutes of the 1953 meeting were accepted, a quorum being present.

Dr. Sheppard then introduced Dr. Dale Cameron, new medical director of the Minnesota Department of Public Welfare who spoke briefly on medical problems of the Department of Public Welfare, stressing particularly the state program for the mentally ill and asking for the close co-operation of physicians of the state in solving them.

After assuring Dr. Cameron of the full co-operation of the medical association, Dr. Sheppard called on Dr. A. O. Swenson of Duluth, chairman of the Council, to report on Council meetings held Saturday afternoon and evening, June 5, and Sunday morning, June 6, 1954, at the Hotel Duluth.

Report of The Council

Dr. Swenson: The Council first considered the report of Dr. J. M. Stickney, chairman of the Finance Committee (see following) and approved sixteen applications for life membership in the association; twenty-four to residency membership; twelve to service membership; one to junior associate membership and five to associate membership. In view of some differences of opinion as to the Physicians Assistance Fund, a special committee to study the fund, the need for it and methods for using it, was appointed by the Council and the Speaker of the House. The report of that committee included a recommendation that the program be continued by means of compulsory assessments when needed; that candidates for aid be recommended first by the county medical society for investigation by a member of the state office staff; that it should be operated and designed to keep physicians off the indigent rolls and that no use be made of welfare aids for the physician or his wife or widow; that the maximum amount of aid be set at $125 a month for the single individual and $175 for the physician and his wife, the awards to be reviewed at six-month intervals. The Council recommended the permanent committee of five be enlarged to eighteen, with two members from each councilor district, that the work of the committee should continue to further clarify the undertaking, with no recommendation for immediate addition of funds, and with commendation to the committee for the fine work it has done to date.

Other actions included rejection of a request for a contribution to the Minnesota Indian Scholarship Committee, with the recommendation that the committee be informed of the Association's rural medical scholarship which is available to Indian as well as white medical students who qualify; approval of financial aid for one delegate to the Student American Medical Association from Minnesota and for sending Minnesota Medicine to all members of the Junior and Senior classes at the University of Minnesota medical school; a recommendation that present methods of solicitation for the American Medical Education Foundation Fund be continued and that no compulsory assessment be levied for the purpose; rejection of the proposed camp for diabetic children because of legal and financial complications; postponement of the transfer of the Mower County Medical Society from the first to the second councilor district, pending further developments; reduction of the number of members on the Editing and Publishing Committee with the editor to be an ex-officio member, this in view of the fact that the newly functioning Board of Associate Editors provides good representation for the state and with the understanding that the chairman of the committee will also be an ex-officio member of the board and with the further suggestion that publication of historical data be restricted to four issues annually with the dates to be set by the editor, so as to provide additional pages for publication of scientific matter in accordance with the request of the board.

The Council also considered the AMA questionnaire on fee-splitting and decided to send delegates to the AMA uninstructed on the matter except for a general expression that any change in the Code of Ethics should protect the public from evils of fee-splitting, but at the same time provide workable interprofessional relations in the care of the patient.

The Council also instructed the delegates to oppose a resolution of the California Medical Association which would declare osteopathy no longer a cult and permit physicians to teach in schools of osteopathy not previously investigated or approved by the Council on accreditation and advised waiting for final draft of AMA simplified insurance report forms before taking any separate action on such forms. It approved the television program called "How's Your Health" developed for the Association by Dr. James Rogers Fox and commended Dr. Fox for his work.

Two other problems were considered at length. The first was the cancellation of the Veterans Medical Service contract with the Association due to, as explained to the Council by Dr. Donald R. McCarthy, a decrease in the amount of service requested with resultant rise in cost of administration. Two possibilities were offered for future handling of the service. One called for a grievance committee to represent members of the Association in dealing with the Veterans Administration and would require a new contract. The other provided that about 2,000 Minnesota physicians, to be called "designated physicians," would negotiate with the Veterans Administration for care of veterans on an individual basis. These physicians would, in that case, deal directly with the VA as to care and reimbursement. It was agreed that the Council would sign no further contracts with the Veterans Administration, inasmuch as the Association will have no control over rules or regulations, and that the Veterans Administration should be allowed to negotiate with individual physicians, though the Association will give unofficial aid wherever possible to expedite home-town care for disabled veterans.

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*In accordance with action by the Council of the Minnesota State Medical Association taken in 1944, publication of the proceedings of the House of Delegates is limited to summary.

The other problem concerned malpractice insurance, and the Council reviewed at length the report of Dr. A. E. Ritt, chairman of a special committee to study the malpractice insurance situation, which showed that there has been a sharp rise in costs, that rates are higher in Minnesota than any other state except California and that they are equalled by only one state, Oregon; that most insurance companies are refusing to write new policies or renew old ones unless collateral insurance is purchased also and that very few, if any, insurance companies are interested in developing a commercial group plan for malpractice insurance. It was further reported that only fourteen out of seventy-three cases handled by one insurance company over the period studied had availed themselves of the services of the Association's Medical Advisory Committee. The special committee was instructed to continue its studies and the Council urged all members threatened with malpractice suits to inform the Medical Advisory Committee promptly, declaring that full utilization of the services of the Medical Advisory Committee could be expected to help correct the unfortunate situation in which physicians of Minnesota currently find themselves in relation to malpractice insurance.

Finally, the Council appointed a committee to revise and clarify certain ambiguities in the constitution and by-laws, reaffirmed its previous stand as presented in the report of Dr. R. H. Creighton, chairman of the Committee on Veterans Medical Service, on medical service for veterans with non-service-connected disabilities and instructed delegates to act in conformity with it at the AMA House of Delegates meeting in San Francisco. The Council nominated Dr. Edward E. Novak of New Prague as Minnesota's choice for the AMA Doctor of the Year award.

Subsequent discussion underlined the fact that no further action was proposed for the present on recommendations by the special committee to study the Physicians Assistance program, but that the committee had been instructed to work out details more completely and report at a later date. Also, the fact that the grave malpractice situation in Minnesota is not due to a higher average of cases in Minnesota was emphasized. The trouble lies in the large verdicts allowed by judges and juries in Minnesota, it was pointed out, damages awarded being out of line with those of other states.

* * *

The report was unanimously accepted by the delegates, and Dr. Sheppard called for the report of Dr. J. Minott Stickney, Chairman of the Finance Committee.

Report of the Finance Committee

Dr. Stickney said that the permanent investment fund of the Association, now $104,863.18, has not yet reached an amount equal to one year's budget and should therefore not be drawn on for any purpose. No additional money, other than interest, has been added to the fund since 1946. He also pointed out that operating expenses of the Association had increased in comparison with 1953, largely because of increased committee expense. Income for 1953 exceeded expenses by $1,323.03, but he warned that the budget for 1955 will have to provide a larger budget for the Committee on Public Policy. He also pointed out that termination of the contract for Veterans Medical Service with the Veterans Administration may result in a net loss to the Association. The loss of $550 income from Minnesota Medicine in 1953, as compared with 1952, was accounted for by reason of increased expense in all departments of publication except editorial expense. Cost of the journal to the Association amounts to approximately $1.54 of each member's dues and will increase somewhat when the journal is provided, as directed by the Council, to an estimated 200 junior and senior medical students.

* * *

It was moved, seconded and carried that Dr. Stickney's report be accepted and Dr. Sheppard then called for reports of the reference committees. The first was by Dr. C. W. Moberg, Detroit Lakes, Chairman of the Reference Committee on Medical Education Reports.

Medical Education Reports

Dr. Moberg: The committe considered the following reports:

Committee on Cancer—David P. Anderson, Jr., M.D., Austin, Chairman
Committee on Conservation of Hearing—K. M. Simonton, M.D., Rochester, Chairman
Committee on First Aid and Red Cross—John S. Lundy, M.D., Rochester, Chairman
Heart Committee—F. J. Hirschboeck, M.D., Duluth, Chairman
Committee on Hospitals and Medical Education—H. S. Diehl, M.D., Minneapolis, Chairman
Committee on Public Health Nursing—M. McC. Fischer, M.D., Duluth, Chairman
Committee on Syphilis and Social Diseases—H. G. Irvine, M.D., Minneapolis, Chairman
Committee on Tuberculosis—J. F. Briggs, M.D., St. Paul, Chairman
Committee on Vaccination and Immunization—R. N. Barr, M.D., Minneapolis, Chairman

All the above reports were considered and commended as well-written, concise and to the point, with the following special observations; that first-aid training, as outlined in the report of the Committee on First Aid and Red Cross, be developed in the public school system; that attention should be called to the increase in the number of tuberculosis deaths among people over forty-five in comparison with earlier ages; that the committee is in agreement with the policy of the Committee on Vaccination and Immunization with respect to delaying approval of polio vaccination in Minnesota; that special commendation should be given to the University of Minnesota as indicated in the Heart Committee report, for its outstanding accomplishments in the field of heart surgery; that Minnesota now has fourteen physicians per 10,000 population, as indicated in the report of the Committee on Hospitals and Medical Education which is a high ratio in comparison with other parts of the country and that the 12-month courses arranged by the University for training laboratory aides and technicians be continued; that Dr. H. G. Irvine and his department of the State Department of Health be commended especially for their excellent work in the control of syphilis and social diseases and that all practicing physicians should be reminded, in accordance with the recommendation in the excellent report of the Committee on Tuberculosis, to report all active cases of tuberculosis to the department of health.

At the request of Dr. Moberg, Dr. K. M. Simonton, Chairman of the Committee on Conservation of Hearing, commented on the work of his committee as follows:

He called attention of the delegates to the exhibit arranged by the committee and on view at the Armory. Also he told them about the circular on hearing problems both in therapy and rehabilitation, which is to go to all physicians of the state and about the letter to parents of hearing-handicapped children, suggesting sources of treatment and directing them to consult their physicians. This letter is to go to all parents known to have such children in the state.

* * *

The House of Delegates unanimously accepted Dr. Moberg's report and the Speaker called for the report of the Reference Committee on Medical Economics Reports, Dr. L. E. Steiner of Albert Lea, Chairman.

Medical Economics Reports

Dr. Steiner: The following reports were considered:

Editing and Publishing Committee—E. M. Hammes, Sr., M.D., St. Paul, Chairman
Committee on Hospitals and Professional Relations—J. Minott Stickney, M.D., Rochester, Chairman
Committee on Interprofessional Relations—W. P. Gardner, M.D., St. Paul, Chairman
Medical Advisory Committee—W. H. Hengstler, M.D., St. Paul, Chairman
Committee on Medical Economics—George Earl, M.D., St. Paul, Chairman
Committee on University Relations—R. L. J. Kennedy, M.D., Rochester, Chairman

All reports were approved with the following comments: the sub-committee on the Rural Medical Scholarship should be commended for its diligence in selection of scholarship recipients. The work of the Committee on University Relations, and the Committee on Interprofessional Relations were both commended for helping to bring about the 12-month course for laboratory aides and x-ray technicians at the University; also the reference committee suggests that the Committee on Interprofessional Relations should advise all members on the status and ethical issues involved in advertising by pharmacists on prescription blanks. The reference committee commended appointment of Dr. A. H. Wells of Duluth as editor of Minnesota Medicine and the appointment of an editorial board to assist Dr. Wells and provide more active participation by members in direction of the journal. The committee likewise urged co-operation on the part of all members threatened with malpractice suits with the Medical Advisory Committee.

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The delegates accepted the report and the Speaker called for the report of the Reference Committee on Miscellaneous Scientific Reports, Dr. Donald McCarthy of Minneapolis, Chairman.

Miscellaneous Scientific Reports

Dr. McCarthy: The following reports were reviewed:

Committee on Anesthesiology—R. C. Adams, M.D., Rochester, Chairman
Committee on Blood and Blood Banks—John S. Lundy, M.D., Rochester, Chairman
Committee on Child Health—G. B. Logan, M.D., Rochester, Chairman
Committee on Chronic Illness—E. L. Tuohy, M.D., Duluth, Chairman
Editorial Committee—A. H. Wells, M.D., Duluth, Chairman
Committee on Diabetes—J. R. Meade, M.D., St. Paul, Chairman
Committee on Fractures—E. T. Evans, M.D., Minneapolis, Chairman
Committee on General Practice—James A. Blake, M.D., Hopkins, Chairman
Historical Committee—Robert Rosenthal, M.D., St. Paul, Chairman
Committee on Industrial Health—L. S. Arling, M.D., Minneapolis, Chairman
Committee on Maternal Health—J. J. Swendson, M.D., St. Paul, Chairman
Committee on Medical Testimony—E. M. Hammes, Sr., M.D., St. Paul, Chairman
Committee on Military Affairs—J. H. Tillisch, M.D., Rochester, Chairman
Committee on Nervous and Mental Diseases—W. P. Gardner, M.D., St. Paul, Chairman
Committee on Ophthalmology—T. R. Fritsche, M.D., New Ulm, Chairman
Committee on Public Health Education—F. J. Elias, M.D., Duluth, Chairman
Radio and Television Committee—R. M. Burns, M.D., St. Paul, Chairman
Speakers' Bureau—H. M. Carryer, M.D., Rochester, Chairman

All the above reports were approved with the following comments: That Dr. R. C. Knutson of St. Paul be asked to present the substance of his report on the relationship of anesthesiologists to Blue Shield, mentioned in the report of the Committee on Anesthesiology, before the House of Delegates; that the Committee on Blood and Blood Banks be complimented for having secured a definite ruling from the Attorney General to the effect that osteopaths are not permitted by law to give blood transfusions; that the Committee on Child Health is to be commended for its close and continuous co-operation with health, welfare and professional organizations to advance child health and particularly for its assistance in preparation of the booklet on first aid for children, which is to be distributed to parents all over the state, also for its co-operation with the Minnesota dietetics society in a projected booklet on nutrition in the prenatal period and infancy and its participation, through its chairman, in the advisory committee on poliomyelitis vaccination. The committee approved and seconded a request of the Committee on Chronic Illness for suggestions on management of problems associated with chronic illness. It also suggested, in accordance with the recommendation of the Fracture Committee, that a fracture symposium be an annual feature of state meeting programs that the program of the Committee on Maternal Health be continued and that time be set aside at the next annual meeting for a report on findings of the maternal mortality sub-committee; that the emergency medical care phase of the Civil Defense program in which the Committee on Military Affairs is co-operating, be approved and that new health department and welfare department services for the aging, as well as for mental health, in general, be approved and that the State Advisory Board on Problems of Alcoholism, in which the Committee on Nervous and Mental Diseases is co-operating, have the doctors' support. The committee also noted the appeal of the Committee on Ophthalmology for members to earmark contributions to the Greater University Fund for an Eye Pathology Laboratory which is greatly needed and declared that the Committee on Public Health Education has made an outstanding contribution to the Association, a contribution in which the Radio and Television Committee and the Speakers' Bureau have played an important part which also should be continued. Finally, the reference committee approved a special report just handed to it by the Committee on Tuberculosis and on its policy with respect to chemotherapy outside the hospital and recommended that it be widely published through Minnesota Medicine and all other media for reaching doctors and nurses of the state.

* * *

The delegates unanimously approved the report and the Speaker called on Dr. E. M. Hammes, Sr., Chairman of the Committee on Medical Testimony, for discussion (Dr. Knutson not being present at this time).

Report of Committee on Medical Testimony

Dr. Hammes re-emphasized the necessity of submitting complaints in writing if the committee is to make investigation of unethical medical testimony. He said that only verbal complaints had been made by physicians and that the program of the committee which has had nation-wide attention is endangered because the committee can consider only written complaints and the only admissible complaints have come from a judge and three attorneys.

* * *

Speaker Sheppard then called for the report of the Reference Committee on Miscellaneous Medical Economics Reports, Dr. M. J.. Anderson of Rochester, Chairman.

Miscellaneous Medical Economics Reports

Dr. Anderson: The Reference Committee on Miscellaneous Medical Economics Reports considered reports of the following committees:

Insurance Liaison Committee—Clarence Jacobson, M.D., Chisholm, Chairman

Committee on Medical Service—J. A. Bargen, M.D., Rochester, Chairman

Committee on Rural Medical Service—Paul C. Leck, M.D., Austin, Chairman

Committee on State Health Relations—C. E. Proshek, M.D., Minneapolis, Chairman

Committee on Veterans Medical Service—R. H. Creighton, M.D., Minneapolis, Chairman

Committee on Medical Ethics—Harry Klein, M.D., Duluth, Chairman

Membership Committee—H. F. R. Plass, M.D., Minneapolis, Chairman *(For a supplement to the Membership Committee report Dr. Anderson called on Dr. Plass before presenting the reference committee report)*

Dr. Plass: We want to urge all county societies to make a ceremony of inducting new members into their societies and hope that the delegates will carry the message back to their societies. Also, we wish to report that the plan for a new members' dinner had to be abandoned because of lack of interest at this meeting. We believe newness of the idea and too little work on it at home were responsible. With more work to get the men interested, we hope to have such a dinner next year.

Dr. Anderson: The reference committee approved all the reports with these additional comments: that the Veterans Medical Service committee be commended for its work; that a vote of thanks be given to the Committee on Medical Service for arriving at a solution of the problems involved in providing medical service under the Minnesota High School Athletic League policy, this solution calling for a perforated attachment to the policy which states that the policy is not a service, but an indemnity policy which the parent must sign and return to the high school authorities before high school athletes can participate; that the Committee on State Health Relations be urged to continue its effort to secure a medical examiner in place of the coroner system in Minnesota; that the Membership Committee be commended for the originality of its report and that it be noted that neither the Committee on Rural Medical Service nor the Committee on Medical Ethics had found it necessary to meet during the year; that the Insurance Liaison Committee is still engaged in a study of insurance policies and has not yet arrived at a definite recommendation.

Before moving acceptance of the report Dr. Anderson called on Dr. Bargen who reported that Mr. H. R. Peterson, Executive Secretary of the Minnesota High School League, has proved most co-operative in the new plan, having followed all suggestions of the committee including a suggestion that all questions involving medical matters be referred to Dr. W. E. Proffitt of Minneapolis who serves as medical director of the League without pay. Many matters have been referred to Dr. Proffitt of late with the result that the program is now working well and Dr. Proffitt should be especially commended for his service.

* * *

Dr. Anderson's report having been approved, Speaker Sheppard asked for the report of the Reference Committee on Officers and Councilors Reports, Dr. A. E. Ritt of St. Paul, Chairman.

Officers and Councilors Reports

Dr. Ritt: Reports of the Secretary and Executive Secretary, Treasurer, Chairman of the Council and Councilors of Districts 1 through 9 were considered by the reference committee. All were approved and commended with the following comments: that an excellent job continues to be done by the secretary and executive secretary and that the work of the field secretary, under the direction of the executive secretary, has proved very helpful to committees and local societies throughout the state; that no additions or modifications could be suggested for the reports of the treasurer, chairman or members of the Council.

* * *

Dr. Ritt's report was unanimously approved and the meeting recessed at 4:30 p.m. until 8:15 p.m. when Speaker Sheppard called the meeting to order for the evening session.

Second Meeting, Sunday, June 6, 1954 (8:15 p.m.)

At the request of the Speaker, Dr. Herman Drill of Hopkins, Minnesota, Chairman for the American Medical Education Fund, called attention of the delegates to the fact that more contributors to the fund are needed from Minnesota; also that the total contribution from Minnesota since 1951 was $26,279.00 but that the University has received a total of $66,956 for the same period. Dr. Drill introduced Mr. Hiram Jones of Chicago, Executive Secretary of the American Medical Education Foundation.

AMEF Fund

Mr. Jones: The American Medical Education Foundation is a sponsored project of the American Medical Association, receiving its operating funds from the Association and it is thus enabled to turn over the entire proceeds of its fund raising activities for medical education to the National Foundation for Medical Education, a lay body organized to receive and distribute these funds and to raise funds for the same purpose from business and industry. There are seventy-nine medical schools in the country upon whom we rely to provide at least 6,000 medical graduates a year. Local funds upon which the parent University depends, are not sufficient to cover costs of medical education of current high standards and the deficit must be made up either by private contribution or by the federal government. It is estimated that ten million dollars a year contributed for the purpose for a period of years will effectively prevent federal subsidies and maintain the medical schools. A contribution of $25 a year from every physician would achieve it, plus gifts of lay groups and industry. The latter obviously cannot be expected to place their gift dollars in this fund unless physicians themselves have made substantial contributions. About four and three quarter millions have been contributed so far, less by a good deal than the goal set, but growing each year. So far this year more than 11,000 contributors have given $786,000. Funds are given to the schools without restrictions since most funds received by the schools are dedicated to special purposes which allow for no shifting and cannot be used for necessary cost-of-living increases in salaries and other similar urgent needs. With your help we can get the money we need. With failure will come federal subsidy.

At the request of the Speaker, Dr. A. J. Chesley, Secretary of the Minnesota State Board of Health, first mentioned the need for a separate building on the University Campus devoted exclusively to the State Board of Health activities and including all departments now housed separately in St. Paul. He then took occasion to express his appreciation for the cooperation of the medical profession in the work of the board and asked the delegates to hear Dr. Helen L. Knudsen, Chief of the Division of Hospital Services of the board.

Dr. Knudsen reviewed the Hill-Burton hospital building program in Minnesota to date, reporting that from 1947 to 1952 a total of 121 hospitals, nursing homes and homes for the aged, providing 7,900 additional beds have been built in Minnesota at an estimated total cost of more than 73 million dollars. Of this amount 58.1 per cent came from local funds, 25 per cent from state funds and 16.8 per cent came from federal funds. The objective has been to see that regional hospital centers with adequate equipment and personnel are built or planned for all sections of the state at the same time that plans are worked out for additional beds and services at the University, especially in the new Mayo Memorial Medical Center, for use of the entire state. For staffing new facilities and raising standards of services, the department and the University, with cooperation of the physicians and the hospital association, has set up new short refresher courses held out in the state and new short training courses of two years or less for x-ray technicians, laboratory aides, medical record librarians and others held at the University. Dr. Knudsen emphasized the great need all over the country for additional beds for the aging and chronically ill, including rehabilitation facilities as well as long-term care, and praised the program now in progress in St. Louis county which is unique in the entire country and now provides 5.09 beds for this service per thousand population.

Dr. R. N. Barr, Director of the Division of Local Health Services, was also called on at Dr. Chesley's suggestion and reported that the entire program of the department of health is being re-evaluated and that communicable diseases, once so important, are being displaced as major problems by accidents, chronic diseases, care of the aged and the saving of infant lives in the first two or three days of life. In spite of tremendous progress in cutting death rates among children, no progress in saving lives in the first three days of life has been made in the last 40 years, he said. Dr. Barr also reported that there is less polio in the state than last year, that a very small percentage of children would have been offered vaccine if the state had approved the Salk vaccination program in any case. The program in many other states got underway so late that approval was withdrawn and for that reason it is likely to be impossible to study results of ten thousand vaccinations, this year, which the board with approval of the Council of the Minnesota State Medical Association, required before approving the program for this state. No protest has come in Minnesota as a result of the board's action.

* * *

Speaker Sheppard then called on Dr. J. A. Bargen of Rochester, substituting for Dr. E. M. Jones, Secretary, who is ill, to report for the State Board of Medical Examiners.

State Board of Medical Examiners

Dr. Bargen reviewed the number of physicians, osteopaths, and others licensed in Minnesota and particularly the number of foreign graduate physicians licensed since the current program for foreign physicians began in 1949. Regulations for licensing foreign graduates as revised in 1949, call for admission on an individual basis provided credentials can be verified, plus a year of training in an approved hospital (increased to two years in May, 1954) and a requirement that these licensees obtain citizenship within two years after they are licensed. Under this program, fifty-three have been licensed but only eight are practicing in rural districts of the state in spite of the fact that all were asked when they appeared before the board if they were willing to practice in rural districts and all said yes.

Dr. H. R. Tregilgas of St. Paul reported on the work of the Minnesota State Board of Basic Sciences and outlined briefly methods used by the examiners for drawing up examination questions, methods used by some candidates for achieving passing grades in the examinations and the hope of the Board for new legislation to raise standards and require at least two years of college for admittance to a basic science examination. Also he reviewed reciprocity regulations and rulings. Reciprocity with other states is now limited and partial, he said, and is likely to be reduced still further.

* * *

Speaker Sheppard called on Dr. F. F. Callahan of St. Paul, Director of the Tuberculosis Control Section of the Division of Public Welfare, for a report from his section.

Report of Tuberculosis Control Section

Dr. Callahan outlined the change in treatment of tuberculosis as a result of chemotherapy and lung surgery which has reduced the need for long-term sanatorium care and resulted in closing of the Preventorium in St. Paul and conversion of four sanatoria in the northern section of the state to nursing homes for the aged. The new tuberculosis treatment calls for modern hospital facilities rather than custodial care, he said, and reduces length of hospital or sanatorium stay from several years to a few months if cases are discovered early. Cost of treatment per day is greater but the total cost for the patient is greatly reduced and many more are returned to become self-supporting, tax-paying citizens. He advocated a larger allowance per day by the state so that all can have modern effective treatment. The biggest current problems so far as undiscovered infections is concerned, lie in the jails among prisoners, Mexican laborers and Indians. The effort of the federal government to accomplish absorption of Indians into the general population has resulted in directives which complicate the care of tuberculosis patients among them. The answer is larger state appropriations for all tuberculosis care.

Dr. R. H. Wilson of Winona, Chairman of the State-Wide Medical Advisory Committee of the Department of Social Welfare, reported that one of the most difficult problems involved in maintaining and defending the right of the welfare patient to free choice of physician is concerned with repeated, expensive diagnostic studies requested by the patient. He cited, among other examples, the case of a woman with heart trouble who had received adequate diagnostic studies and hospitalization near her home but for whom the same procedure was unnecessarily repeated twice in larger, more expensive centers on visits to daughters who lived in these two different centers, with a cost to the welfare department that ran to more than $3,000. Some way will have to be found, he said, to avoid that kind of expenditure. Also he noted that some doctors should be cautioned against running up unnecessary bills for welfare patients. A uniform fee schedule for use of Welfare Boards throughout the state is being considered. When it is in final form it will be submitted to all the component medical societies as well as the welfare boards for their approval, Dr. Wilson said.

* * *

Speaker Sheppard then called for the Necrology Report from Dr. Richard Bardon of Duluth, substituting for Dr. Robert Rosenthal of St. Paul, Chairman of the Historical Committee.

A moment of silence was observed after the reading of Dr. Bardon's report and then the Speaker called for the report of the Resolutions Committee, Dr. P. E. Hermanson of Hendricks, Chairman.

Report of Resolutions Committee

Dr. Hermanson first read four resolutions, two of which were tabled. The first one, offered by Dr. James Blake as chairman of the delegation from the Sixth Councilor District, commended the University of Minnesota for developing a one-year course for training of laboratory aides and proposing that the Minnesota State Medical Association recommended creation of a board with authority to accredit laboratory aides trained at the University or other Minnesota training schools whose curriculum meets standards set by the board. This resolution was adopted.

The second introduced by the Rice County Medical Society recommended that courses in the techniques of cancer detection be given in various sections of the state in order to bring these techniques to the general practitioner, the cost to be defrayed by the Minnesota Division of the American Cancer Society. This resolution was recommended by the committee for adoption, to be referred first to the Cancer Committee for consideration and then to the Council for immediate action. The recommendation was unanimously adopted.

The third resolution of this group, from the Renville-Redwood County Medical Society, called for a change in the Veterans Administration Act by which medical and hospital care by the Administration would be provided only for veterans with service-connected disabilities. After some discussion, the delegates decided unanimously to table this resolution, and another resolution from the same society, asking that candidates for licensure to practice medicine and surgery be limited to graduates of Class "A" medical schools, was tabled also because of confusion on the part of several delegates as to its meaning.

Remaining resolutions reported at this meeting had to do with the Blue Shield's Plan "B" contract and were offered either as statements of policy or resolutions, both for and against, from several societies including the Renville-Redwood County Medical Society, the Clay-Becker County Medical Society, the Red River Valley Medical Society and the Hennepin County Medical Society. The Resolutions Committee, after extended discussion and deliberation on the matter, offered a substitute for these resolutions, reported by Dr. Hermanson as follows:

WHEREAS Plan "B" of the Minnesota Medical Service has received so much discussion both pro and con, and

WHEREAS there is a large segment of the Minnesota State Medical Association that is now opposed to the existing plan of Blue Shield, and

WHEREAS there is also a large segment of the membership that is in favor of Plan "B," and

WHEREAS there are contracts now in effect which must be honored, and

WHEREAS the present Plan "B" now is such a controversial subject,

BE IT HEREBY RESOLVED that Plan "B" as it now exists be honored to the termination of the present contracts, but in the interim we highly recommend that the Blue Shield Board meet with the opposing societies and that an attempt be made to iron out the differences as to

1. Top income of the service policy.
2. Definition of pay as to specialty groups, and further that
3. If any major change is made in income levels in service contracts,

BE IT RESOLVED that such contemplated change be presented to each member of the Minnesota State Medical Association for their information before adoption and approval by our State Medical Society.

* * *

Dr. Hermanson moved adoption of this resolution. His motion was seconded and Dr. James Blake, Hopkins, proposed an amendment to the effect that all further sales of Plan "B" contracts be stopped until the meetings suggested in the resolution have been held and the difficulties have been ironed out.

Speaker Sheppard then called for discussion, and a large number of delegates from all parts of the state discussed the entire Plan "B" contract fully, with all who wished to speak in favor or in opposition to the resolution being allowed unlimited time to address the delegates. When there were no further requests for the floor, Speaker Sheppard called first for a vote on a proposal made in the course of his remarks by Dr. C. W. Moberg of Detroit Lakes to the effect that the voting on Plan "B" be conducted by standing vote. The motion was lost and the Speaker called for a vote by ballot on the amendment offered by Dr. Blake, the object of this amendment being to make it explicit that all further sales of Plan "B" contracts should be discontinued. Members of the Credentials Committee were appointed tellers and delegates were instructed to come forward personally and deposit their ballots in the box as their names were called.

The amendment to the resolution was approved by a vote of 42 to 36, and the Speaker asked for a ballot to be cast by the same method on the substitute resolution proposed by the Resolutions Committee and including the amendment just voted by the delegates. He explained that a "no" vote on this resolution would leave Blue Shield free to carry forward plans for selling Plan "B" contracts, while a "yes" vote would be a request to the Blue Shield Board to cease selling Plan "B" contracts until difficulties could be ironed out in a series of meetings to be held for the purpose.

The motion for this resolution was lost by a vote of 41 to 37. Dr. James A. Blake of Hopkins then moved that the delegates be permitted to vote on the original resolution presented by the Hennepin County Medical Society to the Resolutions Committee.

After two motions were rejected by voice vote, one to table Dr. Blake's motion until the following day and the other to vote on it by voice vote, the Speaker informed the delegates that a motion to bring the resolution out of committee to the floor of the House would have to be made and duly voted before the resolution could be considered.

The necessary motion was accordingly made and seconded and the delegates voted 39 to 38 by ballot to bring the Hennepin County resolution, which was read as follows, to the floor:

WHEREAS the recent introduction by the Minnesota Medical Service of the service contract designated as Plan "B" is opposed by many physicians as representing a radical departure from the original philosophies and basic concepts which were accepted when the Blue Shield Plan was promulgated; and

WHEREAS a large number of physicians now participating in Blue Shield have expressed their intentions to withdraw from such participation if Plan "B" is continued which will definitely limit the free choice of physicians by patients holding Blue Shield contracts; and

WHEREAS while the Minnesota Medical Service is a completely separate organization having no responsibility to the Minnesota State Medical Association, and has in the past requested approval of various phases of its plans of operation by the Minnesota State Medical Association, and as it is the considered opinion of the House of Delegates of the Minnesota State Medical Association that the best interests of medicine will be served by the fullest possible understanding and cooperation between the State Medical Association and the Minnesota Medical Service;

BE IT RESOLVED THEREFORE that the House of Delegates rescind its previous action approving Plan "B" and that it recommends to the Minnesota Medical Service—

1. That Minnesota Medical Service discontinue any further sale of Plan "B" contracts.
2. That Minnesota Medical Service not renew such Plan "B" contracts as are now in effect after each such contract expires.
3. That Minnesota Medical Service limit future contracts for medical service to such income bracket groups as may be approved by the House of Delegates of the Minnesota State Medical Association.

Another motion to adjourn until later in the day was lost by a showing of hands, and it was then moved and seconded that the delegates adopt the Hennepin County resolution.

The Speaker called for a vote by ballot, emphasizing the fact that a "yes" vote would mean the effective end of Plan "B," while a "no" vote would mean approval of Plan "B." Several substitutes for members of various society delegations who had left the floor were accepted by the delegates before the voting. The final vote by ballot was 52 to 26 against adoption of the resolution. The meeting then adjourned at 2:15 a.m. until Monday, June 7 at 1:15 p.m.

Third Meeting, Monday, June 7, 1954 (1:15 p.m.)

The House of Delegates reconvened at 1:15 p.m. in the Ballroom, Hotel Duluth, Duluth, Minnesota, Speaker Sheppard presiding.

Dr. C. A. Anderson of Hector asked the delegates to reconsider the resolution on licensure tabled at the previous session and, after a better explanation of its purpose, to vote to suspend the rules and admit the resolution to the floor of the House for action. It was explained that the purpose of the resolution which was introduced by the Renville-Redwood County Medical Society but concurred in by other societies was to protect the people of Minnesota against attempts by cultists to liberalize the law and permit them to practice medicine and surgery in spite of the fact that they are not graduates of approved schools.

After considerable discussion, a slightly amended resolution was prepared, the rules were duly suspended, and the delegates unanimously adopted the following resolution:

WHEREAS for many years applicants for licensure to practice medicine and surgery in the State of Minnesota have been required to be graduates of Class A medical schools, and

WHEREAS such requirements are in the best interests of the people of the State of Minnesota, and

WHEREAS attempts are being made to amend the laws of the state to permit graduates of schools other than schools of medicine and surgery that have not been inspected and found to be Class A medical schools to practice medicine and surgery in all of its branches in the State of Minnesota, and

WHEREAS in our opinion, such proposals are not in furtherance of the health and welfare of the people of the state, therefore be it

RESOLVED that we go on record as being opposed to such legislative proposals and be it further

RESOLVED that we support the efforts that are being made to require all applicants for licensure to practice medicine and surgery in the State of Minnesota to be graduates of Class A schools of medicine and surgery, and be it further

RESOLVED that a copy of this resolution be sent to each member of the Legislature of this state.

Resolutions of thanks to the St. Louis County Medical Society, the newspapers and press services of the state, radio and television stations, the Duluth hotels and the Duluth Chamber of Commerce for their respective contributions to the success of the meeting were proposed by Dr. P. E. Hermanson of Hendricks, Chairman of the Resolutions Committee and unanimously adopted by the delegates.

Dr. James A. Blake of Hopkins then proposed adoption of a resolution from the Hennepin County Medical Society as follows:

RESOLVED: That this House of Delegates express to the Blue Shield Board of Directors its sincere thanks for their efforts on behalf of the doctors of Minnesota, and

BE IT FURTHER RESOLVED that the House of Delegates give the Blue Shield Board its vote of confidence and express to the Board its assurance that all elements of the profession in the state will work with the

Board in the hope of developing a Blue Shield policy which will overcome some fundamental objections expressed at this session and which will be mutually satisfactory to the Board and to participating doctors.

The resolution was unanimously adopted, and Speaker Sheppard asked for the report of the Nominating Committee, Dr. Willard D. White of Minneapolis, Chairman.

Report of Nominating Committee

Following recommendations made by Dr. White, these officers and councilors were unanimously elected by the delegates:

President-Elect—A. O. Swenson, M.D., Duluth
First Vice President—F. W. Behmler, M.D., Morris
Second Vice President—Karl E. Johnson, M.D., Duluth
Secretary—B. B. Souster, M.D., St. Paul
Treasurer—W. H. Condit, M.D., Minneapolis
Speaker of the House of Delegates—C. G. Sheppard, M.D., Hutchinson
Vice Speaker of the House of Delegates—H. M. Carryer, M.D., Rochester
Councilor, Fourth District—H. J. Nilson, M.D., North Mankato
Councilor, Sixth District—H. B. Sweetser, M.D., Minneapolis
Councilor, Eighth District—C. L. Oppegaard, M.D., Crookston

Inasmuch as Dr. A. O. Swenson was councilor of the Ninth District until elected president-elect at this session, no recommendations were made for his successor by the Nominating Committee and Speaker Sheppard called for nominations from the floor.

Dr. Paul Boman of Duluth then nominated Dr. Clarence Jacobson of Chisholm and Dr. Jacobson was unanimously elected councilor for the Ninth District to succeed Dr. Swenson.

The following nominees, whose names were submitted by the Council for delegates to the American Medical Association were also unanimously elected: F. J. Elias, M.D., Duluth; J. Arnold Bargen, M.D., Rochester; A. O. Swenson, M.D., Duluth, alternate to Dr. Elias, and Paul C. Leck, M.D., Austin, alternate to Dr. Bargen.

Minneapolis having been chosen in 1953 for the 1955 annual meeting at the special request of the Hennepin County Medical Society which will celebrate its 100th anniversary in that year, the Speaker asked for invitations for the 1956 meeting and the delegates voted unanimously to accept the invitation of the Olmsted-Houston-Fillmore-Dodge County Medical Society to hold the 1956 meeting in Rochester.

There being no further business to come before the meeting, the House of Delegates of the Minnesota State Medical Association adjourned its 101st annual session at 2:30 p.m., June 7, 1954.

CHRONIC ULCERATIVE COLITIS

(Continued from Page 663)

163. Strax, S., Marks, J. A., Wright, L. T.: Treatment of non-specific ulcerative colitis with aureomycin. Rev. Gastroent, 18:413-422, 1951.
164. Sullivan, J. M.: The surgical treatment of ulcerative colitis. Wisconsin M.J., 49:773-777, 1950.
165. Svartz, N.: The pathogenesis and treatment of ulcerative colitis. Acta. Med. Scand., 141:172-184, 1951.
166. Svartz, N.: The treatment of ulcerative colitis. Gastroenterology, 26:26-28, 1954.
167. Texter, E. C., Legerton, C. W., Jr., Ruffin, J. M., Boyd, J. W., Jr., Smith, A. G.: Idiopathic ulcerative colitis—giant ulceration following ACTH and cortisone therapy. A.M.A. Arch. Int. Med., 91:744-757, 1953.
168. Tumen, H. J., Cohn, E. M.: Pregnancy and chronic ulcerative colitis. Gastroenterology, 16:1-11, 1950.
169. Tumen, H. J., Monaghan, J. F., Jobb, E.: Hepatic cirrhosis as a complication of chronic ulcerative colitis. Ann. Int. Med., 26:542-553, 1947.
170. Turnbull, R. B.: Management of the ileostomy. Am. J. Surg., 86:617-624, 1953.
171. Turnbull, R. B., Brown, C. H.: Carcinoma of the colon in chronic ulcerative colitis. Cleveland Clin. Quart., 19:166-170, 1952.
172. Van Patter, W. N., Bargen, J. A., Dockerty, M. B., Feldman, W. H., Mayo, C. W., Waugh, J. M.: Regional enteritis. Gastroenterology, 26:347-447, 1954.
173. Warren, S., Sommers, S. D.: Pathogenesis of ulcerative colitis. Am. J. Path., 25:658-680, 1949.
174. Warren, S., Sommers, S. C.: Pathology of regional ileitis and ulcerative colitis. J.A.M.A., 154:189-193, 1954.
175. Webster's New International Dictionary, 2nd Edition. Springfield: G. C. Merriam Co., 1948.
176. Weckesser, E. C., Chinn, A. B.: Carcinoma of the colon complicating chronic ulcerative colitis. J.A.M.A., 152:905-908, 1953.
177. Weekly Clinicopathological Exercises: New England J. Med., 246:664-667, 1952.
178. Weekly Clinicopathological Exercises: New England J. Med., 247:531-533, 1952.
179. Weekly Clinicopathological Exercises: New England J. Med., 243:967-970, 1950.
180. Weinstein, A.: Ulcerative colitis. J. Tenn. State M. A., 46:357-366, 1953.
181. Welbourn, R. B.: Benign ulcers of the greater curvature associated with ulcerative colitis and cirrhosis of the liver. Brit. J. Surg., 39:303-308, 1952.
182. Wener, J., Hoff, H. E., Simon, M. A.: Production of gastric and duodenal ulcers by the prolonged administration of mecholyl. Gastroenterology, 11:904-922, 1948.
183. Whipple, R., Jr.: Procaine penicillin anaphylactic shock, acute ulcerative cystitis and acute ulcerative colitis, J.M.A., Georgia, 42:334-337, 1953.
184. Willard, J. H., Pessel, J. F., Hundly, J. W., Bockus, H. C.: Prognosis of ulcerative colitis. J.A.M.A., 111:2078-2086, 1938.
185. Wilson, C. C.: Medical management of chronic ulcerative colitis. South. M. J., 44:797-801, 1951.
186. Wilson, J. A.: Personal communication.
187. Wirts, C. W.: Nutrition in ulcerative colitis. Pennsylvania M. J., 55:1120-1121, 1952.
188. Wirts, C. W., Rehfuss, M. E., Yantes, H. A.: Long term results in corticotropin treated ulcerative colitis. J.A.M.A., 154:36-39, 1954.

Minneapolis Surgical Society

Meeting of March 4, 1954
President Robert Tenner, M.D., in the Chair

PRESENT STATUS OF SURGERY IN THE TREATMENT OF TOXIC GOITER

EDWARD S. JUDD, JR., M.D.
Rochester, Minnesota

It is fascinating to reflect on early attempts to control the complex and as yet not completely understood syndrome of hyperthyroidism. Although it seems a long time since iodine became well established as an element playing some mysterious role in this condition, many physicians practicing today recall the early efforts to bring about some change in this rather drastic upheaval of obscure metabolic relationships.

It is interesting to think back on the days when boiling water was injected directly into the thyroid gland in the hope that something might happen at least to stave off the dire circumstances. I witnessed the last such injection at the Mayo Clinic and I remember Dr. Pemberton commenting at the time that it was largely a psychotherapeutic procedure. The patient had counted on having an operation on that day; since the patient was not entirely ready for surgery, it was considered that something had to be done. Indeed, the temporary destruction or at least derangement of the gland may have altered the condition for a few days. Most surgeons can remember pole ligation, in which the superior artery on one or both sides was approached and gingerly tied off; if this were successful, the inferior artery underwent the same process later. This led to multiple-stage techniques in which one lobe of the gland was removed or resected; if the patient survived, the second one was attacked later. The wonder today is that patients who had extreme degrees of toxicity rallied enough stamina to put up with this long ordeal. However, this procedure was all that the surgeon had to offer and, since no one else had anything to offer, it was carried out for a number of years. The full development of the use of iodine as a preoperative agent led in logical fashion to the somewhat radical one-stage thyroidectomy as the treatment of choice.

In the intermediate years the surgeon enjoyed a position comparable to that of the supreme court, and when the diagnosis was established the only question that remained was "How soon can the patient be put into reasonable condition to withstand the operation?" It was granted that a certain percentage of these patients might not survive even the gentlest surgical maneuver.

Disadvantages of Surgical Treatment

Surgeons should be the first to acknowledge those drawbacks which are peculiar to surgical treatment only. The hospital mortality rate already has been mentioned and will be elaborated on later. Next is the problem of the recurrent laryngeal nerve. No surgeon, regardless of training, experience and the exercise of utmost caution, can surgically treat an extensive series of patients without some concern regarding these nerves. Considerable time and effort have been expended in the past on efforts to minimize the danger to them and these efforts have been rewarding. The exact incidence of permanent unilateral paralysis of the vocal cord is not known. It is known that a few patients will exhibit temporary paralysis on the basis of edema of the nerve and its sheath, perhaps from stretching or pinching of the nerve during operation. My associates and I have proved to our own satisfaction that this paralysis will clear up within twelve weeks. We have considered paralysis of longer duration to be permanent. We are continuing to employ local anesthesia combined with nitrous oxide in the average case so that we may determine immediately any compromise of the physiologic function of the nerve and maintain a status of temporary rather than of permanent paralysis. That is, a suture passed about the nerve may be removed at once or encroachment in the region of the nerve may be corrected. We have no argument with those who insist on dissecting the nerve, as we routinely bring the nerve into view in almost every case. However, without local anesthesia the nerve must be dissected completely and kept free during the entire procedure. Even in the most experienced hands, permanent unilateral paralysis may occur in nearly 1 per cent of patients. We are of the opinion that bilateral paralysis is completely avoidable.

The other great concern peculiar to surgical treatment is tetany. Once again, it is almost impossible to determine the exact percentage of permanent tetany in any series of cases but review of the literature suggests that it may well be 1 per cent. Cautious as surgeons may be in attempts to avoid removal of a parathyroid gland, it is still possible that such removal might be coincidental. Perhaps much more important is devitalization of the parathyroids brought about by radical attempts to rid the patient of the hyperplastic thyroid gland. Although tetany now may be treated much more easily than before it still is to be avoided if possible.

Disadvantages of Any Treatment

The possibility of recurrence of the original condition is always present. Regardless of the type of treatment

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From the Section of Surgery, Mayo Clinic and Mayo Foundation. The Mayo Foundation is a part of the Graduate School of the University of Minnesota.

used today, hyperthyroidism will recur in a few patients. The surgeon should be the first to admit that if sufficiently lengthy postoperative follow-up is allowed, the rate of recurrence in exophthalmic goiter will be about 5 per cent. This follow-up may require thirty years, as some patients may appear to be cured for long periods. Fortunately, recurrence after operation is extremely uncommon in the other types of hyperthyroidism. Attempts at nonsurgical control of the disease are fraught with a much higher rate of recurrence than this but it is granted that further nonsurgical treatment may ward off deleterious sequelae.

Myxedema is a condition that some surgeons still aim to produce in the patient who has a toxic goiter. My colleagues and I do not consider this to be a logical end point, and for that reason we still refuse to carry out routine total thyroidectomy in the treatment of benign goiters. The preservation of several grams of functioning tissue can provide a much happier existence for the patient. Even so we have seen myxedema in perhaps 10 per cent of our patients. Of course, this is far more acceptable than recurrence of the hyperthyroidism and is much easier to treat. Myxedema is much commoner when evidence of thyroiditis is found in the specimen. Induced hypothyroidism may not be permanent but if it can be avoided we prefer to do so.

Nonsurgical Treatment of Hyperthyroidism

During World War II, it became apparent that goitrogenic drugs might have a large bearing on the treatment of hyperthyroidism. Since 1943, Astwood has led the way in informing physicians concerning this most interesting field. A variety of products has been tested thoroughly and much information is available at present. It is beyond the scope of this discussion to delve deeply into that type of treatment, but my associates and I have experienced approximately 50 per cent failures in definitive treatment with goitrogens alone. Thiouracil was much too toxic but propylthiouracil has been used to good advantage in some cases. We have had limited experience with methylthiouracil. Toxic reactions are now few in number and often may be avoided with reasonable caution. However, we have found that the accurate, periodic, prolonged observation that is essential in every instance is so difficult that it discourages both patient and physician. It would be a rare patient indeed who would welcome the suggestion of thyroidectomy after a year or eighteen months of periodic checks in the belief that the hyperthyroidism was being brought under control. At present we are employing propylthiouracil in an occasional case as a preoperative preparatory agent. Prolonged preparation will be indicated in no more than 10 to 15 per cent of cases at most. We discontinue use of the goitrogens at least a week before operation if possible and carry along on administration of Lugol's solution alone. Should it be impossible to discontinue use of the goitrogens, Lugol's solution is also given during the last 10 days or so in an attempt to counteract the hemorrhagic, friable condition brought about in the gland by the goitrogens.

Radioactive iodine was received with great enthusiasm shortly after World War II. Its effect on the thyroid gland is obvious and it was only natural that an attempt should be made to replace surgical intervention completely. Large series of cases have been reported from several centers where radioiodine is employed in every case of toxic goiter. It is a simple method that appears to be extremely effective and rather permanent in many patients. However, some recurrence of hyperthyroidism has taken place in everyone's experience. It may be argued that recurrence may be treated by further exposure to radioactivity. However, we have encountered a few patients who have been uncontrolled after as many as seven doses of radioiodine.

The operative specimens removed after treatment with radioiodine often reveal an extremely interesting appearance of the gland in that it appears "cooked," and one certainly can see why hyperthyroidism might be controlled in many cases. Some patients have experienced temporary depression of the basal metabolic rate after use of therapeutic radioiodine, but subsequent doses have failed to produce much lasting effect on the hyperthyroidism. In addition, myxedema has been prone to develop, since it is difficult to establish the proper dosage. Estimation of the weight of the thyroid is essential in attempting to arrive at the correct dosage and it is well known that such estimation based on physical examination may be in error by as much as 75 per cent. It is to be admitted that myxedema after treatment is more acceptable than recurrence of the original disease, but a normal metabolic state is even more preferable.

At present, we are perhaps a little too conservative in our institution in the use of radioiodine in hyperthyroidism. Being aware of the changes produced in the thyroid gland, we have been concerned about possible changes in other endocrine glands. For that reason we have been reluctant to employ radioactivity in the childbearing years. The exact effect on the bone marrow and other hematopoietic structures in infants and children has not been clarified. With regard to possible carcinogenic properties of the agent, we are not aware of any documented case of cancer being produced in humans. However, we have been concerned occasionally about the kidneys, ureters and bladder, since the element is excreted in such concentrations by way of the urinary tract; as far as can be determined at present, no ill effects on this system have occurred. We use radioiodine in older patients who have hyperthyroidism. It is also used in younger patients who are poor candidates for surgical therapy for any reason. In addition, those patients who have had one or more thyroidectomies previously, who have little or no palpable glandular substance but who present themselves with recurrence of hyperthyroidism yield more material for radioactive therapy. This group, of course, will include those patients who have paralysis of the vocal cord or tetany or both, in which event the surgeon is not eager to re-explore the neck.

Major Types of Toxic Goiter

My colleagues and I have learned to draw sharp distinction between the two major forms of toxic goiter.

TABLE I. SURGICAL HOSPITAL MORTALITY RATE IN EXOPHTHALMIC GOITER

Period	Patients	Hospital Deaths	
		Number	Per Cent
1943 through 1945	1,006	5	0.5
1946 through 1953	1,378	0	0

TABLE II. SURGICAL HOSPITAL MORTALITY RATE IN ADENOMATOUS GOITER WITH HYPERTHYROIDISM

Period	Patients	Hospital Deaths	
		Number	Per Cent
1943 through 1945	713	8	1.1
1946 through 1953	992	3	0.3

Diffuse parenchymatous hypertrophy of the thyroid associated with Graves's disease, or exophthalmic goiter, always should be discussed separately, because this type responds much more quickly and completely to various forms of nonsurgical management than does adenomatous goiter with hyperthyroidism. When more and more medical agents became available, a critical analysis of the situation was offered by Black, in 1946.[1] At that time he reported on 302 consecutive patients who had undergone one-stage radical thyroidectomy for exophthalmic goiter; the hospital mortality rate was 0.3 per cent. The rate of recurrence was less than 5 per cent in this group of patients. He concluded at that time that the great majority of such patients were best treated surgically. He considered that prolonged preoperative preparation with propylthiouracil or an allied substance might convert the situation into a far more suitable one in the 2 to 3 per cent of patients not considered suitable surgical risks. In 1949, Pemberton and co-workers[2] followed up with a report on 455 consecutive patients who had thyroidectomy for exophthalmic goiter and in whom no hospital deaths occurred. In view of new therapy and the trend of selection of patients, I have brought the records at the clinic up to date in regard to hospital mortality in exophthalmic goiter by studying the most recent eleven-year period (Table I).

A greatly different situation exists with regard to adenomatous goiter with hyperthyroidism. We are convinced now more than ever before that these are the goiters which certainly should be removed surgically. The possibility of carcinoma in an adenomatous goiter no longer should be subject to any argument. Discussion still exists concerning the statistics of this coexistence, but the fact remains that all authorities find a definite incidence. It has been argued in the past that the association of hyperthyroidism with an adenomatous goiter renders the possibility of carcinoma extremely minute. However, our studies and those from other institutions indicate that this is no safeguard whatsoever. Fortunately thyroidectomy in these patients proves to be most gratifying. Control of the hyperthroidism is almost immediate and is indeed permanent. True recurrence of hyperthyroidism in the form of toxic nodular goiter is extremely rare, being in the neighborhood of 0.5 per cent after properly performed thyroidectomy. Somewhat higher morbidity and mortality rates are encountered in these patients. In the first place, these patients are older than those who have diffuse parenchymatous disease and all the ills that accompany advance in years are coincidental possibilities. In addition, the usual history is one of an insidious onset. The goiter may have been present for many years but fibrillation and other cardiac disturbances may have been of indefinite onset, so the patient is not aware of trouble until actual cardiac failure has ensued. Unfortunately, none of the medical agents is strikingly effective in reversing this phenomenon so that it is with some misgiving that the occasional patient is finally operated on, all other methods having failed. That proper preparation by all known means is of some value is demonstrated in the comparisons of mortality rates presented in Table II.

The situation in the most recent period with regard to the causes of hospital deaths after thyroidectomy in toxic goiter of any variety is presented in Table III.

TABLE III. POSTTHYROIDECTOMY HOSPITAL MORTALITY RATE
1946 Through 1953

Condition	Patients	Hospital Deaths	
		Number	Per Cent
Toxic goiter	2,370	3*	0.1
Adenomatous goiter without hyperthyroidism	3,732	2	0.05

*All three patients had adenomatous goiter with hyperthyroidism; in two the cause of death was cardiac failure and in one it was thryoid crisis.

Comment

The present report indicates the present stand of my colleagues and me with regard to the treatment of toxic goiter. We have taken this stand and consider that we are in a position to defend it. It is realized that progress in all fields of medicine and allied sciences compels us to take stock from time to time and always to be in a position to revise our thinking.

At present from 85 to 90 per cent of the patients undergoing surgical treatment for toxic goiter respond well enough to administration of Lugol's solution in large doses over a period of ten to fourteen days that we may proceed with surgical treatment with a high degree of confidence as to the outcome. The remaining patients will require preparation with a goitrogen carried out over a period lasting a minimum of six weeks and often much longer. The results of complete preoperative preparation are certainly evident from this report. The surgeon should be the first to give full credit to the clinicians for having developed the ultimate in proper selection of patients for the various methods of treatment.

Since any method of treatment for toxic goiter is known to possess a definite rate of recurrence and to be associated with certain failures and other disappoint-

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Minnesota Academy of Medicine

Meeting of December 9, 1953

The regular monthly meeting of the Minnesota Academy of Medicine was held at the Town and Country Club on Wednesday evening, December 9, 1953. Dinner was served at 7 p.m. and the meeting was called to order by the president, Dr. E. A. Regnier, at 8:20 p.m.

There were fifty-two members and four guests present.

Minutes of the November meeting were not read.

The president announced the death of Dr. Lee Barry, of Saint Paul, who died recently in Florida.

Dr. Ohage made a motion that any candidate who writes a thesis approved by the executive committee or the committee on credentials, becomes a member and then presents his thesis when the committee decides.

Dr. Wangensteen wished to amend this motion making it in conformity with the constitution and by-laws. Motion as amended, seconded and carried.

The scientific program followed.

Dr. Burtis J. Mears, of Saint Paul, read his inaugural thesis on "Fat Embolism."

FAT EMBOLISM

BURTIS J. MEARS, M.D.
Saint Paul, Minnesota

Fat embolism is possibly a common disorder that may be fatal, but it is difficult to diagnose with certainty. There is no pathognomonic sign, laboratory procedure, or X-ray findings to indicate that there are fat globules in the blood stream large enough to obstruct the pulmonary or cerebral arterioles. As will be shown, the history is probably the most reliable diagnostic tool.

Fat embolism is rare in children either because at this age there is insufficient subcutaneous and bone marrow fat[15] or because the proportion of the fluid olein to palmitin and stearin is much smaller.[4,11] Olein is in a semi-fluid state at body temperatures. Scuderi[10] agrees with the consensus of opinions that after injury to the bone marrow or adipose tissue there is liberation of fat which is aspirated by injured veins. Milaslavich[6] believes that bleeding into the injured area increases the pressure and enhances the liberation and aspiration of the fatty mixtures. Gauch[1] postulated that fat embolism was more common in fractures than in crushing injuries because the lacerated veins could not collapse when enclosed in a bony wall. The above hypothesis of the mechanism causing fat globules in the blood stream large enough to cause arterial obstruction, leaves many questions unanswered. For example, Lehman[5] found that there was approximately 65 cc of fat in the marrow of the femur, and if man could tolerate as much as the experimental dog, a lethal intravenous dose would be 120 cc. Also why, in some cases, should there be a latent period of forty-eight hours after injury or manipulation before symptoms occur? Ikeda,[2] in 1935, concluded that a few fat emboli do not embarrass the pulmonary circulation and it takes hours or several days for enough emboli to accumulate in the pulmonary bed to cause symptoms.

Warthin, in 1913,[13] stated that a relatively large amount of fat can obstruct many pulmonary capillaries without too much damage, but a very small amount of that fat oozing through the pulmonary capillaries and lodging in the cerebral vessels can be disastrous. This is important, as most cases dying of fat embolism, uncomplicated by shock and other factors, seem to tolerate the pulmonary symptoms relatively well. The brain injury seems to be the most disastrous sequel. Various authors[13,14] found fat globules in the lungs of a high percentage of patients dying without a history of trauma, and concluded, therefore, that fat embolism can occur without injury. This may be true, but it must be noted that no mention is made by these authors of finding fat emboli in the brain without the usual history of trauma to a long bone; and one of these authors[14] points out that very few fat globules were found in the lungs of nontraumatic deaths.

The metabolic disorders causing marked lipemia have not been proven to cause or increase the hazard of fat embolism. Scuderi[10] attempted to find oil droplets under the dark field in hyperlipemia without success, yet when he injected 5 cc of olive oil intravenously into the dog, he found droplets of oil under the dark field in the blood drawn from the opposite leg two minutes later. Fat emulsions given therapeutically to humans with oil droplets of the magnitude of one micron or less have given no evidence of fat embolism although a few have shown transitory substernal tightness during the administration.[3,9,12]

In the past twenty-two years at Ancker Hospital, Saint Paul, Minnesota, there have been eleven cases of fat embolism of the brain proven at autopsy by special staining of the frozen section. These figures are not significant as to the frequency of fat embolism because the examination of the frozen section has not been routine in accident cases and no record has been kept of the number of times this special examination has been made.

The eleven cases of proven fat embolism all had two anatomical findings in common:

1. Fat globules in the capillaries and small arterioles of the brain.
2. Petechial hemorrhages throughout the brain tissue.

The clinical history of these fatal cases showed a startling similarity. Three cases were eliminated from this study because one apparently died of a respiratory failure due to a traumatic pneumothorax, the second had no nurses notes or progress notes from which to glean a clinical picture, and the third died three weeks after injury with marked senile changes of the brain. Of the remaining cases, all were men. The ages ranged from twenty to seventy-seven, the average being 50.4. Alcoholism was apparent in one case and questionable in two. All eight cases had fractures of the lower extremities and two cases had additional fractures of the ribs. In only two cases was there evidence in the hospital records of definite respiratory embarrassment, and in one of these, cyanosis and Cheyne-Stokes respiration were the predominant symptoms prior to death. Two cases had no symptoms for a long period of time (three months and eight months) after injury but had symptoms and a fatal outcome occurring promptly following manipulation of their fractures. In seven of the eight cases severe delirium was the prominent symptom developing eight to thirty hours after injury or manipulation, the average waiting period being 13.7 hours. The nurses notes usually included descriptive notes as "talked incessantly," "tried to strike nurse," "visual hallucinations," "very restless," "pulled off dressings," et cetera. Three cases required restraints.

From this limited series of cases we get a consistent history and clinical picture. Given an adult male with a fracture of the lower extremity, who, about eight to fifteen hours after injury or manipulation of the fracture becomes suddenly confused with a severe degree of delirium, fat embolism of the brain is to be strongly suspected.

To aid in the diagnosis of fat embolism, a search for fat globules in the sputum and urine has been made by many investigators. Mussleman[7] made perhaps the most significant study of fat in the urine and sputum. He and his co-workers demonstrated fat in 32 per cent of the sputums but in only 2 per cent of the urines in a control series. On 109 cases of moderate to severe injuries, including cases of burns covering 10 to 20 per cent of the body surface, over one half showed fat globules in the urine and one third of these had pulmonic signs and one fourth had cerebral symptoms. Three of these patients coming to autopsy had fat globules in the cerebral and pulmonary vessels. All fatal cases in this series had fractures. It would seem, therefore, that fat in the sputum occurs without injury too often to be significant, but the urine carefully gathered and examined is a definite aid to diagnosis. Any fat present would float on top of the urine. The bladder has to be completely emptied, preferably with a catheter, and without an oily lubricant, and the last few cc of urine tested for the telltale fat.

Another confirmatory sign would be the finding of petechiae, especially on the palate and in the conjunctival sacs. Scuderi[10] demonstrated transitory X-ray changes in lungs of dogs following intravenous administration of fat and thought that serial X-ray studies would be of value. There were X-ray studies, all bedside films, on three of the eleven cases studied by this author. Review of these plates by a competent roentgenologist failed to reveal any edema or consolidation that could be attributed to a focal or widespread embolization.

There are many statements in the literature that fat embolism may occur without fracture of the long bones. In fact it is stated that such embolism may occur after severe burns, widespread bruising of the subcutaneous tissue, and surgery in obese patients. One author[8] in discussing the treatment of carbon tetrachloride poisoning stated that no milk or other fatty liquids should be used because of the dangers of fat embolism. These statements would seem unjustified until there has been further proof that fat embolism is a contributing factor to the morbidity or death of the patient suffering from conditions other than fracture of the long bones.

It is my belief that it is wrong to consider fat embolization in the differential diagnosis unless there has developed:

1. Petechiae of skin or mucous membrane.
2. Fat globules in urine.
3. Delirium in a patient eight to thirty hours after injury.

Conclusions

The literature on fat embolism, and eight cases of fat embolism of the brain proven at autopsy, are reviewed with the conclusion that the diagnosis can be made with accuracy only at the autopsy table, and then, only if special examination of the brain tissue is made. The diagnosis can be suspected antemortem if there is marked mental disturbance eight to thirty hours after a fracture or manipulation of a fracture of a long bone.

I must agree with Warthin that the lungs can usually tolerate a flood of fat emboli without serious results, but I must conclude that a fracture of a long bone is probably necessary to produce a huge number of fat emboli, and it is only in these latter cases that enough fat globules will ooze through the pulmonary capillaries to cause symptoms producing cerebral petechiae.

References

1. Gaush, H.: Arch. Int. Med., 18:76, 1916.
2. Ikeda, K.: Pulmonary fat embolism. Minnesota Med., 18:636, 1935.
3. Johnson, W. A., et al: Some effects of intravenous fat emulsions on human subjects. J. Lab. & Clin. Med., 39:176-183.
4. Landoise: Cited by Scuderi.
5. Lehman, E. P., and Moore, R. G.: Arch. Surg., 17:179-189, 1927.
6. Milaslavich, E. L.: Wisconsin J. Med., 29:139, 1930.
7. Musselman, M. M., et al: Fat embolism. Arch. Surg., 4:551, 1952.
8. Myatt, A. V., and Salmons, J. A.: Carbon tetrachloride poisoning. Arch. Indust. Hyg. & Occup. Med., 6:74-82, 1952.

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In Memoriam

LOOE BAKER

Dr. Looe Baker, pathologist for many years at Fairview Hospital in Minneapolis, died at the age of seventy at Laguna Beach in California, July 9, 1954. He had retired from practice because of ill health in 1948.

Dr. Baker was born in Detroit, Michigan. He received his secondary education at Howard Military School in Lima, Indiana, and attended the University of Michigan at Ann Arbor. He received his medical degree from Rush Medical College at Chicago and served his internship at Swedish Hospital in Minneapolis. After postgraduate training at the Rockefeller Institute, he specialized in clinical pathology and maintained an office for many years at 1111 Nicollet Avenue in Minneapolis as well as at Fairview Hospital.

Dr. Baker was a member of the Hennepin County Medical Society, the Minnesota State Medical Association and the American Medical Association. He is survived by his wife, Margaret; a son Looe, Jr.; and two daughters, Mrs. Charles Harris and Mrs. Arthur Enstis.

MICHAEL W. H. BOCKMAN

Dr. Michael W. H. Bockman, seventy-five years old, who practiced medicine for more than forty years in Minneapolis, died at Hot Springs National Park in Hot Springs, Arkansas, July 29, 1954.

Dr. Bockman was licensed to practice in 1904. He practiced industrial medicine and was a life member of the Hennepin County Medical Society, the Minnesota State Medical Association and the American Medical Association.

He is survived by his wife, Bessie B. Bockman.

BYRON A. KAMP

Dr. Byron A. Kamp of Albert Lea died unexpectedly of a heart ailment on the way to Naeve Hospital, Sunday, July 18, 1954, at 8:30 p.m. He was seventy-one years old and had retired from active practice in 1948.

Dr. Kamp was born in Pleasantville, Iowa, attended high school in Keokuk and received his degree in medicine from Drake University, Des Moines, in 1909. After subsequent postgraduate training in Chicago, he specialized in eye, ear, nose and throat work and became one of the founders, more than thirty years ago, of the Albert Lea Surgical and Medical Clinic.

He served in the Spanish-American War from 1900 to 1903 and also in World War I in 1918 and 1919. He was a member of the Freeborn County Medical Society, the Minnesota State Medical Association and the American Medical Association. He was a member also of the American Legion, the Veterans of Foreign Wars, the Spanish-American War Veterans and the Elks.

He is survived by his wife, the former Ruth V. Ford whom he married in 1920; one son, Gerard P. Kamp of Los Angeles; two daughters, Mrs. Arturo Anaya of El Paso, Texas, and Mrs. James F. White of Sparta, Illinois, and five grandchildren. Two sisters, Mrs. Archibald Crozier of Madison, Wisconsin, and Mrs. Albert Mix of Linden, Iowa, also survive.

WILLIAM C. LOVSHIN

Dr. William C. Lovshin, thirty-one years old, was killed in an airplane accident near Duluth Air Base on May 31. He was on active duty with the U.S. Air Force at the time and was returning from a trip to Indianapolis. Eleven other men lost their lives in the accident.

Dr. Lovshin was born at Chisholm and received his secondary education at the Chisholm High School and Phillips Exeter Academy in New Hampshire. He was graduated in medicine from the University of Wisconsin in 1947 and served a one-year rotating internship at the Indiana University Medical Center in Indianapolis before associating himself with the More-Ewens Clinic in Eveleth in 1948.

As a student, Dr. Lovshin took part in all athletics and was on his University basketball and baseball teams. After establishing himself in Eveleth he took an active part in civic affairs, becoming chairman of public health for the Junior Chamber of Commerce and a memorial in his honor is now planned by Eveleth Jaycees for his outstanding work with the Junior Chamber.

Dr. Lovshin joined the United States Air Force in 1953 and was stationed at San Antonio, Texas, before being transferred to the Duluth Air Base in August of 1953.

Survivors include his wife, Marlyn; two sons, William, Jr. and Ralph Jon; his parents, Mr. and Mrs. Joseph J. Lovshin, Chisholm; one sister Mrs. Joseph Mantel, Minneapolis; two brothers, Dr. Leonard Lovshin associated with the Crile Clinic in Cleveland, and Ralph Lovshin, track coach at Phillips Exeter Academy, Exeter, New Hampshire.

PETER HARVIE MACFARLANE

Dr. P. H. Macfarlane, fifty-nine, long associated with the Mesaba Clinic and the Chisholm Memorial Hospital, died at his home in Chisholm July 9, 1954. He was a leader in civic and educational affairs in Chisholm, a member of the Shrine, a past president of Kiwanis and well known to physicians of the state.

Dr. Macfarlane was born and went to school in Minneapolis and received his medical degree from the University of Minnesota. After a year's internship at Miller

Hospital in Saint Paul, he went to Chisholm in July, 1926, his medical education having been interrupted for service with Base Hospital 26 in France and a year's study at the University of London after World War I. He was a member of the St. Louis County Medical Society, the Minnesota State Medical Association and the American Medical Association and of the American Legion and the Veterans of Foreign Wars.

Survivors are his wife, Marcia, and two sons, Keith, who is a student at Carleton College, and David, who is at home.

WILLIAM JOSEPH NOONAN

Dr. William J. Noonan, Minneapolis urologist, died at the age of forty-four, Thursday, July 21, 1954, following a heart attack. He had been in apparent good health until the day before his death.

Dr. Noonan was born in Minneapolis, went to school in Watertown, South Dakota, and received a B.S. degree from the University of Minnesota. He was graduated in medicine from the University of Chicago and served his internship at Albert Merritt Billings Hospital in Chicago. He was licensed to practice in Illinois after more than four years' postgraduate work in urology at the University of Chicago. After service with the United States Army from 1941 to 1945, he established himself in Minneapolis and practiced urology there until his death.

He was a member of the staffs of St. Barnabas, Abbott and Midway Hospitals and of the teaching staff of the University of Minnesota. He was also a member of the Twin City Urological Society, the Minneapolis Academy of Medicine, the Hennepin County Medical Society, the Minnesota State Medical Association and the American Medical Association. He belonged to the Minneapolis Athletic, University and Dial Clubs and to Theodore Peterson Post of the American Legion.

Two brothers, Charles F. of Minneapolis and Willard F. of Grand Rapids, Michigan, survive him.

FRED WHARTON RANKIN

Dr. Fred W. Rankin, internationally known surgeon and former head of a section on surgery at the Mayo Clinic, died May 22, 1954, after a long illness at his home in Lexington, Kentucky. He was sixty-seven years old.

Dr. Rankin had been president of the American Medical Association in 1942 and president of the American Surgical Association in 1949 and he was president of the American College of Surgeons when he died. He was the third person to serve as president of all three major medical groups. The other two were Dr. C. H. Mayo and Dr. William J. Mayo, the former having been Dr. Rankin's father-in-law.

Throughout his illustrious career, Dr. Rankin received many other honors including a recent installation with four other American surgeons as honorary fellows of the Royal College of Surgeons at Edinburgh, Scotland. His military career was outstanding, also. He was a colonel in the medical reserve corps at the outset of World War II but became chief consulting surgeon attached to the office of the Surgeon General with rank of Brigadier General in 1942. He was awarded the distinguished service medal for this work and held service ribbons for most of the war theaters. He had served as a major in World War I and as commanding officer of Base Hospital No. 26 in France.

Dr. Rankin came to Minnesota first as a fellow in surgery of the Mayo Foundation in 1916. After the war, he returned to complete his fellowship and left to become professor of surgery at the University of Louisville. He returned to Rochester as head of a section in surgery at the Mayo Clinic in 1926 and remained until 1933 when he went back to Lexington and established himself again in the practice of surgery there.

He was born in Mooresville, North Carolina, and was educated at Davidson College in North Carolina and at the University of Maryland from which he received his medical degree. He held many honorary degrees from universities and colleges throughout the country.

He married Miss Edith Mayo, in 1923, and she and three sons and a daughter survive him. The children are Fred Wharton, Jr., Edith Graham, Charles Mayo and Thomas Alexander.

ORVILLE M. ROTNEM

Dr. Orville M. Rotnem of Harmony died as the result of an automobile accident at St. Mary's Hospital in Rochester, July 25, 1954. He was thirty-five years old.

Dr. Rotnem had been driving with his wife, Marjorie, when his car went out of control two miles south of Mabel in Fillmore County. He was returning from Iowa City where he had been attending postgraduate courses in medicine when the accident occurred. Mrs. Rotnem was also injured in the crash and was taken to the hospital for treatment.

Born at Caledonia, Dr. Rotnem went to high school at Edison in Minneapolis and received his degree in medicine from the University of Minnesota. He served a nine months' internship at Milwaukee County Hospital in Milwaukee, Wisconsin, followed by nine months' postgraduate work in surgery at the same hospital. He served in the United States Army Medical Corps and, after his discharge, he practiced briefly at Spring Grove and at Mabel before moving to Harmony. He was a member of the staffs of the Harmony Community Hospital and the Decorah Hospital at Decorah, Iowa, and had served as health officer at Mabel. He was a member of the Olmsted-Houston-Fillmore-Dodge County Medical Society, the Minnesota State Medical Association and the American Medical Association.

Survivors include his wife, Marjorie; a daughter, Diane; a son, Richard; and his parents, Mr. and Mrs. Thomas Rotnem of Minneapolis.

◆ Reports and Announcements ◆

MAYO MEMORIAL DEDICATION

"Medical Education and Research Freedom and Progress in Mid-Twentieth Century" will be the theme of the two-day activities dedicating the University of Minnesota's new Mayo Memorial.

According to University Medical School Director Robert B. Howard, M.D., all physicians and interested persons are invited to attend Mayo Memorial dedication programs, Thursday and Friday, October 21-22.

Highlighting activities will be the dedication banquet Thursday evening, October 21, in Coffman Memorial Union main ballroom. Harold S. Diehl, Dean, College of Medical Sciences, will preside. Speakers will include Donald J. Cowling, chairman, Committee of Founders, Minnesota Governor C. Elmer Anderson, Charles W. Mayo, Professor of Surgery, Mayo Foundation, Leonard A. Scheele, Surgeon General, U.S.P.H.S., James L. Morrill, president of the University of Minnesota, and Dr. Alan Gregg, Vice President of the Rockefeller Foundation.

The complete dedication program is as follows:

Thursday, October 21

Morning Session—10 a.m.

SPEAKERS:

William P. Shepard, Second Vice President, Health and Welfare Division, Metropolitan Life Insurance Company.

A. C. Furstenburg, Dean of the University of Michigan Medical School and Professor and Chairman of the Otolaryngology Department.

Osborne A. Brines, Professor of Pathology, Wayne University College of Medicine.

Frank H. Krusen, Professor and Head of the Section of Physical Medicine and Rehabilitation, Mayo Clinic, Rochester.

Afternoon Sessions—1:30-5:00 p.m.

SPEAKERS:

J. Chassar Moir, Professor of Obstetrics and Gynecology, Oxford University, England.

I. S. Ravdin, John Rhea Barton, Professor of Surgery, University of Pennsylvania.

Mrs. Lucile Petry Leone, Assistant Surgeon General and Chief Nurse, office of the U. S. Public Health Service.

Henry W. Woltman, Professor and Chairman of Neurology and Psychiatry Sections, Mayo Clinic, Rochester.

Francis J. Braceland, Psychiatrist-in-chief, Institute of Living, Hartford, Conn.

Dedication Banquet—6:30 p.m.

Friday, October 22

Morning Sessions—9:00-11:35 a.m.

SPEAKERS:

Rene Dubos, member of the Rockefeller Institute for Medical Research.

Jack Masur, Assistant Surgeon General and Chief of the U. S. Public Health Service Bureau of Medical Services.

William H. Sebrell, Director, National Institutes of Health, U. S. Public Health Service.

Charles A. Janeway, Thomas Morgan Rotch Professor of Pediatrics, Harvard University Medical School.

R. R. Newell, Professor of Medicine, Stanford University School of Medicine.

Afternoon Sessions

12:00-1:30 p.m.—University Hospitals Staff Meetings.—Herman R. Drill, Past President, Minnesota Medical Alumni Association, and Ray M. Amberg, Director, University of Minnesota Hospitals.

1:30-2:00 p.m.—Minnesota Medical Alumni Association annual meeting.

2:00 p.m.—Annual Medical Alumni Association Clinics.

6:30 p.m.—Radisson Hotel, Minneapolis, Medical Alumni Association banquet.

FAMILY DOCTORS' DAYS
at the
University of Minnesota Hospitals

All physicians are cordially invited to attend the following Family Doctors' Days which will be presented under the direction of the department indicated.

October—No Family Doctors' Day
(Oct. 21-22—Dedication of Mayo Memorial)
November 17—Department of Pathology
December 15—Division of Ophthalmology
January 19—University Hospitals Administration

Activities begin with luncheon at 12:15 p.m. with the staff of the department in the Hospital Dining Room and will continue throughout the afternoon.

For further information, write the head of the department concerned or the Director, Department of Continuation Medical Education, University of Minnesota Hospitals.

CRIPPLED CHILDREN'S SERVICES

The following Crippled Children's services fall clinic schedule has been released:

Place	*Date*	*Building*	*Counties*
*Crookston	Sept. 16	High school	Norman, Polk
*Bemidji	Sept. 25	High school	Beltrami, Hubbard, Clearwater, Cass.
*Little Falls	Oct. 2	Lindbergh school	Morrison, Todd, Mille Lacs
*Marshall	Oct. 9	High school	Lac qui Parle, Redwood, Lincoln, Yellow Medicine, Lyon
**Virginia	Oct. 13	City Hall	St. Louis
*Fergus Falls	Oct. 16	High school	Otter Tail, Wilkin
**Winona	Oct. 20-22	City Building	Fillmore, Winona, Wabasha, Houston, Olmsted
**Hibbing	Oct. 27	Village Hall	St. Louis
*Willmar	Oct. 30	High school	Chippewa, Meeker, Renville, Kandiyohi, McLeod, Swift
*Mankato	Nov. 6	New High school	Blue Earth, Sibley, Nicollet, LeSueur, Martin, Watonwan, Faribault
**Hibbing	Nov. 10	Village Hall	St. Louis
*Anoka	Nov. 13	Lincoln school	Anoka, Chisago, Hennepin, Isanti, Kanabec, Pine, Ramsey, Sherburne, Washington, Wright

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*County Welfare Board social service for clinic intake arranged by field representative. New patients to bring written referrals from physicians.

**Social problems at clinic to be referred to County Welfare Board on day of clinic. Intake service not necessary from Welfare Board. Gillette patients not to attend clinics. New patients to bring written referrals from physicians. All patients to have appointments arranged through Public Health Nurse.

APPLICATIONS ACCEPTED

The American Board of Obstetrics and Gynecology, Inc., announces that applications for certification (American Board of Obstetrics and Gynecology) for 1955 Part I examinations are still being accepted. Candidates may contact Dr. Robert L. Faulkner, Secretary, 2105 Adelbert Road, Cleveland 6, Ohio.

MEDICAL MEETINGS

National

American Cancer Association, Board of Directors and special committee meetings, Hotel Radisson, Minneapolis, Minn., June 2, 1955.

American College of Gastroenterology, First annual convention, Shoreham Hotel, Washington, D. C., October 25-27, 1954.

American Public Health Association, Eighty-second annual meeting, Buffalo, New York, October 11-15, 1954.

Association of Military Surgeons of the United States, Sixty-first annual convention, Hotel Statler, Washington, D. C., November 29-December 1, 1954.

Muscular Dystrophy Associates of America, Inc., Third annual conference, New Yorker Hotel, New York City, October 8-9, 1954.

National Gastroenterological Association, Nineteenth annual convention, Shoreham Hotel, Washington, D. C., October 25-27, 1954.

National Society of Crippled Children and Adults, Hotel Statler, Boston, Mass., November 3-5, 1954.

Pharmacy and the Conquest of Disease, Bicentennial conference, Columbia University, New York City, October 14-15, 1954.

International

International Anesthesia Research Society, Los Angeles, Calif., October 10-15, 1954.

International Congress of Diseases of the Chest, Barcelona, Spain, October 4-8, 1954.

AMERICAN COLLEGE OF CHEST PHYSICIANS

The American College of Chest Physicians registered 1150 physicians and guests at its twentieth annual meeting held in San Francisco, California, June 17-20, 1954. This was the largest registration of any of the previous College meetings held on the west coast.

Officers elected for 1954-1955 are:

President—William A. Hudson, Detroit, Michigan
President-Elect—James H. Stygall, Indianapolis, Indiana
First Vice President—Herman J. Moersch, Rochester, Minnesota
Second Vice President—Burgess L. Gordon, Philadelphia, Pennsylvania
Treasurer—Charles K. Petter, Waukegan, Illinois
Assistant Treasurer—Albert H. Andrews, Jr., Chicago, Illinois
Chairman, Board of Regents—Donald R. McKay, Buffalo, New York
Historian—Carl C. Aven, Atlanta, Georgia

Dr. John F. Briggs of Saint Paul is Regent for the District and Dr. Arthur M. Olsen of Rochester is Governor of the College for Minnesota.

The twenty-first annual meeting of the College will be held in Atlantic City, New Jersey, June 2-5, 1955.

FELLOWSHIPS FOR BASIC RESEARCH IN ARTHRITIS

The Arthritis and Rheumatism Foundation is offering the following research fellowships in the basic sciences related to arthritis:

1. Predoctoral fellowships ranging from $1,500 to $3,000 per annum, depending on the family responsibilities of the fellow, tenable for 1 year with prospect of renewal.

2. Postdoctoral fellowships ranging from $4,000 to $6,000 per annum, depending on family responsibilities, tenable for 1 year with prospect of renewal.

3. Senior fellowships for more experienced investigators will carry an award of $6,000 to $7,500 per annum and are tenable for 5 years.

The deadline for applications is October 15, 1954. Applications will be reviewed and awards made in January, 1955.

For information and application forms, address Medical Director, The Arthritis and Rheumatism Foundation, 23 West 45th Street, New York 36, N. Y.

CONFERENCE ON SILICOSIS AND OCCUPATIONAL CHEST DISEASES

A conference on silicosis and occupational chest diseases jointly sponsored by the McIntyre Research Foundation of Toronto, Canada, and the Saranac Laboratory of Saranac Lake, New York, has been arranged for Monday, Tuesday, and Wednesday, February 7, 8, and 9, 1955, in the Town Hall at Saranac Lake.

The papers to be presented in the five full sessions will all report on original work conducted or sponsored by either the McIntyre Research Foundation or the Saranac Laboratory. In addition there will be papers presented by guest lecturers.

Anthony J. Lanza, M.D., formerly director of the Institute of Industrial Medicine and now emeritus professor of Industrial Medicine at New York University-Bellevue Medical Center, has been named chairman of the conference.

Business arrangements, including reservations, will be handled by Norman R. Sturgis, Jr., and the treasurer will be Clarence L. Wagner, both of the Trudeau-Saranac Institute staff. All communications concerning the conference should be addressed to N. R. Sturgis, Saranac Laboratory, Saranac Lake, New York.

SYMPOSIUM TO BE TELECAST

A nationwide closed circuit hookup will be used for the first time for postgraduate medical education September 23, when a symposium on the subject "Management of Hypertension" will be broadcast.

The program, which is sponsored by the American College of Physicians, will be received from 6 to 7 p.m., September 23, at the Radisson Hotel, Minneapolis.

GRANT AWARDED

The Eli Lilly Company of Indianapolis, Ind., has announced that a one-year research grant has been awarded the University of Minnesota. The grant will be used for studies on the relationship of Vitamin B_{12} and intrinsic factors under the direction of Dr. Paul S. Hagen, assistant professor of medicine.

COURSE FOR X-RAY TECHNICIANS

The Minnesota Society of X-Ray Technicians announces a regional refresher course for x-ray technicians for the Twin City region.

The course, jointly sponsored by the Society, the Minnesota State Medical Association, the Minnesota Hospital Association and the Minnesota Department of Health, will be held Tuesday and Thursday evenings from September 28 through November 4 on the University of Minnesota Campus.

The course is open to technicians in hospitals and in physicians' offices. Registrations should be sent to Mr. Glen Taylor, Executive Secretary, Minnesota Hospital Association, Student Health Service, University of Minnesota, Minneapolis 14, Minnesota. Tuition fee is $25.00.

MINNEAPOLIS HEALTH DAY

October 7 is *Health Day* in Minneapolis.

Conducted as an open house, *Health Day* will be held from 9 a.m. to 3:30 p.m. in the Radisson Hotel ballroom. It is a free community service project devoted to health problems and their solutions. The public is invited.

Dr. Walter C. Alvarez will speak on problems of the aging and Dr. Leo Rigler will talk on cigarettes and lung cancer. A movie on heart surgery, "One Victory," will be shown and discussed by Dr. C. Walter Lillehei and Dr. Richard Varco.

Dr. Reginald Dunton and Dr. R. F. Johnson will talk on "corrective dentistry." "What Can be Done about Lung Tumors" is Dr. Kenneth Jensen's topic. Russell Nye, Northwestern Hospital administrator, will discuss "You and Your Hospital."

There will be twenty-five various exhibits and anyone may obtain free chest x-ray, diabetic test and blood type.

Health Day is presented by the Women's Auxiliaries of the Hennepin County Medical Society and Minneapolis District Dental Society. General chairman is Mrs. Robert B. Potter, with Mrs. Milton T. Lundblad as dental chairman. Co-chairmen are Mmes: Rolf L. Andreassen, Edward Donatelle, Freeman Kovack, Malcolm McCannel, William T. Smith and Cabot Wohlrabe.

Thomas P. Cook, executive secretary of the Hennepin County Medical Society, is acting as advisor.

CONTINUATION COURSES

A continuation course in *The Use of the Minnesota Multiphasic Inventory (MMPI)* will be presented October 14 to 16, 1954, at the Center for Continuation Study. The course is intended primarily for clinical psychologists but will also be open to psychiatrists and other physicians who may be interested in the use of the MMPI. Guest faculty will include Drs. George Welsh, Associate Professor, Department of Clinical Psychology, University of North Carolina, Chapel Hill; and Harrison Gough, Assistant Professor, Department of Psychology, University of California, Berkeley. The course will be presented under the direction of Dr. Starke R. Hathaway, Professor and Director, Division of Clinical Psychology, and the remainder of the faculty will be drawn from the faculty of the University of Minnesta.

On November 4 to 6, 1954, the University of Minnesota will present a continuation course in *Anesthesiology for General Physicians* at the Center for Continuation Study. The course will deal with all types of anesthetic techniques, including those used in obstetrics. Newer anesthetic agents will be discussed. The course will be presented under the direction of Dr. Frederick H. Van Bergen, Associate Professor and Acting Director, Division of Anesthesiology, University of Minnesota. The faculty will include several outstanding guest speakers and members of the faculty of the University of Minnesota Medical School and the Mayo Foundation.

* * *

The annual continuation course in *Radiology for Specialists* will be held at the Center for Continuation Study November 9 to 13, 1954. *Bone and Joint Diseases* will be the subject of this year's course. Guest faculty will include Drs. C. Howard Hatcher, Professor of Orthopedic Surgery, University of Chicago School of Medicine; Paul C. Hodges, Professor, Department of Radiology, University of Chicago School of Medicine; John F. Holt, Professor, Department of Radiology, University of Michigan Medical School, Ann Arbor; G. S. Lodwick, Chief, Department of Radiology, Veterans Administration Hospital, and Clinical Assistant Professor, Department of Radiology, State University of Iowa College of Medicine, Iowa City; Edward E. Neuhauser, Associate Radiologist, Harvard Medical School, Boston; and Isidore Snapper, Director of Medicine and Medical Education, Beth-El Hospital, Brooklyn, New York. The course will be presented under the direction of Dr. Leo G. Rigler, Professor and Head, Department of Radiology, and the remainder of the faculty will include members of the faculty of the University of Minnesota Medical School and of the Mayo Foundation.

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The University of Minnesota announces a continuation course in *Infectious Diseases for General Physicians* which will be held at the Center for Continuation Study next November 18 to 20. Practical and fundamental aspects of the diagnosis and management of infectious diseases of all types will be taken up. Guest speakers will be Drs. Harry Eagle, Chief, Section of Experimental Therapeutics, National Microbiological Institute, National Institutes of Health, Bethesda, Maryland; and Ellard Yow, Assistant Professor of Medicine, Baylor University School of Medicine, Houston, Texas. Dr. Eagle will also present the annual Journal-Lancet lecture Thursday evening, November 18. The course will be presented under the direction of Dr. W. W. Spink, Professor, Department of Medicine, and members of the faculty of the University of Minnesota Medical School and the Mayo Foundation will complete the faculty.

PRESENT STATUS OF SURGERY IN THE TREATMENT OF TOXIC GOITER

(Continued from Page 685)

ments, my colleagues and I still resort to surgical treatment in many patients since it alone has stood the test of time.

Summary and Conclusions

The steady decline in hospital mortality rate after thyroidectomy has extended almost to the end point.

Correct preparation of the patient by means of Lugol's solution, or with propylthiouracil whenever definitely indicated, has been responsible for this agreeable state of affairs.

Clinicians should get full credit for this continuing improvement.

Administration of radioactive iodine appears to be a simple and often extremely effective method of controlling exophthalmic goiter. Someday it may prove to be the method of choice. At present its unknown factors limit its universal use.

References

1. Black, B. M.: The present status of the treatment of toxic goiter. Mississippi Valley M. J., 69:143-148 (Oct.) 1947.
2. Pemberton, J. deJ., Haines, S. F. and Keating, F. R., Jr.: The current treatment of hyperthyroidism. J. Clin. Endocrinol., 9:1232-1237 (Nov.) 1949.

FAT EMBOLISM

(Continued from Page 687)

9. Neptune, E. M., et al: The successful administration of large quantities of fat emulsions to man. Surg. Gynec. & Obst., 92:365-369.
10. Scuderi, C. S.: Fat embolism. Surg. Gynec. & Obst., 72:732-746, 1941.
11. Speed, K.: Fractures and Dislocations. 1st ed. New York: Lippincott, 1915.
12. Stare, F. J., et al: Fat in parenteral nutrition. Surg. Gynec. & Obst., 92:246-249.
13. Warthin, A. S.: Traumatic lipemia and fatty embolism. Int. Clinic, 4:171-227, 1913.
14. Wright, R. B.: Fat embolism. Ann. Surg., 96:75, 1932.
15. Zenker: Cited by Scuderi.

Gareth Hiebert, of the editorial department of the *Saint Paul Dispatch*, then gave a talk on "The Doctor and the Press."

There being no further business to come before the academy, the meeting was adjourned.

Robert E. Priest, M.D.,
Secretary.

Of General Interest

NEW LOCATIONS

Dr. Vernon D. Erickson of Minneapolis has joined the staff of the Grand Rapids Clinic. Dr. Erickson studied internal medicine last year at General Hospital, Minneapolis.

* * *

Dr. Arthur Neumaier, who has practiced medicine in Glencoe since 1941, has joined the Glendive Clinic, Glendive, Montana, as internist.

* * *

Dr. Alex Barno has returned to his practice in St. Louis Park after spending eighteen months with the Navy at Key West, Florida.

* * *

Dr. C. A. Rohrer, formerly of Waterville, has joined the staff of the Worthington Clinic. Dr. Rohrer, a University of Minnesota medical school graduate, has done radiologic work at Des Moines, Iowa, and the University of Iowa.

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Dr. Vincent Ryding, who has been resident surgeon at Northwestern Hospital in Minneapolis and Methodist Hospital in Dallas, Texas, is now with the Surgical and Medical Clinic in Albert Lea.

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Dr. John Sarno, a former Newark, New Jersey, surgeon, has become affiliated with the Falls Clinic in Thief River Falls.

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Dr. Gregory Olson, a former Redwood Falls physician, is now associated with **Dr. J. E. Verby** and **Dr. David Allison** of Litchfield.

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Dr. C. J. Swendseen, who has practiced medicine in Minneapolis and Graceville, is now located in Alexandria.

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Dr. Henry A. Johnsen, Jr., a former fellow in the section of Internal Medicine at Mayo Clinic, Rochester, has joined the staff of the Lenont-Peterson Clinic in Virginia.

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Dr. R. J. Camp of Woodward, Oklahoma, has become associated with the Madison Clinic at Madison.

* * *

Dr. Donald M. Odland of Dell Rapids has begun general practice of medicine in Luverne. Dr. Odland recently completed advanced work in surgery at the University of Minnesota.

* * *

Dr. Eldore B. Nash has taken over duties with Dr. E. Lillian Parson and Dr. L. R. Parson in their Elbow Lake Clinic. Dr. Nash, a University of Minnesota Medical School graduate, took his internship at Memorial Hospital in Phoenix, Arizona.

Dr. Malcolm M. Fifield recently announced the opening of an office for the practice of urology in the Duluth Medical Arts Building. Dr. Fifield has completed a three-year residency at Veterans Hospital and University Hospitals, Minneapolis.

* * *

Dr. G. Charles Wilcox, who has practiced medicine in Wood Lake the past year, has moved to Albert Lea to join the Medical and Surgical Center. Dr. Wilcox previously resided in St. Peter.

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Dr. B. R. Kirklin, Rochester radiologist, will move his offices from the First National Bank building to the Kahler Hotel.

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After fifteen years with the Mesaba Clinic, **Dr. A. C. Tingdale** has opened offices for private medical practice in Hibbing. Dr. Tingdale is a staff member of Hibbing General Hospital and Chisholm Memorial Hospital.

Dr. Clarence J. Rowe, Jr., is director of the newly opened Hamm Memorial Psychiatric Clinic in St. Paul. The clinic offers out-patients psychiatric therapy to Ramsey County adults unable to pay for private care.

* * *

A Mountain Lake physician, **Dr. Peter J. Pankratz,** has filed as a candidate for state senator in the 20th District, which includes Cottonwood and Jackson counties. A graduate of the University of Minnesota School of Medicine, Dr. Pankratz has practiced for twenty-one years. He served with the U. S. Army Air Force as flight surgeon during World War II.

* * *

Dr. Nellie Holman's experiences as a medical missionary to China in the 1920's are recorded in a new book release "My Most Unforgettable Patients." The author, a former Appleton resident, was graduated from the University of Minnesota medical school in 1919. A missionary of the Evangelical Lutheran Church, Dr. Holman spent eleven years in China. In 1931, Dr. Holman contracted tuberculosis and returned to Minneapolis' Glen Lake Sanatorium. Dr. Holman now makes her home in Melbourne, Florida.

* * *

Dr. J. F. Shronts, medical director of General Mills, Inc., has been elected president of the Minnesota Academy of Occupational Medicine and Surgery. Other officers include **Dr. Leslie Foker** of Minneapolis, vice president; **Dr. James Fox,** Minneapolis, secretary; **Dr. Tracy E. Barber,** Austin, treasurer, and **Dr. Gordon MacRae,** Duluth, recorder. The academy was organized last year to promote sound medical practices in industry.

An open house observing the fourth anniversary of the Sandstone State Hospital was held last month. **Dr. Kenneth Douglas,** director, was in charge of the program, which included guided tours of the various departments and wards and talks by staff members.

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Dr. Norman B. Metcalf, who has been serving as lieutenant in the Navy hospital at Jacksonville, Florida, has resumed medical practice in Princeton; He is associated with the Blomberg-McManus Clinic.

* * *

Now serving as chief of staff physicians affiliated with Northwestern Hospital of Thief River Falls is **Dr. M. D. Starekow. Dr. William Fiegal** was elected vice president and **Dr. D. E. Greene** will serve as secretary-treasurer.

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A Superior Service Award was presented **Dr. Irvin Kerlan,** a former resident of McGregor, by the U. S. Department of Health, Education and Welfare at its third annual Honor Awards ceremony. Dr. Kerlan, a graduate of the University School of Medicine, is employed by the department in Washington, D. C. Last year he contributed more than 1,800 children's books and illustrations for use by the University's Library Instruction and Education sections.

* * *

Studying medical programs in Europe is **Dr. James R. Fox** of Minneapolis, Dr. Fox, who is associated with the University of Minnesota Health Service, will study medical facilities and payment programs in the Scandinavian countries and will incorporate his findings in a report similar to that prepared after a survey of Great Britain in 1952. Dr. Fox will also visit Vienna and Berlin during his stay. Dr. Fox is director of the radio and television program "How's Your Health," a joint project of the Minnesota State Medical Association and the University of Minnesota. His trip abroad is sponsored by the American Medical Association.

* * *

The appointment of **Dr. Otto E. Benell** of Virginia, as staff radiologist of Falls Memorial Hospital, International Falls, has been announced. Dr. Benell will spend one day each week in International Falls to supervise x-ray work.

* * *

Dr. Elizabeth A. Leggett has resigned her position as head of the Indian out-patient office of the Indian Welfare Department in Bemidji. Dr. Leggett will be associated with the Santa Ana, California, Health Department. Before moving to Bemidji, Dr. Leggett practiced at the Ah-Gwah-Ching sanatorium and at Cass Lake.

* * *

New president of the Minnesota Radiological Society is **Dr. Chauncey N. Borman** of Minneapolis. Other newly-elected officers of the society are: **Dr. Harold O. Peterson,** St. Paul, vice president; and **Dr. John R. Hodgson,** Rochester, secretary-treasurer.

Reporting for duty at Fort Sam Houston, Texas, this month is **Dr. H. M. Brown,** formerly of Walker. Dr. Brown will report to the Medical Officers service school for orientation and temporary duty before receiving a permanent duty. His tour of service will be twenty-one months.

* * *

Dr. George Nelson Aagaard, former director of Post-Graduate Medical Education at the University of Minnesota, has been appointed dean of the University of Washington Medical School by the University's Board of Regents. Dr. Aagard, who received his medical degree from the University of Minnesota in 1937, was named to the University faculty in 1941. In 1948 he was appointed Post-Graduate Medical Education director and associate professor of medicine. He held this appointment until 1952, when he was named dean of the University of Texas' Southwestern Medical School. The Washington post has been open since 1953, when **Dr. Edward L. Turner,** first dean of the School of Medicine, resigned to become secretary of the Council of Medical Education and Hospitals of the American Medical Association. Dr. Aagaard assumed his new post the first of this month.

* * *

G. Albin Matson, director of the Minneapolis War Memorial Blood Bank, is attending two international conferences in Paris this month. He will give a paper at the fifth International Congress of Blood Transfusions and will attend the fifth International Congress of Hematology. Matson's paper will deal with hereditary blood factors among the American Indians.

* * *

A Marshall physician, **Dr. Robert F. Gray,** has left for two years of research work in British East Africa. Sponsored by the Ford Foundation, Dr. Gray will use the fellowship to survey both health problems among the natives and medical research on these problems. Dr. Gray has served with both the American and British Armies, and was stationed in Senegal, French West Africa and British East Africa. He returned to Africa for a year in 1950 on a Fulbright research grant for study of native medicine in Tanganyika.

* * *

Commencement speaker at exercises ending the University's second summer session was **Dr. Charles Mayo,** Rochester physician and statesman. Some 400 students and their guests heard Dr. Mayo, who is member of the U. S. delegation of the UN as well as a University Regent, speak on the subject "The Factor of Time."

* * *

Dr. Moses Barron of Minneapolis has been named governor of the American Diabetes Association for the state of Minnesota, **Dr. Henry Mulholland,** association president, has announced. Dr. Barron also was re-appointed to the committee on detection and education and will supervise state-wide effords at further education in diabetes and its control.

* * *

More than 1,000 visitors attended the grand opening of Hawley's new Medical Center recently. Included in activities were a band concert, refreshments and tours of the Center. **Dr. Arnold M. Berg** and **Dr. V. D. Thysell** will be located in the new building.

* * *

Dr. Alfred Doscherholmen, resident in medicine at the University of Minnesota, has been awarded a $4,000 grant for research on the absorption of vitamin B-12 into the blood. Dr. Doscherholmen will conduct his research at Veterans Hospital, which is affiliated with the University Medical School.

* * *

A member of the Naval Reserve, **Dr. L. E. Steiner,** of Albert Lea, has been called to Washington, D. C., for a tour of active duty. He reported to the Navy Bureau of Medicine. Dr. Steiner, who has been active in Albert Lea civic groups, has been secretary of the Freeborn County Medical Society for five years, member of the House of Delegates of the Minnesota State Medical Association and on the board of editors of MINNESOTA MEDICINE.

* * *

Making his first bid for public office is **Dr. T. J. Jensen** of Duluth. Dr. Jensen, who has practiced medicine in West Duluth for forty-four years, is a candidate for coroner. Dr. Jensen not only is active in a number of West End church and civic groups, but is a member of the St. Louis County Medical Society and the Minnesota and American Medical Associations. He is on the staff of St. Luke's, St. Mary's and Miller Memorial Hospitals.

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Training as a lieutenant at Great Lakes Training Station, Illinois, is **Dr. Leong Hom** of Battle Lake. Dr. Hom plans to return to his practice in Battle Lake after his tour. He has been in the Minnesota community since August, 1950.

* * *

Dr. Carl Lembke, Minnesota Lake physician and farmer, proved his farming prowess by taking high point honors in all horticulture-agriculture exhibits at the community's Festag celebration recently. Dr. Lembke exhibited grains, flowers, wildflowers and weeds at the annual Minnesota Lake agricultural festival.

* * *

"Local Care of Infantile Eczema" was the topic when **Dr. Lloyd S. Nelson** spoke to members of the Freeborn County Medical Association. The meeting was held August 19 in Albert Lea.

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A gift contribution to the St. Louis County Medical Library is Volume II of Clinical Roentgenology (Head, Neck and Spinal Column) by deLorimier, Moehring and Hannan. The donor was **Dr. Henry G. Moehring** of Duluth, a co-author.

Dr. Arthur H. Wells of Duluth attended the International Congress of Clinical Pathology in Washington, D. C. The group met September 6-10.

BLUE SHIELD—BLUE CROSS NEWS

At the annual meeting of the Minnesota State Medical Association in Duluth in June more than one hundred interviews were held with physicians by representatives of Blue Shield. Among the subjects that were most important to the physicians was the physician-patient relationship. Many were of the opinion that prompt and correct filing of Blue Shield claims and rapid processing of the patient's claim by Blue Shield lead to an improvement of the relationship between the doctor, his patient and Blue Shield.

An analysis of Blue Shield claims shows that prompt payment of claims is delayed by the lack of such information on the Medical Service Report as: the status of Workmen's Compensation, the patient's birth date, the relationship of the patient to the subscriber, the income of the subscriber, the date professional services were rendered, the dates of admission and discharge from the hospital, the doctor's charges for the services rendered and the signature and Blue Shield serial number of the attending physician.

Omission of these items requires obtaining additional information from the doctor's office and also results in delay in the payment of the claim by Blue Shield. Of greater importance is the fact that such delay can impair the doctor-patient relationship and also frequently leads to dissatisfaction between the physician, the patient and Blue Shield.

The Medical Service Report or claim blank is worded in such a manner as to reduce to a minimum the time and effort required for its proper completion.

The birthdate of the patient is frequently omitted by both doctor and subscriber but it is important in the correct payment of Blue Shield claims. For example, it is not possible to pay some claims such as those for tendon repair unless the age of the patient is known, due to the fact that Blue Shield allowances for tendon repair in children under five years of age are greater than those for persons of more advanced years. Other considerations such as the validity of a claim for a dependent are dependent upon the subscriber's birthdate.

Incorrect payment of a claim results from inaccuracies and incompleteness in that portion of the Medical Service Report set aside for stating the services rendered. With all services, accurate listing of the services performed together with the date the services were rendered is necessary to assure prompt and proper payment of claims.

Moreover, it is necessary that the doctor's name, address, and participating serial number as well as his signature be furnished at the bottom of the medical report. Lack of this information delays the payment of claims until each item is obtained. Furthermore, it is difficult to prepare and send checks without the correct spelling of the doctor's name and address.

Minnesota Medicine

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THE DIGNITY OF MEDICINE

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Saint Paul, Minnesota

THE HONOR of being president of the Minnesota State Medical Association is one that I never anticipated and never felt worthy of, and still I look forward to next year when this Association will pay me a greater honor and that is the opportunity to sit among those noble gentlemen, the past presidents of this Association—the men who did so much, who gave so much of their wisdom, their time and energy to make this, the Minnesota State Medical Association, one of the greatest, one of the finest, and one of the friendliest medical associations in these United States. These men have built the road so well that it has been very easy for me to follow in their footsteps. I wish to thank them, and may God bless them.

For several years past, there has been a tendency among the presidents to make their addresses shorter and shorter. I also shall be brief, but it is not our intention to condemn the presidential address to complete oblivion.

Having celebrated our centennial last year, we physicians may now feel more at ease, I suppose, in the face of history. Somehow, when some great event in our lives has been duly celebrated, we are inclined to relax and feel a little self-satisfied. However, we have shown that we are here to stay. More than that—we can look forward without fear, knowing that we can surmount the difficulties to come, just as we did the seemingly unsurmountable hazards of the past. Our inherited tradition, we are confident, will see us through these hazards. Let us hope that we are worthy of this tradition.

The dignity of medicine is what sets it apart from other professions, what creates confidence of patients in physicians and permits them to entrust their lives to our hands.

The dwelling place of the dignity of medicine is in the conduct of the men and women who practice medicine and also in those who are associated with them in the healing arts and professions. This dignity has deep roots. It goes back centuries. It revealed in many phrases of the ancient Hippocratic oath. For example, "Whatever houses I may visit, I will come for the benefit of the sick." And again, "In purity and holiness, I will guard my life and my art."

Everyone who comes in contact with physicians learns to recognize the dignity of medicine in its practitioners. It is something more than integrity, gravity, responsibility, knowledge, acumen or skill—though it partakes of all of them. I suspect that the dignity of medicine is closely akin to what we mean by the essential dignity of man.

Actually, a rather curious and uncomfortable parallel can be traced between those days of 1854 and this year of our Lord, 1954. Minnesota was enjoying a boom then and real estate men were revelling in the glories of getting rich fast. Physicians were arriving in haste on boats from the East to the new and promising territory, and just around the corner were hard times and war.

And, just around the corner for us here tonight may be hard times and war. We cannot be sure. Looking at the present-day situation from a broad view, it is not easy to be optimistic. For in the background and under the surface of this happy occasion, the sensitive listener and viewer might hear and see again the relentless

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Presidential Address delivered at the 101st Annual Meeting, Minnesota State Medical Association, Duluth, Minnesota, June 8, 1954.

march to a war more devastating than any of our fathers, or any of us for that matter, ever dreamed of.

But, we can hope sincerely that future generations will honor us justly for the steady courage and devotion which we use to lay a firm foundation for things they will take for granted—just as we revere and respect the foundation built for us by our own forebears.

Physicians can trespass upon the dignity of medicine when they maintain rigid attitudes toward the changes in medical practice and organization that must inevitably occur—and at a rapid tempo in our time. They trespass when—except in emergencies—they deny the team concept of medicine.

The art of medicine does not, however, depend solely on skill and knowledge. There is much still to be said for the presence of the physician as an element in the healing process. I recall to you statements made about a distinguished physician of an earlier generation, Sir William Osler.

His biographer said of him that he entered the sickroom "with an air of gaiety, an atmosphere that lifted the invalid out of his ills" and made half a ward forget its symptoms.

Likewise, I nurture a reasonable hope that they will have no cause to censure us for any failure to live up to the strong ideals handed to us from the past. The thought of such a failure becomes to us one of the greatest challenges we must ever face. The results of that failure would surely be more disastrous to the future of medicine and our society than most of war's great harm—so great is the value of this inherited foundation.

My own father was a physician and so were my grandfathers—a heritage of which anyone would be proud. But that is a background which is not uncommon in medicine. A strong thread of continuity of calling and duty runs unstinted through many generations of medical people. For that very reason, we physicians, as much as, if not more than, most men should feel a strong, personal, and intense sense of grave responsibility as we look in both directions—to the safe years of the past, and to the uncertain years of the future.

Recently, medicine has been the open target of criticism. Some of this criticism may be easily dismissed as uninformed and ill-natured. And yet, some of these criticisms, if considered with deep humility and carefulness, can show us clearly the direction in which we may have failed. Some of them are genuine grievances against some phase of medical practice, and it is only natural for the complainer to transfer his singular problem to a condemnation of the entire group.

As physicians, we can never afford to lose sight of the individuality and the personality of each and every patient we see. This is our tribute, paid daily, to the dignity of man. It is reflected back on us in the high regard that our patients and the public have for the ultimate dignity of medicine.

Somehow, I cannot believe that our inherited tradition has served us vainly. I have a deep and abiding faith in the high ideals so nobly advanced to us by our forebears. It is within our power to enliven, enrich and entrench that glowing tradition so firmly and deeply that the profession of a century hence will thank us for strength and courage, as we thank our forebears today.

We have reaped a fine harvest, we have been endowed with uncompromising responsibility, we have been given a near-sacred trust. We must not fail that trust—we must be faithful to it.

625 Lowry Medical Arts Bldg.

LAURELS FOR AMA FILM

Two honors recently were heaped on the popular documentary film, "A Citizen Participates," on which the AMA holds television rights. The film received top rating as an educational motion picture at the Cleveland Film Festival and was chosen by *Scholastic Teacher* for a national film award as a 16 mm information film. This film describes how members of a rural community can work together to get a physician.

AMA's exclusive TV rights to this film have been extended to December 31, 1954, so that only state and county medical societies may book the film for their local TV stations. After that date, however, it will still be available for 16 mm showings to church, school, club and similar gatherings. Bookings may be arranged through AMA's TV Film Library.

PITFALLS IN THE ROENTGEN DIAGNOSIS OF PULMONARY DISEASE

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I AM DEEPLY conscious of the responsibility of speaking to you on this occasion dedicated to the memory of Russell D. Carman. It is an honor I shall always cherish.

From what I have learned about Dr. Carman, I am sure there is no need for me to attempt to recall here his contributions to medicine. Those of you who knew him personally or were raised in his shadow in this part of the country can recount them far more effectively than I. Sympathetic understanding of the problems of others, a dedicated sense of justice, boundless energy, and above all, selflessness characterized the man for whom this honor lecture is named. In remembering Dr. Carman, one cannot help but remember the things he stood for. By so doing, we are afforded the opportunity to re-dedicate our lives to the ideals that were Dr. Russell D. Carman's.

Uncritical adherence to established principles can be dangerous, for it tends to stop progress. Equally true, in all walks of life "uncritical adherence" is particularly undesirable in medicine where the dogma of today often is frowned upon tomorrow. It is in this spirit that we have chosen to discuss some of the pitfalls that exist in the roentgen diagnosis of pulmonary disease.

As indicated by Rigler,[8] Newell,[6] Garland[4] and others,[1,7,9] the roentgen examination of the chest is beset with many difficulties and shortcomings. Yet, many continue to regard it as the last word in the recognition of pulmonary disease. Last word though it is, and highly informative as it is, all physicians must realize its limitations for it "is better to know you are wrong than hope you are right."

In the chest, as in all radiography, good films are of fundamental importance. It matters little how expert a radiologist may be, he cannot see what has not been imprinted upon his roentgenograms.

It is not within our province to discuss here the factors that play a part in good film quality. Suffice it to say, they encompass every possibility from the handling of the patient to the exposing and finally the processing of the film. Even the film viewing boxes cannot be overlooked.

In the chest, air is an important contrast medium. Infiltrates, effusions, or masses displacing normal air produce contrasts which are interpreted in terms of disease by the radiologist. Differences in air contrast occur normally in the chest with every breath. Indeed faulty aëration due to poor technique is a common source for error in this examination. Unless properly controlled abnormalities will be reported where none exist. Remarkable differences in the healthy chest can be produced deliberately by guided changes in pulmonary ventilation.

The ease with which lesions may be overlooked in films of poor technical quality is clearly demonstrated by the following example. Routine roentgenograms of the chest revealed a spherical mass 3 cm. in diameter in the paracardiac portion of the right lower lobe. Roentgenograms made with the patient slightly rotated failed to reveal the mass now hidden by the cardiac silhouette. Even more impressive is the obliteration of the soft tissue mass which followed poor film processing with over-developing.

As most roentgen findings are not pathognomonic, the radiologist constantly judges roentgen findings in the light of the patient's clinical picture. Consider diffuse pulmonary nodulation as an example.

Felson and Heublein[3] tabulated approximately eighty diseases capable of producing disseminated pulmonary changes. Among them, they included lesions due to aspiration, cystic pulmonary lesions, abnormalities due to noxious agents and hazardous dusts, diseases due to deposited foreign bodies, embolization, trauma, vascular abnormalities, bronchial abnormalities, abnomalities due to infections or infestations, allergy, pulmonary fibrosis, changes due to the hemopoietic system, sarcoid, malignancy and tuberculosis.

From the Department of Radiology, Hospital of the University of Pennsylvania, Philadelphia, Pa.

Russell D. Carman Memorial Lecture delivered at the annual meeting of the Minnesota State Medical Association, Duluth, June 7, 1954.

Faced with a differential diagnosis encompassing this host of diseases, the radiologist can be of little or no assistance as a consultant unless adequate clinical information is supplied him. Only then are the patient's best interests served, for the roentgen examination alone often proves meaningless. Witness the following examples; the first patient revealed generalized cardiac enlargement associated with multiple nodular lesions distributed throughout both lungs. Investigation revealed the patient had been a hard coal miner for thirty-five years; this patient had silicosis.

The next patient also revealed disseminated pulmonary nodulation radiographically. This individual, however, presented a history of rheumatic fever, which suggested that the roentgen changes were due to rheumatic pneumonitis. This seemed a reasonable conclusion, for these changes are known to occur in young adults in as high as 60 per cent of the patients with severe rheumatic fever.

Disseminated pulmonary mottling was found radiographically in still another individual. This patient, however, had gastrointestinal complaints. When a gastric cancer was identified radiograhically, logic dictated that the lung lesions were metastatic which was ultimately proven.

The next patient might have caused confusion had it not been known, clinically, that the patient revealed skin lesions, peripheral adenopathy, and conjunctivitis. With this history suggesting sarcoid, it seemed reasonable to conclude that the patient's pulmonary abnormalities were due to sarcoid also which proved to be the case.

This dependence upon adequate clinical information may also be demonstrated in patients with solitary masses.

The next patient had no clinical complaints. A mass 10 cm. in diameter was found, radiographically, in the paracardiac portion of the right lower lobe. Fluoroscopically, the mass change easily with changes in position. This change in configuration due to gravity is a common finding in patients with cysts or very soft tumors. For this reason, as well as others, the mass was considered a mesothelial cyst which it proved to be.

A mediastinal mass about 10 cm. in diameter caused substernal distress in another individual. Except for some encroachment upon the trachea and esophagus, nothing distinctive was noted fluoroscopically. One statement in the clinical history gave the clue to the diagnosis; the man had noted fullness in his breasts and some pigmentation of his nipples. This proved to be a choriocarcinoma of the mediastinum; this diagnosis, offered pre-operatively by the radiologist, proved to be correct and was possible only because adequate clinical information was available.

Another patient complained of weakness. Her roentgenograms revealed a small mediastinal mass arising in the carina which revealed no distinctive abnormalities. Clinically, this patient presented the classical findings of myasthenia gravis. With this clinical information, the diagnosis of a thymic tumor seemed logical; the diagnosis was confirmed at operation.

The tendency to ignore one part of the roentgenogram in favor of another is one of the most common pitfalls in radiology. Unless roentgenograms are studied systematically important information may be overlooked entirely. There is an old adage in radiology which says, "look at the four corners of the film." Witness the following which bear ample testimony to the truth of this adage.

The first patient had Hodgkin's disease. He had no complaints referrable to the shoulder. In the routine chest roentgenogram, however, a destructive lesion was found in the corocoid process of the scapula, which ultimately proved to be due to Hodgkin's disease.

The next patient had no complaints except weakness. Whereas, the chest was considered normal, a soft tissue mass was observed, radiographically, in the left supraclavicular fossa. This proved to be a group of lymph nodes which revealed lymphosarcoma. The possibility of supraclavicular lymph nodes was suggested in another chest examination; here the densities were artefacts caused by plaited hair.

The tendency to relax when things seem obvious, radiographically, is another common pitfall. The next patient was examined because of marked anemia. When a large esophagogastric hernia was found it was considered the cause for the anemia. Because the hernia, when full of barium, did not occupy the entire soft tissue mass demonstrated in the chest, the radiologist thought it would be advisable to examine this patient's colon.

The barium enema revealed an anular carcinoma in the transverse colon. Had the entire gastrointestinal tract not been examined, the carcinoma would have been overlooked and the hernia alone repaired.

Mistakes were also made in the next two patients because the roentgen findings seemed obvious. The first was examined soon after a severe automobile accident. The chest had been injured, and the patient was being examined for fractured ribs. Among other findings, the radiologist described a large abscess in the right lower lobe. The patient had been perfectly healthy until the time of the accident. The second patient presented a similar story. She, too, had been in an automobile accident and suffered serious chest trauma. In addition to the fractured ribs and a pneumothorax, the radiologist described an abscess in the right lower lobe and a second abscess in the left upper lobe. Here, too, there were no pulmonary complaints prior to the injury.

In both patients, the radiologist was wrong; both had pulmonary hemorrhages plus pneumatoceles which looked exactly like lung abscesses. Had the clinical histories been carefully evaluated when the patients were first examined, these mistakes would not have been made.

The next patient complained of early heart failure. The clinician thought there was a pleural effusion in the right hemithorax. Routine roentgenograms of the chest revealed no evidence of fluid in the right hemithorax; except for some elevation of the right hemidiaphragm the chest was considered normal. At the insistence of the clinician, the patient was fluoroscoped. Once again, nothing suggesting fluid was observed; the right hemidiaphragm was moderately elevated yet moved freely. On deep lateral bending, however, fluid appeared in the costophrenic sulcus. Routine lateral decubitus films then demonstrated a considerable quantity of fluid in the right hemithorax; 1000 cc. of straw-colored fluid were then removed.

The next patient also demonstrated an error which was the result of haste because something seemed obvious. This patient had no complaints except a slight superficial cough. A nodular density 3 cm. in diameter was observed in the left lower hemithorax; the patient was operated upon immediately for an early bronchogenic carcinoma. At operation, the density proved to be a healing fractured rib; the lungs were normal.

An error was made in the next patient too because things seemed obvious radiographically. The patient had had a breast removed. Routine roentgenograms of the chest revealed an interstitial air and fluid collection in the region of the mastectomy. The latter being brilliantly visualized attracted the radiologist's attention; a pneumothorax in the underlying lung which was 20 per cent collapsed was overlooked.

In differential diagnosis, the element of time plays an important part. Sometimes it is misleading as in the following twenty-four-year-old woman in whom a 5-centimeter spherical mass was found during a chest survey examination. Four years later the same lesion was identified; this time, for the first time, the patient was told what was found. The lesion not having changed in size during the four-year interval and because it was totally asymptomatic, it was difficult to advise the patient concerning its surgical removal. The patient chose to have the nodule removed; it proved to be a well-encapsulated liposarcoma.

The time factor also played a part in the next individual who, for years, had had chest roentgenograms semi-annually because he worked in a tuberculosis sanatorium. Five months following his interval examination, a round parenchymal infiltrate was found in the left lower lobe. Considered cancer because of the patient's age, its nodular character, and its sudden appearance, operation was performed immediately. The lesion proved to be a tuberculoma.

The next patient also worked in a tuberculosis sanatorium. He, too, was examined at regular intervals and, for years, revealed an apparent stable calcific density in the left upper lobe. After many years, the calcific density began to increase in size; it was thought that this patient was reactivating an old tuberculous process. In spite of adequate treatment, the lesion continued to grow larger. When operated upon, this proved to be a bronchogenic carcinoma engrafted upon an old calcified tuberculous lesion which still was healed.

No discussion of pitfalls in the diagnosis of pulmonary disease would be complete without mention being made of the hazards attending the roentgen diagnosis of pulmonary emphysema. Too often is this diagnosis made because the lungs seem hyperaerated. Suffice it to say that the diagnosis of pulmonary emphysema cannot be made on the basis of a single film alone. Of paramount importance in the diagnosis is fluoroscopy. Unless one can demonstrate fluoroscopically (or radiographically) limited motion of the domes of the diaphragm plus some fixation of the costal cage, the probabilities are that the patient does not have emphysema. Hyperaeration as a normal finding is by no means uncommon in glass blowers and wind instrument players.

Worth recording at this time are the possible advantages derived from the use of body section radiographic techniques; advantages which help reduce potential pitfalls in the diagnosis of pulmonary disease. With experience and with better appreciation of the many physical factors that play a part in body section radiography, it is becoming increasingly common to identify by means of this technique pulmonary lesions which might otherwise have been overlooked entirely. The evidence indicates that the body section examination more closely approximates the true size of the lesion than routine roentgen examinations. It is only fair to state, however, that many body section units may not be as effective as they might be for reasons which do not concern us here.

Attention should also be called to advantages that may be derived from the use of new image amplification tubes of the type developed by several x-ray manufacturers. These image-amplifying instruments reveal more brilliantly the detail in certain segments of the chest, the hila particularly, which has proven a distinct advantage. With it, minor abnormalities in the dynamics of the hilar structures have been revealed.

One of the inponderables in the interpretation of chest roentgenograms is the mental attitude of the radiologist. On some days, he tends to "overread," with minor roentgen changes assuming major importance. On other days, he is more optimistic and tends to "underread" what he sees. This change in mental attitude was studied carefully by Birkelo[2] and his group who submitted roentgenograms of the chest in a series of 1256 patients for interpretation to two radiologists and three chest specialists. They demonstrated a real variation in film interpretation between individuals (inter-individual) and between multiple readings of the same individual (intra-individual). From this study, it was concluded that all survey roentgenograms and mass survey work should be read independently by at least two interpretors and all persons whose roentgenograms were interpreted as positive or suggestive for tuberculosis by one or the other interpretors should be recalled for further study. The degree of inter-individual variation in interpretation, for positive cases only, by the five experts was from 5 to 25 per cent. The intra-individual or personal variations for multiple readings were from 3 to 31 per cent. Birkelo's observation has been substantiated by others;[4] thus we are now aware of the fact that survey procedures although good are susceptible to a human error of about 20 per cent.

Lest the medical profession become too critical of its radiological confreres, attention should be called to the fact that other surveys similarly controlled have revealed similar discrepancies. In a study of 221 children, five expert pediatricians were asked to classify them according to their nutritional state. When their study was completed, which included allied diagnostic procedures, there were only seven patients (3 per cent) in whom all five examiners agreed.

In 1944, we began a clinical survey of the chest using roentgen examinations made a little before and immediately after death which were then compared with roentgenograms of the same patient's lungs removed and inflated at autopsy which later were cut and studied grossly and histopathologically.[5]

Our findings in these comparative postmortem studies of the chest may be summarized as follows:

1. As Rigler[8] and Newell[6] predicted, we often failed to identfy the small lesions under 3 mm. in diameter. Particularly was this true of pleural and subpleural nodules along the lateral chest wall and over the domes of the diaphragm. Similar observations have been made at operation; that is, pleural lesions were seen and subpleural nodules were felt, which were not demonstrable on preoperative roentgenograms.

2. Occasionally, we overlooked much larger lesions due to an error on our part.

3. Our greatest concern was the lung lesions we saw in roentgenograms of the excised lungs which we never were able to demonstrate in our original roentgenograms. Stated differently, even though we knew the size and nature of a pulmonary mass from our postmortem roentgenograms and pathologic studies there were occasions when

the lesion was never demonstrated roentgenographically in our original roentgenograms made immediately before death, before the chest was opened.

The following explanations have been suggested for these discrepancies. Some shadows are completely subliminal, below the threshold of visibility. Many lesions occur in the relatively blind areas of the chest which include areas along the parietal pleura, close to ribs, and in the midline where the spine and mediastinum obscure them.

Dr. William Tuddenham,[10] working in our department, has demonstrated that the probability of one seeing a lesion in a chest radiographically appears to be significantly limited by the following factors:

1. The low relative brightness contrast of some images which usually are those superimposed upon bone and mediastinum.

2. The glare and psychological distraction of large bright images which usually are the calcified structures.

3. The random variation in film density associated with the use of fluorescent screens.

Since two of these factors are related to the bright images cast by calcified structures it seemed reasonable to try to reduce contrast by going to the higher kilovoltages where the absorption of x-rays in bone relative to that of soft tissues is greatly reduced. Accordingly, roentgenograms of the chest made at 1,000 kv. were then compared with roentgenograms of the same individual's chest using routine factors at 80 kv. Lucite spheres of varying sizes were attached to the patient's back over the spine and lung fields to test the ability of each technique to record small tissue lesions in these areas.

In the supervoltage film, (1,000 kv. and 2,000 kv.), sphere as small as 6 mm. in diameter were visible in both the lung fields and the mediastinum. The larynx, trachea and the main stem bronchi were delineated, and the lung markings were fairly well recorded with this higher kilovoltage technique. In the control study, too, spheres as small as 6 mm. in diameter were visible in the lung fields, but no objects less than 40 mm. in diameter were seen in the mediastinum and only a portion of the trachea was recorded.

Thus, it would seem, from these observations that the use of supervoltage radiation in chest radiography offers distinct advantages over conventional radiography because it permits us to record on the same film, and with almost equal sensitivity, the soft tissues of the lung fields and of the mediastinum. Unfortunately, however, supervoltage radiography has some disadvantages which do not seem to be insurmountable. The dose the patient receives is still too high. Also, the time required for each exposure is still too long. However, there is the hope that further research in this field will make it possible for us to push farther forward our diagnostic horizons insofar as the chest is concerned and, by so doing, reduce still further our potential pitfalls and opportunities for error.

References

1. Birkelo, C. C. and Brosius, W. L.: Roentgen visualization of pulmonary arterial circulation in autopsy material. Radiology, 31:261-292 (Sept.) 1938.
2. Birkelo, C. C., Chamberlain, W. E., Phelps, P. S., Schools, P. E., Zaks, D. and Yerushalmy, J.: Tuberculosis case finding: comparison of effectiveness of various roentgenographic and photofluorographic methods. J.A.M.A., 133:359-366 (Feb. 8) 1947.
3. Felson, H. and Heublein, G. W.: Some observations on diffuse pulmonary lesions. Am. J. Roent. Genol., 59:59-81 (Jan.) 1948.
4. Garland, L. H.: On the scientific evaluation of diagnostic procedures. Radiology, 52:309-328 (March) 1949.
5. Greening, R. R., and Pendergrass, E. P.: Postmortem roentgenography with particular emphasis on the lung. Radiology, 62:720-724 (May) 1954.
6. Newell, R. R. and Garneau, R.: Threshold visibility of pulmonary shadows. Radiology, 56:409-415 (March) 1951.
7. Resink, J. E. J.: Is a roentgenogram of fine structures a summation image or a real picture? Acta Radiol., 32:391-403, 1949.
8. Rigler, L. G.: Possibilities and limitations of roentgen diagnosis. Am. J. Roentgenol., 61:743-761 (June) 1949.
9. Robbins, L. L.: X-Ray diagnosis of plmonary lesions. New England J. Med., 239:779-786 (Nov. 18) 1948.
10. Tuddenham, W., Pendergrass, E. P., Hale, J.: Supervoltage diagnostic roentgenography. Am. J. Roentgenol., 70:759-765 (Nov.) 1953.

THE EFFECT OF CHEYNE-STOKES RESPIRATION ON PROGNOSIS

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Accurate prognosis is usually the most difficult, treacherous, obscure component in the total evaluation of the condition of a patient. It requires accurate diagnosis and thorough knowledge of the natural history of the disease, of the value of treatment, and of the effect of new developments on the course of a patient's illness. In many instances, prognosis is at best an educated guess, but careful consideration of it is necessary, as it frequently colors the physician's approach to a patient and his illness.

Prognosis may be altered by the spectacular and easily recognizable occurrence of Cheyne-Stokes respiration. As ancient and well investigated a pathologic respiratory condition as this is, it suffers from the label of being a "terminal" event.[11,15] Such a label in itself merits thorough investigation.

The classic papers of Cheyne[2] and Stokes[13,14] are excellent descriptions of the phenomenon known today as Cheyne-Stokes respiration. Many studies have searched for the basic mechanism of such periodic respiration. Eyster, in 1906, stressed a circulatory mechanism. He increased intracranial pressure in dogs and found that when the arterial blood pressure rose and fell alternately above and below the intracranial pressure, Cheyne-Stokes breathing ensued. The studies of Douglas and Haldane, in 1909, emphasizing a chemical mechanism, are still accepted with only slight modification. They proposed that Cheyne-Stokes respiration developed in the following way. Initially, the respiratory center in the medulla is depressed. Weak, shallow respirations result producing hypoxia. Hypoxia stimulates respirations which increase in vigor, but subside as carbon dioxide is eliminated; apnea ensues. Apnea results in hypoxia and prevents carbon dioxide elimination; the center is stimulated and breathing returns; the cycle repeats itself. The excessive loss of carbon dioxide during hyperpnea removes its stabilizing influence upon the respiratory center. (This is compared to a fly-wheel on a motor.) Normally, large carbon dioxide stores in lungs and tissue fluids oppose any sudden fall in blood carbon dioxide tension. Tissue fluids buffer any sharp increase in carbon dioxide tension. Reduced bicarbonate reserve in Cheyne-Stokes respiration permits slight changes in gas tension to produce exaggerated fluctuations in the respiratory center.

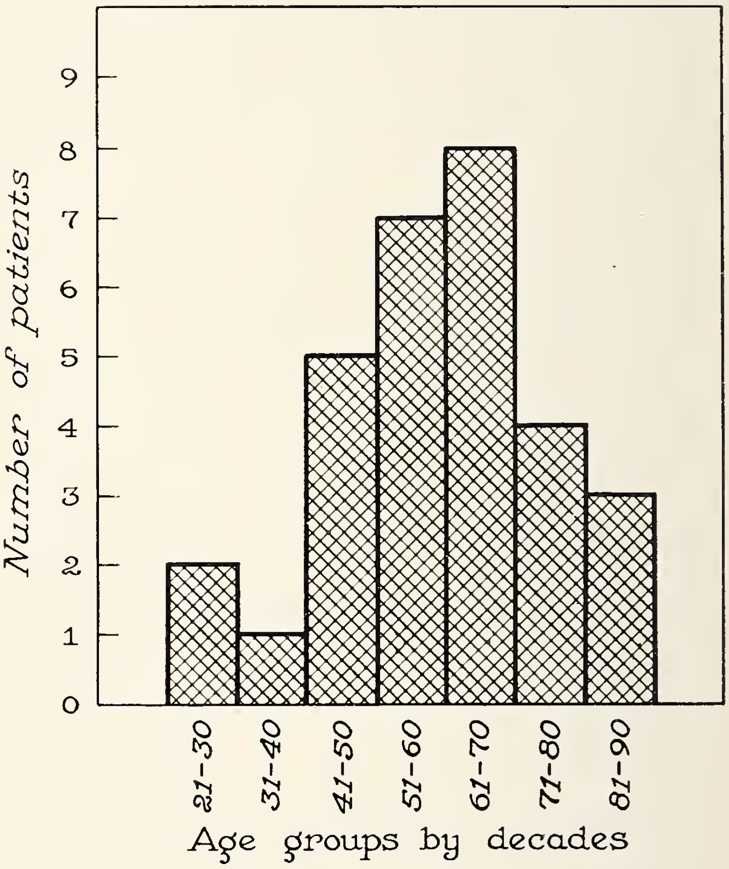

Fig. 1. Ages of thirty patients.

In the 1930's, further clinical[1,3,16] and experimental[7] investigations substantiated Douglas and Haldane. At this time also, the therapeutic use of aminophylline drew the attention of many writers.[6,8,9,12] A more recent paper by Pryor stressed both the chemical and circulatory factors. From his cases he concluded that with marked increase in circulation time, overventilation or apnea can continue for a long period before altered blood can arrive at a sensitive area to correct the situation. While the respiratory center is responding to this blood, the blood in the lungs is being altered in the opposite direction. Once started, periodic breathing is self-perpetuat-

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TABLE I. FATAL PROGNOSIS PREVAILINGLY UPHELD

Case	Age, Yr.	Sex	Fate	Time Followed After Onset of "Cheyne-Stokes"	Basic Disease				Aminophylline Used?	Comment
					Cardiac	Vascular	Cerebral	Renal		
1	60	M	Died	1 day			+		No	Subarachnoid hemorrhage due to ruptured aneurysm.
2	88	M	Died	1 day	+		+		No	Cerebral embolism secondary to myocardial infarction.
3	65	M	Died	1 day			+		Yes, with benefit	Cerebrovascular accident, cancer floor of mouth.
4	55	M	Died	1 day		+	+		No	Hypertension, cerebrovascular accident.
5	69	F	Died	1 day	+		+		No	Multiple cerebral emboli secondary to myocardial infarction.
6	87	F	Died	2 days			+		No	Cerebral infarction, pulmonary emboli, hip fracture.
7	63	F	Died	1 day	+	+		+	No	Kimmelstiel-Wilson's disease with uremia, hypertensive and coronary disease.
8	24	M	Died	1 day	+			+	No	Chronic nephritis, uremia and pulmonary edema.
9	26	M	Died	3 days		+		+	No	Hypertension group IV with uremia.
10	81	M	Died	4 days	+			+	No	Chronic nephritis, uremia, myocardial infarction.
11	46	M	Died	4 days		+		+	Yes, with benefit	Hypertension group IV with uremia.
12	65	M	Died	5 days		+		+	Yes, with benefit	Hypertension group IV with uremia.
13	40	M	Died	6 days				+	No	Acute carbon tetrachloride poisoning with uremia.
14	55	F	Died	9 days		+		+	Yes, with benefit	Kimmelstiel-Wilson's disease, uremia and hypertension.
15	73	F	Died	14 days			+		No	Cerebrovascular accident.
16	70	M	Died	15 days			+		No	Metastatic cancer of cerebellum, primary in cecum.
17	68	M	Died	5 weeks	+	+			Yes, with benefit	Coronary and hypertensive heart disease with heart failure; probable intracranial arteriosclerosis.
18	77	F	Died	2 months	+		+		No	Multiple cerebral emboli secondary to mitral stenosis.
19	43	M	Dismissed; home	7 days				+	No	Chronic diffuse nephritis, impending uremia.
20	68	M	Dismissed; home	3 days	+	+			Yes, with benefit	Hypertension group IV with heart failure.

ing because of the delay in communicating events in the lungs to the respiratory center.

This study was undertaken to elaborate on and to evaluate further the gloomy prognosis "terminal" attached to Cheyne-Stokes respiration, and to discover, if possible, information pertaining to the total status of patients with that respiratory mechanism.

Material

The records of about 300 seriously ill patients of the Mayo Clinic, hospitalized in 1950, were reviewed. No attempt was made to conduct a complete canvass of all hospitalized patients; however, special emphasis was placed on patients with vascular, cardiac, cerebrovascular and renal disease. Thirty patients were found to have had Cheyne-Stokes respiration at some time in the course of their illness. It is this group which we studied.

Data

In the group of thirty patients with Cheyne-Stokes respiration, men outnumbered women in the proportion of 22 to 8 and ages varied from twenty-four to eighty-eight years, averaging 60.2 years. The distribution by age is shown in the figure.

A summary of the patients is provided in Tables I and II. Patients reported in Table I upheld the standard prognostic implication that Cheyne-Stokes respiration is a "terminal" or "late" development in the course of a disease. Those reported in Table II tend to refute that view, as their course seemed to be unaffected by the appearance of periodic respiration.

Discussion

A glance at the preceding tables makes certain points clear. These points are:

1. From cases 1 to 6, 15 to 17, 21 to 23 inclusive: When cerebral disease was complicated by the appearance of Cheyne-Stokes respiration, three patients survived the episode whereas nine did not.

2. From cases 7 to 14 inclusive, nineteen: When Cheyne-Stokes respiration developed in the presence of severe, progressive renal insufficiency, the situation was essentially terminal. One such patient survived the episode but eight did not.

3. From cases 21 to 25 inclusive: If the patients' illness consisted of reversible cerebral or renal embarrassment, the prognosis was not adversely affected by the presence of Cheyne-Stokes respiration.

4. From cases 26 to 30 inclusive: If Cheyne-

TABLE II. FATAL PROGNOSIS PREVAILINGLY REFUTED

Case	Age, Yr.	Sex	Fate	Time Followed After Onset of "Cheyne-Stokes"	Basic Disease				Aminophylline Used?	Comment
					Cardiac	Vascular	Cerebral	Renal		
21	71	M	Well	3 days			+		No	Insulin reaction; probable intracranial arteriosclerosis.
22	59	M	Living with hemiplegia	8.5 months		+	+		No	Hypertension, cerebrovascular accident.
23	48	F	Living with hemiplegia	27 months			+		No	Cerebrovascular accident.
24	56	F	Well, asymptomatic	3 weeks				+	No	Nephrolithiasis, pyelo-nephritis, renal insufficiency.
25	76	M	Well	4 months				+	No	Transurethral resection, lower nephron nephrosis.
26	56	M	Well, asymptomatic	5 weeks	+	+			Yes, with marked benefit	Early hypertension group IV, with acute left heart failure.
27	45	M	Living with symptoms	3 months	+	+			Yes, with marked benefit	Hypertension group IV, pneumonitis, heart failure.
28	64	M	Died of heart failure	10 months	+	+			Yes, with benefit	Hypertension group III with acute heart failure.
29	48	M	Living, asymptomatic	5 weeks	+	+			Yes, with marked benefit	Hypertension group II, heart failure, toxic psychosis.
30	60	M	Living with symptoms	5 weeks	+	+			Yes, with benefit	Hypertension, paroxysmal tachycardia, pericarditis.

Stokes respiration appeared in association with hypertensive cardiovascular disease, but in the *absence* of severe cerebral or renal damage, the prognosis apparently was not worse than before the Cheyne-Stokes respiration appeared.

5. Eleven of thirty patients received aminophylline. All appeared to be benefited by it. Whether or not such treatment has any influence on prognosis is equivocal.

Again, these results are not reported as being conclusive. They are more in the nature of guideposts in the difficult question of prognosis in the seriously ill.

With further clinical and experimental study of Cheyne-Stokes respiration, it is hoped that more insight into its basic mechanism may be had. Perhaps the original observations of Cheyne and Stokes will be even more valuable in future direction of clinical prognosis and treatment.

Summary

In the course of the illnesses of thirty patients Cheyne-Stokes respiration developed. Nineteen (63.3 per cent) of the patients died. The prognosis was worse when this phenomenon developed in cases of severe cerebral or severe renal disease than when it developed in cases of cardiac disease. We believe that, when Cheyne-Stokes respiration occurs, one is justified in giving a grave prognosis but not necessarily a fatal one.

References

1. Anthony, A. J., Cohn, A. E. and Steele, J. M.: Studies on Cheyne-Stokes respiration. J. Clin. Investigation, 11:1321-1341 (Nov.) 1932.
2. Cheyne, J.: A case of apoplexy, in which the fleshy part of the heart was converted into fat. M. Classics, 3:705-709 (Mar.) 1938-1939.
3. Comeau, W. J.: Cheyne-Stokes respiration and auriculoventricular conduction. New England J. Med., 219:977-982 (Dec. 22) 1938.
4. Douglas, C. G. and Haldane, J. S.: The causes of periodic or Cheyne-Stokes breathing. J. Physiol., 38:401-419 (June 15) 1909.
5. Eyster, J. A. E.: Clinical and experimental observations upon Cheyne-Stokes respiration. J. Exper. Med., 8:565-613 (Oct. 12) 1906.
6. Forster, D. E.: Cheyne-Stokes respiration: Theophylline-ethylenediamine in treatment. Northwest Med., 38:172-173 (May) 1939.
7. Harrison, T. R., Calhoun, J. A., Cullen, G. E., Wilkins, W. E. and Pilcher, C.: Studies in congestive heart failure. XV. Reflex versus chemical factors in the production of rapid breathing. J. Clin. Investigation, 11:133-154 (Jan.) 1932.
8. Marais, O. A. S. and McMichael, J.: Theophylline-ethylenediamine in Cheyne-Stokes respiration. Lancet, 2:437-440 (Aug. 21) 1937.
9. Nathanson, M. H. and Fitzgibbon, J. P.: Pharmacology of Cheyne-Stokes respiration. Am. Heart J., 17:691-700 (June) 1939.
10. Pryor, W. W.: Cheyne-Stokes respiration in patients with cardiac enlargement and prolonged circulation time. Circulation, 4:233-238 (Aug.) 1951.
11. Purves-Stewart, James: The Diagnosis of Nervous Diseases. Ed. 9, pp. 139, 737. Baltimore: The Williams & Wilkins Company, 1945.
12. Stannus, D. G. and Roche, C. F.: Aminophylline in Cheyne-Stokes respiration: Report of a case in coronary disease. J. Florida M. A., 28:489-491 (Apr.) 1942.
13. Stokes, William: Observations on some cases of permanently slow pulse. M. Classics, 3:727-738 (Mar.) 1938-1939.
14. Stokes, William: Fatty degeneration of the heart. M. Classics, 3:739-746 (Mar.) 1938-1939.
15. Wechsler, I. S.: A Textbook of Clinical Neurology: With an Introduction to the History of Neurology. Ed. 6, p. 318. Philadelphia: W. B. Saunders Company, 1947.
16. Wendkos, M. H. and Robertson, H. F.: Treatment of bronchospastic dyspnea and Cheyne-Stokes respiration with iodized caffeine. Internat. Clin., 2:68-78 (June) 1941.

DEVELOPMENT OF A SIMPLIFIED FILTER PAPER ELECTROPHORESIS WITH A STUDY OF HUMAN SERUM PROTEINS

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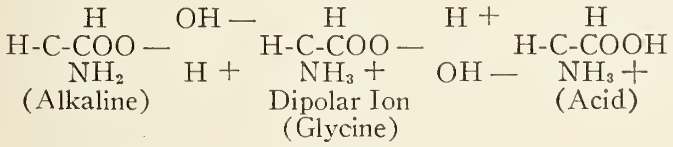
> "Electrophoresis may be defined as the migration of colloid particles under the influence of an electric field. The speed and direction of the movement of the particles is dependent upon the free charges on the surface of the particles."[14]

IN 1807, the Russian physicist, Reuss, observed that when electricity was passed through glass tubing containing water and clay, colloidal clay particles moved toward the positive electrode.[21]

Hardy, in 1899, reported that negatively charged particles of denatured egg albumin could be made positive by the addition of acid and that with the change from alkaline to acid the particles migrated in the electric field in the opposite direction. "The proteid particles, therefore, have this very interesting property that their electrical characters are conferred upon them by the nature of the reaction, acid or alkaline, of the field. If the latter is alkaline the particles become electronegative; and vice versa."[9]

In the case of proteins, the change from electronegative to electro-positive with the addition of acid can be explained by the existence in proteins of free amino groups ($-NH_2$) and free carboxy groups ($-COOH$), as shown in the equation, using the amino acid glycine as an example:



$$\underset{\text{(Alkaline)}}{\text{H-}\overset{\text{H}}{\underset{\text{NH}_2}{\text{C}}}\text{-COO}-}\quad \overset{\text{OH}-}{\underset{\text{H}+}{}}\quad \underset{\text{Dipolar Ion (Glycine)}}{\text{H-}\overset{\text{H}}{\underset{\text{NH}_3+}{\text{C}}}\text{-COO}-}\quad \overset{\text{H}+}{\underset{\text{OH}-}{}}\quad \underset{\text{(Acid)}}{\text{H-}\overset{\text{H}}{\underset{\text{NH}_3+}{\text{C}}}\text{-COOH}}$$

Since proteins are made up of many amino acids joined together, the phenomenon of dipolarity applies to them as well as to amino acids. For each amino acid and protein molecule there is a definite hydrogen ion concentration (pH) at which the free negative charge balances the free positive charge. This particular pH is called the *isoelectric point,* and the net electrical charge is zero. The addition of acid causes a shift in this balance to a preponderance of positive charge, and the addition of base a shift toward dominance of negative charge.

Since these basic observations, many electrically charged biological materials have been studied by the electrophoretic principle. There are, at present, three different electrophoretic methods, namely—the microscopic method, the moving boundary method, and zone electrophoresis.

In the microscopic method, particulate material such as red blood cells[1] and bacteria,[23] for example, are suitable for study. They are composed of protein and, therefore, exhibit an electrical surface charge. Of course, in addition to a charged particle, a second requisite is a solution that conducts electricity. Distilled water is a poor conductor of electricity but the addition of a pinch of salt, acid, alkali or other ionizing compound (known as an electrolyte) renders it conducting.

In this method, particles suspended in electrolytic solution and contained in a glass tube are placed on the stage of the microscope and their behavior when subjected to an electric current simply observed microscopically.

There are, however, many colorless substances of submicroscopic size and of biological importance which cannot be studied electrophoretically by simple observation. For their study more sensitive equipment is required.

When Arne Tiselius developed a new apparatus which permitted a quantitative electrophoretic study of protein mixtures, a new era in the study of proteins was initiated.[24] This technique was termed the moving boundary method by Tiselius because it relied upon measuring optically the index of refraction between the colloid materials and the supernatant liquid. Thus, when a colloid, or protein mixture is introduced into one end of a U-tube made of optical glass, and current is applied, the colloid mixture is arranged in a series of moving boundaries in the supernatant liquid, their position depending on their electric charge. A delicate optical device measures the

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index of refraction of each of the separated colloid materials as they move past the measuring point, and the quantity of each constituent may be calculated from the curve obtained.

In Cushing's syndrome a typical protein pattern was found.[15] The plasma albumin and gamma-globulin were greatly decreased, while the alpha$_2$-globulin was elevated.

Fig. 1. This demonstrates the type of zoning produced by filter paper electrophoresis, the five major protein fractions are well deliniated. It can be seen that the darkest zone farthest from the original starting point (indicated by the pencil line) is albumin. Then alpha$_1$, alpha$_2$, beta and gamma globulin follow in that order. Azocarmine dye was used in staining, but this dye has not proved satisfactory for quantitative studies.

With this costly and delicate device, Tiselius was able to show for the first time that normal human serum protein consists of albumin, alpha$_1$, alpha$_2$, beta, and gamma globulins, and not simply albumin and globulin, as had been demonstrated by earlier chemical methods.[24]

For this important discovery, Tiselius was awarded the Nobel prize. Research workers the world over used this new tool in the study of colloids, particularly the proteins, and an enormous number of papers presenting important new information on the nature of proteins were published.[2] In medicine, human serum and other body fluids were extensively studied, and many interesting findings were reported. We shall mention only a few by way of example.

Longsworth, Shedlousky and MacInnes[18] found a very high beta-globulin value in the electrophoresis of plasma from nephrotics, as well as a lowered albumin and gamma-globulin. Alpha-globulin was found to be high. Examination of the protein of nephrotic urine demonstrated that the beta-globulin elevation so evident in the nephrotic plasma is not present in the urine. The nephrotic urine protein rather resembles that of normal human serum.[16]

In multiple myeloma, some of the most bizarre protein patterns have been obtained by electrophoretic study.[11,20] Four types of protein changes have been noted, all involving a large component of abnormal protein, which is located in a variable position in relation to the other protein fractions, apparently due to its variable mobility when subjected to an electric current.

In the early days of moving boundary electrophoresis, it was hoped that the method would be useful in many diseases as a specific diagnostic tool. With the exception of the above-mentioned diseases, as well as certain liver diseases,[8] this has not been the case. The stage to which any given disease has progressed at the time of the study and the severity of the disease process appears to affect the electrophoretic pattern significantly.[14]

In addition to its use in the specific diseases mentioned, the electrophoretic method is useful for studying the progressive changes in proteins during the course of many other diseases, and in following changes resulting from therapy. In addition, information concerning the changes in the various protein fractions is much greater than that obtained by standard chemical fractionation methods.[14]

A serious impediment to the more widespread use of this new technique in clinical medicine has been the great expense of the delicate Tiselius-type of equipment used in standard moving boundary techniques.

Recently, a simpler and less expensive method has been developed which promises to allow studies similar to those undertaken with the moving boundary method.[7]

This technique has been called zone electrophoresis because instead of moving through a free electrolyte solution, as in the moving boundary method, the colloidal matter migrates through a porous solid material which is saturated with an electrolyte to permit a current to pass through it.

When the migration has been completed and the various proteins separated, the solid material is dried and the isolated proteins are deposited in zones where they are accessible for further study, if desired. Starch columns, filter paper, or other similar porous, non-reactive material may be used as a supporting medium (Fig. 1).

Our interest in zone electrophoresis was recently aroused because we felt that if a simple technique could be worked out, zone electrophoresis might become a useful addition to the clinician's armamentarium in the study of those diseases producing alterations in human serum proteins.

The technique employed was basically developed by Kunkel and Tiselius, using filter paper as the supporting medium.[13] After reading this and other procedural literature on filter paper electrophoresis,[22] we found that a great deal of technical experimentation was necessary to finally work out a method which, in our hands, would enable us to produce quantitative results consistant with themselves and with moving boundary findings reported in the literature. Once the following method had been worked out, however, it became very simple to operate the equipment and produce dependable results.

The apparatus used is shown in Figure 2. This was assembled at a total cost of around fifty dollars. Pre-assembled zone electrophoresis instruments of a slightly different type, are now available from scientific houses at a higher price.

Power is obtained from a Heathkit variable voltage regulator which delivers the desired voltage to the apparatus by means of small platinum electrodes immersed in the buffer solution contained in the battery jars. The filter paper, previously moistened with electrolyte solution, is dipped into each jar of electrolyte solution and covered with glass plates to prevent evaporation. The current is thus conducted by the electrolyte solution from one platinum electrode through the filter paper to the second platinum electrode. The basic idea is that the protein mixture added to the filter paper is then exposed to a controlled voltage which causes the various protein fractions to migrate at different velocities (depending primarily on their surface charge).

When migration has proceeded so that all the fractions are separated sufficiently for analysis, the current is shut off, and the proteins are deposited in separate zones on filter paper where they are available for further study.

In order to achieve precise results considerable, attention must be paid to technical detail. For this reason, we shall present our method in detail.

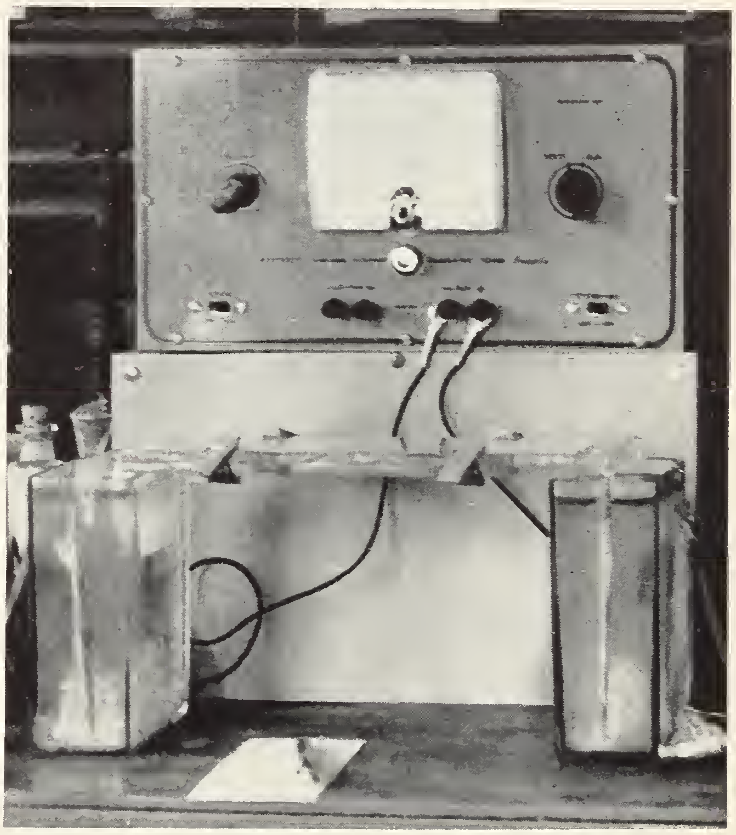

Fig. 2. The simplified filter paper electrophoresis apparatus.

The electrolyte buffer solution is prepared by dissolving 1.86 grams of diethyl barbituric acid and 150 grams of sodium diethyl barbiturate (both U.S.P. grades) in distilled water to make one liter of solution. This provides a constant pH of 8.8.

The two battery jars (each 6 x 2½ x 6 inches) are filled with this solution. Each jar is divided by a glass plate, but a thin slit is left at the bottom to permit passage of the current from the platinum electrode through both chambers of the jar. Glass wool may be packed into this slit. Splitting the jars in this way prevents serious changes in pH from electro-osmosis as the equipment is used. Beckman pH meter checks showed that after fifty-four hours of use the pH change in these compartments does not exceed 0.2 units. We are now changing the current direction with each "run" and have had no difficulty with pH changes.

A variety of filter paper has been tried, including Whatman 3 MM, 11, and 2. Of these, Whatman 2 has proved most satisfactory in our experience. The paper size found most convenient for our work is 9 x 36 cm., but size is not essential.

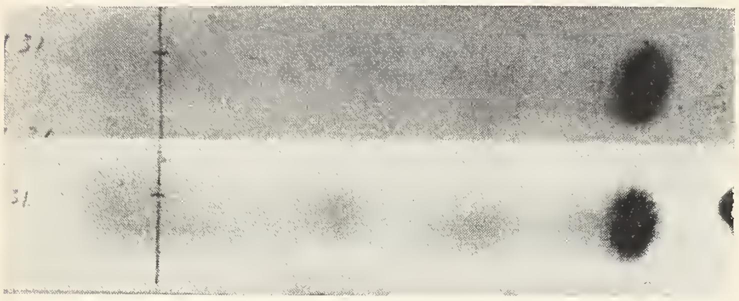

Fig. 3. The upper chromatogram was obtained using two-day-old serum. The lower was obtained using the same serum after fourteen days of refrigeration. No essential change is seen. The five major zones are not as well delineated as with the azocarmine dye used in Figure 1, but accurate quantitative results are obtainable from this chromatogram.

In setting up the equipment for use, a pencil line is drawn at the mid-point of the paper. It is then dipped into the barbital buffer. The excess buffer solution is removed by careful blotting. The paper is then placed on top of one of the plate-glass plates which must be thoroughly polished with silicone grease. This glass plate rests on the edges of the battery jars, and is a few centimeters wider than the paper and about 10 cm. shorter. About 5 cm. of each edge of the paper is immersed in the electrolyte solution in the chambers of the battery jars.

Exactly five lambdas (0.005 ml.) of serum is applied to the mid-point of the paper on the previously made pencil mark with an ultra-micro pipette. The second glass plate, the same size as the first, and similarly polished with silicone grease, is carefully placed over the paper. The long edges of the plates are sealed with additional silicone grease to prevent evaporation. Steel spring clips or clothes pins are clamped over the edges to maintain the seal at the edges.

In our earlier work, a drop of dilute brom phenol blue dye was added to the serum before subjecting it to electrophoresis. This stains the fastest moving albumin fraction and the migration may then be followed visually.

We have found that serum specimens may be kept for weeks without significant changes in the protein fractions, provided the red cells had been promptly removed and the same kept refrigerated (Fig. 3).

TABLE I. COMPOSITION OF BLOOD SERUM PROTEIN BY PAPER ELECTROPHORESIS

Effect of Variations in Staining, Fixing, and Washing.

	Stain	Fixing with ½% Acetic	Washing	A/G
1	Tiselius	4 rinses in 20 min.	None	0.85
2	Tiselius	4 rinses in 20 min.	None	0.64
3	Durrum	Soaked for 5 min.	None	0.70
4	Durrum	5 min. in ½% HAc	5 min. in cold H_2O	2.30
5	Durrum	5 min. in ½% HAc	5 min. in CH_3OH	1.86
6	Durrum	3 min. in ½% HAc	10 min. in H_2O	1.72
7	Durrum	3 min. in ½% HAc	10 min. in CH_3OH	1.88
8	Durrum	None	10 min. in H_2O	2.25
9	Durrum	None	10 min. in CH_3OH	2.21
10	Durrum	None	10 min. in H_2O	1.13
11	Durrum	None	10 min. in CH_3OH	1.72
12	Durrum	1 min. in ½% HAc	3-5 min. in CH_3OH	1.53

Chemical analysis of the serum used in the above tests by micro-Kjeldahl gave the following:

Non-protein Nitrogen =32 mg./100 ml.
Total Serum Protein =7.0%
Albumin =4.8%
Total Globulins =2.2%
A/G =2.18

Note: For reasons explained elsewhere in the paper, the chemical and electrophoretic A/G ratios are not the same, the latter being lower. The technique which produced the best results, and which was subsequently used, is number 12.

Three or four drops may be run simultaneously, since the spread of drops of this size is only about 10 to 12 mm.

After the serum samples are added and the plates sealed, a ten-minute wait is allowed to permit the buffer solution to be absorbed by and to saturate the filter paper.

The current is then applied. We have found that the voltage is not critical, and have been employing 350 volts. With this potential, the albumin fraction moves at a rate of about 2.5 cm. per hour. The albumin is migrated through a distance of 9 to 10 cm., requiring between three and one-half to four hours. This permits a clear-cut separation of the protein fractions, even of the alpha$_1$ globulin and albumin fractions, which tend to remain closely related because of a similar mobility.

With this voltage and paper size, the amperage ranges from 10 to 15 milliamperes at room temperature. Cooling is not essential, but if a higher temperature develops, as is the case with Whatman 3MM paper, an ice tray atop the glass plates provides adequate cooling.

At the end of the "run," the ends of the paper are cut off. The plates are carefully separated by resting them vertically on the table and prying them apart rapidly with a sharp knife blade to prevent smearing of the protein spots.

After the protein fractions are separated on the paper, the very critical matter of drying, fixing, and staining is carried out.

We have tried the methods recommended by Kunkel and Tiselius,[13] Schneider and Wallenius,[22] and Durrum,[5] and have found that with these techniques the globulin portions are abnormally high when compared with standard methods (Table I).

After much trial and error, a procedure was settled upon which does produce acceptable results.

The disengaged paper is first mounted horizontally on a wooden frame with thumb tacks and placed in the electric oven at 100° C. for five minutes. The dried paper is them immersed in Durrum's stain[5] for five minutes. Then the stained paper is fixed by immersion in a 0.5 per cent acetic acid solution for one minute. Next the wet paper is washed with pure methanol until the stain is removed completely from the protein-free portion of the paper, which usually requires from three to five minutes. The paper is then allowed to dry at room temperature.

With this method of staining, fixing, and washing, quantitative results consistent with themselves and with standard quantitative chemical methods are obtained. Table II shows a comparison between our zone electrophoretic analyses and standard micro-kjeldahl chemical analyses carried out on fourteen different human serum specimens. The ratio obtained by dividing the zone electrophoretic A/G by the chemical A/G compares very favorably with the accepted ratio obtained using the standard moving boundary method, namely, 0.68 and 0.72, respectively. The chemical A/G is higher than the electrophoretic A/G because in the chemical separation of albumin from the globulins, some of the globulins remain be-

TABLE II. SOME ALBUMIN-GLOBULIN RATIOS OBTAINED BY THE DYE ELUTION METHOD COMPARED WITH VALUES FOUND BY STANDARD CHEMICAL ANALYSIS

Sample Number	Dye Elution Method Relative Composition in Percent			Chemical Method Percent Protein		
	Albumin	Globulins	A/G	Albumin	Globulins	A/G
1	51.7	48.3	1.07	4.2	3.0	1.40
2	58.5	41.5	1.41	4.4	2.2	2.00
3	56.3	43.7	1.29	4.8	2.2	2.18
4	30.3	69.7	0.42	2.0	3.2	0.62
5	27.6	72.4	0.38	2.9	4.1	0.71
6	44.0	56.0	0.79	2.6	2.9	0.89
7	59.1	40.9	1.45	3.8	2.3	1.67
8	37.7	62.3	0.61	3.6	3.0	1.20
9	45.7	54.3	0.84	3.3	2.1	1.57
10	53.9	46.1	1.18	4.5	2.9	1.58
11	50.0	50.0	1.00	3.9	2.7	1.44
12	36.0	64.0	0.56	2.6	3.4	0.76
13	44.3	55.7	0.80	6.4	5.6	1.14
14	44.2	55.8	0.79	3.9	3.4	1.26
Average			0.90			1.32

$$\frac{\text{(A/G) Electrophoretic}}{\text{(A/G) Chemical}} = \frac{0.90}{1.32} = 0.68$$

$$\frac{\text{(A/G) for Free Moving Boundary Electrophoresis}}{\text{(A/G) for Chemical Precipitation Method}} = \frac{1.45}{2.00} = 0.72$$

TABLE III. COMPARATIVE ABSORPTION OF BROM-PHENOL BLUE BY PURIFIED HUMAN ALBUMIN AND PURE HUMAN GAMMA GLOBULIN

	Optical Densities	
Drops	Albumin	Gamma Globulin
No. 1	0.422	0.390
No. 2	0.485	0.406
No. 3	0.458	0.398
No. 4	0.442	0.426
No. 5	0.523	0.458
No. 6	0.295	0.481
No. 7	0.478	0.416
No. 8	0.542	0.455
Average	0.443	0.429

Protein concentration of albumin = 2.81%.
Protein concentration of Gamma globulin = 2.73%.

$$\text{Correction Factor for Difference in Concentration} = \frac{0.0273}{0.0281}$$

$$\text{Brom Phenol Index} = \frac{0.443}{0.429} \times \frac{0.0273}{0.0281} = 1.03$$

NOTE: This table shows the similarity in the absorption of dye by human serum albumin and human gamma globulin. The albumin absorption of the dye is 3% greater than dye absorption by gamma globulin, which may be considered negligible for ordinary work. The eight drops used for each protein fraction varied in size, but in each series of drops the total quantity was 40 lambdas. The percentage concentration of protein was determined by the standard micro-kjeldahl technique. A number of other determinations were made, all of which were similar to the findings shown here.

hind with the albumin, whereas this does not occur during electrophoresis.

The dye elution method mentioned in Table II is an accepted quantitative technique used to measure the amount of dye absorbed by the various protein fractions spectrophotometrically, and thus, indirectly, the amount of protein.[10,13]

TABLE IV. RELATIVE COMPOSITION OF BLOOD SERUM PROTEIN IN NORMAL AND SOME PATHOLOGICAL CONDITIONS OBTAINED BY PAPER ELECTROPHORESIS

Number	Relative Composition in Per Cent Globulins						Diagnosis
	Albumin	Alpha$_1$	Alpha$_2$	Beta	Gamma	A/G	
K-36A	39.4	6.8	9.0	21.8	22.8	0.65	Maternal Blood
K-36B	56.6	6.1	7.1	11.1	18.9	1.30	Cord Blood
K-33A	44.3	7.8	14.9	16.0	17.2	0.79	Maternal Blood
K-33B	59.1	5.6	8.0	9.9	17.3	1.45	Cord Blood
K-39A	50.5	5.2	11.4	15.5	17.1	1.02	Maternal Blood
K-39B	60.0	6.2	7.7	11.2	14.8	1.54	Cord Blood
K-41A	40.7	5.9	9.4	19.2	24.7	0.68	Maternal Blood
K-41B	57.8	3.4	4.0	7.8	26.9	1.34	Cord Blood
K-43A	49.5	7.6	10.8	16.5	15.4	0.95	Maternal Blood
K-43B	59.0	6.0	8.8	9.1	16.9	1.43	Cord Blood
K-44A	51.2	6.7	10.7	13.2	18.0	1.02	Maternal Blood
K-44B	63.4	3.9	6.0	6.7	19.9	1.61	Cord Blood
K-45A	45.8	5.7	9.1	21.9	17.2	0.86	Maternal Blood
K-45B	54.7	7.1	7.5	13.1	17.4	1.21	Cord Blood
K-46A	58.4	5.0	9.2	18.3	9.1	1.40	Maternal Blood
K-46B	61.2	5.3	6.6	10.6	16.2	1.58	Cord Blood
Cohn*	59.1	6.0	9.1	14.0	11.8	1.44	Normal
K-32	58.5	5.0	6.0	11.0	19.3	1.41	Normal
MAK	56.3	4.4	6.0	17.3	15.9	1.29	Normal
WK	58.6	5.4	8.6	12.5	14.8	1.42	Normal
K-34	53.9	5.9	7.9	12.3	19.9	1.18	Pregnancy—3 mo.
K-35	50.0	7.3	10.9	17.2	14.3	1.00	Pregnancy—at term
K-28	44.2	6.6	8.8	21.5	18.2	0.79	Pregnancy—8 mo.
H-10	54.9	5.7	7.9	16.5	15.3	1.19	Pregnancy—4 mo.
K-42	46.9	5.2	8.4	18.4	20.9	0.88	Pregnancy—8 mo.
H-16	51.1	6.4	9.9	18.3	14.0	1.05	Pregnancy—8 mo.
H-18	56.3	4.8	10.3	15.6	12.8	1.29	Pregnancy—7 mo.
H-23	49.0	6.0	9.5	19.2	16.1	1.03	Pregnancy—3 mo.
H-24	34.1	5.8	13.5	14.6	31.9	0.52	Pregnancy—? mo.
H-25	49.2	5.6	13.3	14.4	17.3	0.97	Pregnancy—8 mo.
H-26	43.7	5.8	9.3	22.3	18.8	0.77	Toxemia in Preg.
H-6	36.0	5.5	10.7	10.2	37.4	0.56	Rheum. Arthritis
K-26	44.3	5.1	12.4	11.0	27.0	0.80	Acute Rheum. Fever
H-20	27.9	4.0	5.0	7.9	55.1	0.39	Multiple Myeloma
U-1-45	23.4	7.5	12.7	21.3	34.0	0.21	Hepatitis
H-6	36.0	5.5	10.7	10.2	37.4	0.56	Arthritis
H-9	36.6	10.2	10.4	13.4	29.2	0.58	Cancer of Liver
H-5	27.6	4.7	13.0	20.6	34.0	0.38	Cirrhosis
Average	47.5	6.3	10.6	17.8	17.7	0.92	Maternal Blood
Average	59.0	5.4	7.0	10.0	18.5	1.43	Cord Blood
Average	48.5	5.9	9.9	17.3	18.1	0.97	Pregnancies

*Adapted from the data of Cohn, Oncley, et al.; J. Clin. Invest., 23, 417 (1944).

The stained zones on the paper are cut off with scissors into 1 cm. sections, the dye eluted with 2 ml. of saturated sodium carbonate solution in 50 per cent methanol, and the dye intensity measured spectrophotometrically at 595 millimicrons.

One problem that arises when employing this method is that with some methods differential dye absorption by the protein fractions occurs. Thus, investigators have reported quite a wide variety of "factors" used to compensate for the lower dye binding power of the globulins.[6,10,22] Using our method of fixing and staining, however, we have found that no such factor is required for the globulins.

In addition to obtaining results comparable to accepted moving boundary figures, as shown in Table II, we employed our fixing and staining technique using pure human serum albumin and purified human gamma globulin, after subjecting this material to electrophoresis in the usual manner. These stained zones, or spots, were analyzed as described above. This gave us a factor of only 1.03 for the globulins, which is a difference of only 3 per cent and may be disregarded for practical purposes (Table III).

Table IV is a summary of the zone or filter paper electrophoretic determinations which we have performed on a variety of clinical material.

Serum taken from the placental cord was compared with maternal serum. The individual results are shown in the upper part of the table, and the averages are at the bottom. This work agrees with similar studies using moving boundary methods,[17] and demonstrates that maternal serum contains significantly higher amounts of alpha$_2$ and beta globulin fractions, but appreciably less gamma globulin than the fetal serum.

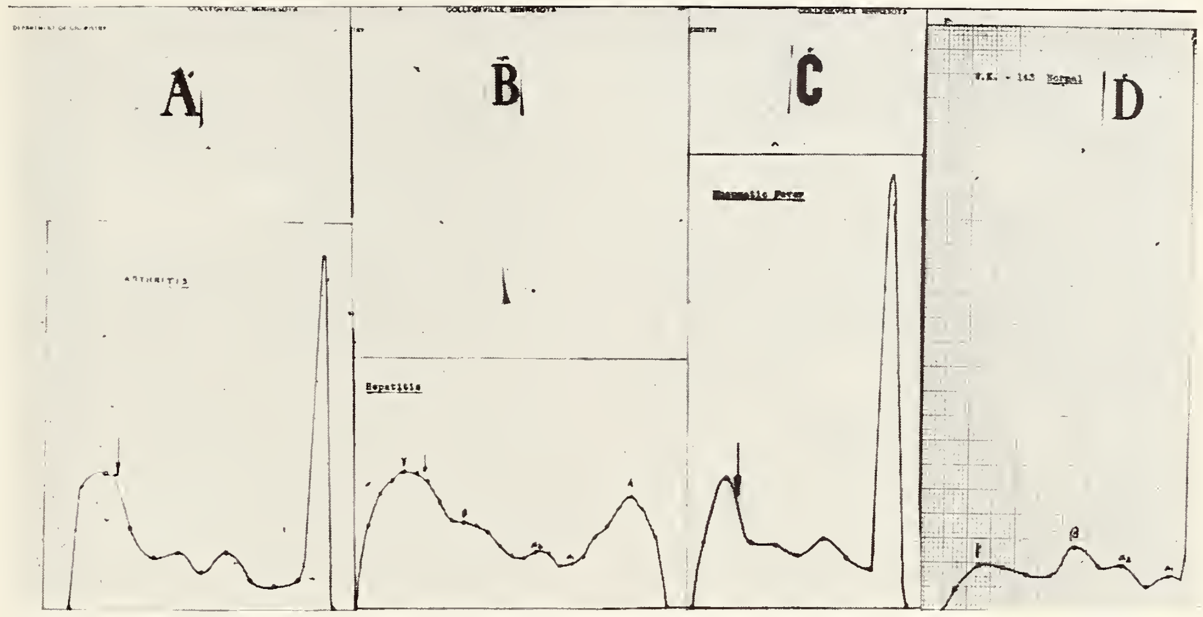

Fig. 4. Graphs plotted from filter paper electrophoresis data and analyzed quantitatively by the dye elution method. The areas under the curve are quantitative. A shows the pattern obtained from a case of rheumatoid arthritis. B represents the protein distribution in a case of hepatitis, being a marked deviation from the normal. C demonstrates serum from a patient with acute rheumatic fever. D is a normal pattern for comparison.

A number of pregnancy cases were also studied and, as expected, were essentially the same as maternal serum drawn immediately after delivery. These cases show, on the average, an elevated beta globulin when compared with the normal values.

A group of normal values are also shown in Table IV. The values adopted from Cohn are the accepted normal values for moving boundary electrophoresis. The remaining data in the normal grouping consist of values obtained using blood drawn from individuals judged as clinically normal, or in good health.

Interestingly enough, K-32, one of the authors, shows an elevated gamma globulin and a slightly decreased alpha$_1$-globulin alpha$_2$-globulin, and beta-globulin. This individual had a mild upper respiratory infection about ten days prior to the acquisition of the serum specimen. It is well known that the gamma globulin fraction is elevated even in minor infections and commonly remains elevated for some time after clinical evidence of disease has disappeared.[7]

This case also illustrates the fact that the standard chemical determination of the serum proteins in this case would not show this variation because the total globulin value would be normal, as would the albumin and the A/G ratio.

The last section of the table shows a random sampling of some clinical cases in which the diagnosis was made by other clinical and laboratory methods. These cases show variations from normal as have already been reported by moving boundary methods,[3,4,19] and also illustrate, we believe, the possible value of the simplified and inexpensive filter paper electrophoretic method of serum protein analysis in clinical medicine's armamentarium.

Curves showing the distribution of the serum protein fractions can easily be drawn from data acquired by the filter paper electrophoresis method. These curves show graphically and conveniently the relative protein composition of any given serum specimen, and results are commonly expressed in this manner when the moving boundary method is employed. Fig. 4 shows a number of curves prepared from our data to illustrate this.

Bibliography

1. Abramson, H. S. and Moyer, L. S.: Electrical charge of mammalian red blood cells. J. Gen. Physiol., 19:601, 1936.
2. American Instrument Company: Electrophoresis Bibliography (Feb.) 1954. This volume contains thousands of electrophoretic references.
3. Dole, V. P. and Rothbard, S.: Electrophoretic

changes in the serum of a patient with rheumatoid arthritis. J. Clin. Investigation, 26:87, 1947.
4. Dole, V. P., Watson, R. F., and Rothbard, S.: Electrophoretic changes in the serum protein patterns of patients with scarlet fever and rheumatic fever. J. Clin. Investigation, 24:648, 1945.
5. Durrum, E. L.: A microelectrophoretic and microionophoretic technique. J.A.C.S., 73:2943-48, 1950.
6. Fabian Lionetti and M. Moira Davison: Comparative analysis of serum proteins by chemical and electrophoretic means: an experiment in biochemistry. J. Chem. Education, p. 378 (July) 1954.
7. Gray, G. W.: Electrophoresis. Sci. American, p. 45-53, (Dec.) 1951.
8. Gray, S. J. and Barron, E. S. G.: The electrophoretic analysis of the serum proteins in diseases of the liver. J. Clin. Investigation, 22:191, 1943.
9. Hardy, W. B.: On the coagulation of proteid by electricity. J. Physiology, 24:296, 1899.
10. Hawk, P. B., Oser, B. L., and Summerson, W. H.: Practical Physiological Chemistry, p. 417. Philadelphia: The Blakiston Company, 1947.
11. Kekivich, R. A.: The serum proteins in multiple myeloma. Biochem. J., 34:1248, 1940.
12. Knapp, E. L. and Routh, J. I.: Electrophoretic patterns of plasma from normal and sick children. Federation Proc., 6:267, 1947.
13. Kunkel, H. G., and Tiselius, A.: Electrophoresis of proteins on filter paper. J. Gen. Physiol., 35:89-118 (Sept.) 1951.
14. Lewis, Lena A.: Electrophoresis in Physiology. (Quotation page 4) Springfield, Illinois: Charles C Thomas, 1950.
15. Lewis, L. A., and McCullough, E. P.: Plasma protein pattern (Tiselius electrophoretic technique) in Cushing's syndrome. J. Clin. Endocrinol., 7:559, 1947.
16. Longsworth, L. G. and McInnes, D. A.: Electrophoretic study of nephrotic sera and urine. J. Exper. Med., 71:77, 1940.
17. Longsworth, L. G., Curtiss, R. M., and Pembroke, R. H., Jr.: The electrophoretic analysis of maternal and fetal plasma and sera. J. Clin. Investigation, 22:46, 1945.
18. Longsworth, L. G.; Shedlousky, T., and MacInnes, D. A.: Electrophoretic patterns of normal and pathological blood serum and plasma. J. Exper. Med., 70:399, 1939.
19. Malmros, H. and Blix, G.: The plasma proteins in cases with high erythocyte sedimentation rate. Acta Med. Scandinav., 170:280, 1946.
20. Moore, D. H., Kobat, E. A., and Gutman, A. R.: Bence-Jones proteinemia in multiple myeloma. J. Clin. Investigation, 22:67, 1943.
21. Reuss, F. F.: Mem. Soc. Imp. des Naturalistes de Moscou, 2:327, 1809.
22. Schneider, G. and Wallenius, G.: Electrophoretic studies on cerebral spinal fluid proteins. Scand. J. Clin. & Lab. Invest., 3: No. 2, 1951.
23. Shaughnessy, H. J. and Criswell, K. I.: Studies on salt action. The influence of electrolytes upon the visibility and electrophoretic migration of Bacterium coli. J. Gen. Physiol., 9:123, 1925.
24. Tiselius, A.: A new apparatus for electrophoretic analysis of colloid mixtures. Tr. Faraday Soc., 33:524, 1937.

PLACEBO THERAPY

The most pointed occasion when the physician must decide whether or not to lie is in the case of the patient with incurable cancer. As individuals, physicians are reluctant to lie, but as physicians we must maintain an elasticity of attitude. Plato, who dwelt at some length on the deportment of physicians, wrote, "A lie is useful only as a medicine to men. The use of such medicines should be confined to physicians." Parkinson embodied a strong defense of the physician's integrity in his succinct observation, "The facts are that in clinical science there is devotion to truth and conformity to scientific standards as scrupulous as anywhere, but in practicing the art truth has often to be softened." Incidentally, as Henderson suggested, what the doctor believes to be true sometimes isn't; the patient may thereby be misinformed, albeit unwittingly on the part of the doctor. Also according to Henderson, what the doctor actually says is not in itself important, but rather what the patient comprehends and what it does to him. A physician can usually evade a possibly harmful truth and still satisfy his patient. Such an evasion is on practical grounds preferable to an unequivocal lie, because a lie can be uncovered, and if this should happen, the physician will lose the patient's confidence and respect. If the patient is insistent, there is, of course, no alternative to the truth. Hippocrates, whose precepts are widely considered to be as good today as they were nearly 2,500 years ago, wrote as follows: "Naught should be betrayed to the patient of what may happen or of what may eventually threaten him, because many patients have been driven in this way to extreme measures." In general, honesty is the best policy, subject to modification according to circumstances. This question in the case of incurable cancer is still moot, and being tangential to the present discussion will not be considered further.

Returning to the consideration of deception as applied in medical practice, I will state that I believe deception is completely moral when it is used for the welfare of the patient. If this is admitted, who then is to decide what constitutes the patient's welfare? Omniscience would be an attribute desirable in any individual responsible for the welfare of another. Being human, however, the physician lacks this quality, but by the very nature of his position, is the best arbiter of this question when it concerns a patient. Therefore, when the patient's welfare dictates the use of a placebo there can be no detrimental reflection on the physician who prescribes it. In fact, the physician who in an appropriate situation refuses to order a placebo, implying in effect that "I can't help you because there is no medicine for your disease," is cruel and is surely not to be praised for his morality.—ALAN LESLIE: Ethics and Practice of Placebo Therapy, Am. J. Med., 16:854-862, June, 1954.

FLUID AND ELECTROLYTE BALANCE

Part III: Medical Considerations

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INTRAVENOUS fluid therapy is of great value and often is lifesaving, but should be used only when oral feeding isn't possible. When it is used indiscriminately, it can also be detrimental and even result in unwarranted fatality. It is important, therefore, to review specific indications and contraindications.

There are certain general rules which were alluded to in the introductory article[4] and which will be discussed in more detail now. A chart such as the one reproduced in Table I is desirable. The arithmetic of intake and output of fluids, a record of weight daily or more often (a weight on admission to the hospital is as important as recording the body temperature), the separate recording of volume of fluid loss from fistula or drainage, blood and urine chemical determinations and other special items depending on the individual are simplified and clarified by using a chart. This chart can be modified to suit the emergency. In diabetic acidosis, for instance, columns for urine tests for sugar, acetone, diacetic acid and insulin can replace the columns for Na Cl and K output.

The record need not be kept in full detail for simple problems, but for patients with diabetic acidosis, metabolic alkalosis, anuria, low salt syndrome in heart disease, or acute dehydration of dysentery, a chart is definitely indicated. A fair approximation of the electrolyte loss from various sources can be made from the information collected by Randall[9] and recorded in Table II.

From this table it is evident that whereas sodium and chloride are lost in considerable amounts in gastrointestinal fluids, potassium losses are not great by this route. Therefore, it should be emphasized that the major avenue of loss of potassium is the kidney. The loss in a sick patient is greater than it is in a normal healthy subject. There is no way of estimating potassium loss in urine short of determining it, but this is rarely necessary in clinical practice.

A person in normal health at rest has a fluid balance about like this:

	Output		*Intake*
Urine	1500	Liquids	1500
Insensible water loss from skin and lungs	750	Water of food	500
Stool	100	Water of oxidation	350
	2350		2350

Note that water from food roughly balances the water lost by stool and insensible loss from skin and lungs; thus the actual water intake is roughly equal to the urine output. When the subject has a fever or is ill in any way, this will vary because the insensible loss will increase and food intake often decreases. Visible sweating obviously increases the fluid and salt loss. Pure water depletion occurs when the patient is unable to get water (lost on a desert or at sea) or is unable to swallow for various reasons such as coma or carcinoma of the esophagus. Thirst is a protective symptom to regulate water intake and prevent simple water depletion. Holmes and Montgomery[5] have studied thirst mechanisms in many illnesses including diabetes insipidus, shock and congestive heart failure. The rate of salivary secretion is a good measure of true thirst versus excessive water drinking. Unfortunately, thirst is not present and therefore not a useful guide to need for water in patients who have the most severe forms of dehydration, namely, those associated with electrolyte depletion. Fluids should be administered by slow drip intravenously unless otherwise specified. Subcutaneous administration should be abandoned entirely.[13]

The following discussions of specific disturbances may seem to be too rigid for some and not specific enough for others, but they are only intended as a general guide to the order of magnitude of therapy needed. It must also be clearly understood that the separate discussions relate to average situations and not to all possible variables. The discussion is not an indorsement of any particular commercially available ionic solutions. Some ready-made polyionic solutions are

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TABLE I. A SAMPLE CHART FOR RECORDING FLUID AND ELECTROLYTE DATA

Time or Date	Wt.	Output						Intake								Chemistries					
		Urine	Other Loss	Total	Na	K	Cl	Oral	Other	Total	Na+	K+	Cl−	$\overline{HCO_3}$ or Lactate	Plasma or Blood	B.U.N.	Na+	K+	CO_2	Cl−	Hbg.

TABLE II. MEAN CONCENTRATIONS OF IONS IN MEQ./1. WITH RANGE OF VALUES IN TWO-THIRDS OF CASES IN PARENTHESIS[9]

	Na+	K+	Cl−
Gastric juice	59 (31- 90)	9.3 (4.3-12)	89 (52-124)
Bile	145 (134-156)	5.2 (3.9-6.3)	100 (83-110)
Pancreatic juice	141 (131-153)	4.6 (2.6-7.4)	77 (54- 95)
Small bowel (suction tube)	104 (72-128)	5.1 (3.5-6.8)	99 (69-127)
Ileum (suction tube)	116 (91-140)	5.0 (3.0-7.5)	105 (82-125)
Ileostomy (recent)	130 (112-142)	16.2 (4.5-14)	110 (93-122)
Cecostomy	80 (48-116)	20.6 (11-28)	48 (35- 70)

alluded to and can be used to advantage under certain circumstances. Because of the recent availability of ion concentrates in ampule form, tailor-made solutions permit greater flexibility than any standard solution for the handling of complex problems.

A few general rules about the safe administration of potassium will be given now since they should be incorporated into the plan of therapy whenever potassium salts are given. An adequate urine flow must be established. In adults potassium solutions should not be given at a rate faster than 20 mEq. per hour. Too rapid administration can cause arrhythmias and even cardiac standstill. The solution should not have more potassium than 80 mEq. per liter and preferably not more than 40 mEq. per liter. Plans should be made so that the total potassium dose for 24 hours be distributed throughout all the fluids to be given for that 24-hour period unless specific indications dictate different policy (v.i. diabetic acidosis).

Diabetic Acidosis

The gravest of all medical emergencies involving fluid therapy is diabetic acidosis. It is important, of course, to be certain that the diagnosis of diabetic acidosis is correct and that the comatose or stuperous state of the diabetic patient is not due to insulin reaction, stroke, or other severe illness. Urinalysis, blood sugar, blood urea nitrogen, and carbon dioxide content (or combining power) of plasma must be determined immediately. The presence of mild acidosis with normal carbon dioxide concentration but with the appearance of ketone bodies in the urine requires the use of more insulin but not any parenteral fluid therapy. Evaluation of the severity of associated conditions and arrival at a calculated severity index is of considerable value.[15] Insulin therapy, of course, is the most important single element in recovery from diabetic acidosis, but insulin dosage will not be discussed in detail because it is beyond the scope of this paper. However, mention should be made of the order of magnitude of the dose. Give a total dose of 300-1000 units in the first day. When blood sugar is below 800 mg. per 100 ml. and acidosis is moderately severe, give 50-100 units initially (half of dose intravenously in severe cases). Repeat this dose every half hour for four doses. In very severe cases and those with blood sugar over 800 give 100-200 units every half hour for six doses. Subsequent doses have to be determined by serial blood sugar determinations and urinalyses.

Butler and co-workers[2] actually determined the losses of electrolytes during a period of insulin withdrawal. The actual and derived data indicate that the water losses approximate 11 per cent of body weight, and suggest a loss per kilogram of 5 mEq. of sodium, 4 mEq. of chloride, 6 mEq. of potassium of which 3 mEq. corresponds to the loss of nitrogen, 70 mg. of phosphorus of which 60 coincides with the loss of nitrogen. This information forms a cornerstone for therapy.

The most urgent need initially is for water and salt. Many patients do not need any alkali therapy, for the kidney will sort out the needed ions from sodium chloride solution. It is sometimes highly desirable to give alkali when acidosis is particularly severe. Occasionally in terminal stages the pH is so low and the respiratory center so exhausted that Kussmaul breathing is not evident. In such extreme instances quick increase of blood pH may be lifesaving. It appears to the author to be more reasonable to give sodium bicarbonate than to give sodium lactate early, for the latter has to be metabolized in order for the sodium to become available. (Later on in therapy sodium lactate is an equally satisfactory source of sodium.) Potassium should not be given immediately because the serum concentration is usually high or high normal at the time of admission in spite of total body deficiency. If an adequate urine flow has been established, potassium therapy should be started four to six hours after initial saline and insulin infusion is begun. If 0.9 per cent sodium chloride solution is used exclusively to correct dehydration, the concentration of chloride ion rises above normal so there is a temporary state of "chloride" acidosis.[11] This empirical observation added to the knowledge of the nature of electrolyte deficit makes it highly desirable to give sodium in excess of chloride.

Glucose should be given as soon as the blood glucose approaches normal. There is no need to give any glucose earlier than this. Initial glucose administration is harmful by perpetuating glucosuria and hyperglycemia. The need for glucose is clear, when the blood sugar approaches normal, for additional insulin usually is needed to correct the remaining acidosis.

The following scheme of fluid therapy has been found to be quite satisfactory in the order given:

1. 1000 ml. distilled water with 50 mEq. of sodium chloride and two ampules of sodium bicarbonate or 89 mEq.
2. 1000 ml. distilled water with 100 mEq. of sodium chloride and one ampule of sodium bicarbonate or 44 mEq.
3. 1000 ml. of Travert No. 2® with one ampule of potassium chloride (20 mEq./ampule) or Ionosol K with invert sugar® or 1000 ml. of 5% dextrose in water with 50 mEq. of sodium chloride and 40 mEq. of potassium phosphate.
4. Repeat No. 3.
5. 1000 ml. of 5% glucose in water with 40 mEq. of potassium chloride.

By this time, oral fluids are usually tolerated. If not, more fluids of the type in 5 are given. The first two liters should be given rapidly over the course of three hours. Subsequent fluids should be given more slowly according to general precautions taken with potassium solutions. This amount of fluid gives more sodium and chloride and less potassium than the average deficit indicated, but it does approach the requirement and replenishes the volume of extracellular electrolytes and fluids lost reasonably well. Tailor-made solutions using ion concentrates offer further refinement of therapy. Deficit of potassium and phosphate will be corrected more gradually but is partially corrected in the first day. Magnesium deficit usually can be corrected simply by diet.

Special conditions alter this program. When there is an associated congestive heart failure, coronary thrombosis, or serious renal insufficiency, the rate of infusion and type of therapy have to be modified. Anuria in diabetic acidosis poses a special problem. In the first place it is advisable to replace all the fluid which has been lost according to standard calculation. However, potassium should not be given unless serious signs develop (by electrocardiogram or respiratory paralysis) and then only in a dose of 40 mEq. until urine flow is established. Once hydration has been accomplished the same rules apply as for anuria in general (v.i.).

As soon as acidosis is corrected the patient should be given food, first as liquids and then graduated to a full diet as tolerated.

Metabolic Alkalosis

The consequences of pyloric obstruction are serious and may cause death unless the obstruction is relieved after correction of the associated metabolic alkalosis. As opposed to a relatively sudden onset of diabetic acidosis this comes on so insidiously that it is often not suspected. There are few clear-cut signs pointing to alkalosis. The most important information is the story of profuse and persistent vomiting. Patients with obstructed duodenal ulcer, though, are often so used to vomiting that they will not mention vomiting without specific questioning. The only safe procedure is to obtain a carbon dioxide content (or combining power) and chloride concentration of serum on every patient with a history of recent vomiting. If alkalosis is found, serum sodium and potassium concentrations and a blood urea nitrogen are indicated procedures. Alkalosis of long standing can cause serious renal damage.[1] The pathogenesis of alkalosis is dependent on the loss of hydrochloric acid (i. e. Cl^- loss in excess of Na^+) and on potassium depletion, both of which augment each other. Potassium depletion without loss of gastric fluid results in metabolic alkalosis[3] of all degrees of severity and can be corrected only by administering potassium salts. Sodium chloride solutions will not correct severe instances of chronic alkalosis because sodium further displaces intracellular potassium and perpetuates or actually increases the severity of the alkalosis. Ammonium chloride alone will not correct alkalosis due to potassium deficiency.

The following general rules will help correct metabolic alkalosis.

1. Each day replace the volume of emesis or gastric drainage material with a solution containing sodium chloride and ammonium chloride in slightly hypotonic concentration. Such a solution can be made by adding 50 mEq. of sodium chloride and 50 mEq. of ammonium chloride to 1000 ml. of glucose in water or by mixing 2 parts of Travert No. 3® or ionosol G.® with 1 part of glucose in water.
2. Give KCl at a rate of 3 mEq./kg. of body weight/day until the potassium depletion is corrected and then at a rate of 60mEq./day to prevent recurrence of depletion.
3. Give an isotonic solution containing sodium and ammonium chlorides at a rate of about 2 liters per day until alkalosis is corrected. (Travert No. 33® or ionosol G® or 50 mEq. sodium chloride and 50 mEq. of ammonium chloride, and 40 mEq. of potassium chloride, per 1000 ml. of glucose in water.)
4. Obtain blood for chemical determinations every other day and occasionally every day.
5. Metabolic alkalosis due to simple potassium depletion (Cushing's syndrome, or therapy with ACTH or cortisone) can be corrected by oral administration of potassium chloride, potassium citrate or acetate alone.

The repair of intracellular potassium deficit is a slow process and requires from three to six days to complete. Darrow[3] states that 3 mEq./kg/day is the maximum rate at which potassium can be utilized.

Chronic Respiratory Acidosis

This has been discussed in the preliminary report[1] and is mentioned again only to emphasize the superficial similarity of chemical findings to those of alkalosis. Except under unusual circumstances parenteral fluid therapy is not indicated. The clinical condition of the patient is the most important diagnostic clue. A seriously-ill patient with inadequate food intake can develop potassium depletion and alkalosis in addition to respiratory acidosis. Potassium replacement therapy is indicated, but it is usually possible to correct the deficit by oral administration of potassium chloride. If pyloric obstruction develops in a patient with emphysema and acidosis, a combined defect develops—respiratory acidosis complicated by metabolic alkalosis. In general the rules of treatment of alkalosis applies to this situation, but the carbon dioxide and chloride concentrations will not return entirely to normal because of the respiratory acidosis.

Acute Dehydration Resulting from Severe Diarrhea

A violent attack of diarrhea can rapidly deplete water and electrolytes in adults as well as infants and children, although the pediatric patients are more susceptible to serious dehydration from this cause. Mild to moderately severe acidosis is common. When 5 to 7 per cent of body weight has been lost, shock may develop, and the probability of shock increases with the severity as well as the duration of dehydration. If the fluid loss is not quickly replaced, anuria may develop and death occur later or death may occur as a result of profound shock. The urgent need is to replace water lost with a solution containing glucose and isotonic sodium chloride or sodium chloride and sodium lactate or sodium bicarbonate. The need for potassium replacement is not great in adults, but it is in infants.[3,14] If diarrhea persists for several days or more, and no food is eaten, potassium depletion does occur. If reliable body weight is available just before onset of the diarrhea, a very accurate determination of the amount of fluid need can be made on the basis of actual weight loss. If not available it is advisable to give a total of 5 to 7 per cent of the body weight as an isotonic solution containing chiefly sodium chloride but also some sodium bicarbonate or lactate. This fluid should contain 5 per cent glucose so that starvation ketosis is corrected. It is usually advisable to give an additional 1000 cc. of 5 per cent glucose in water to allow for water for urine formation. If anuria has developed it still is advisable to give the initial replacement therapy, but after this has been given then the general principles for the treatment of anuria apply.

As long as diarrhea persists, the volume of stools can be measured so adequate replacement can be given to prevent further depletion. If shock persists, blood transfusion or plasma infusion is indicated. If shock still persists after these procedures, a vaso-pressor substance (norepinephrine® or vasoxyl®) should be administered. Prolonged shock unresponsive to plasma expanders alone is apt to occur in patients who have developed a bacteremia.

Anuria or Severe Oliguria from Any Cause

If anuria is due to shock from blood loss or other cause, the shock should be treated by appropriate means (transfusions, saline solutions—as noted under acute dehydration—antibiotics, and ultimately by vaso-pressor substances). Conversely, anuria is common in shock and should be watched for in any such patient.

The etiology of anuria is variable.[12] One cause that may be obscure is exposure to carbon tetrachloride at a time when there has been a simultaneous exposure to alcohol. Acute glomerulonephritis or an acute exacerbation of chronic glomerulonephritis, mercury or oxalate poisoning, sulfonamide crystalluria or toxicity, renal stones with ureteral obstruction, obstruction of the ureters as in cancer of the cervix, hemolytic transfusion reaction, myohemoglobinuria, crush syndrome, shock during or after operation, and massive hemorrhage with prolonged shock are among the most important causes of severe oliguria or anuria. It is not unusual to miss the fact that anuria exists for twelve to twenty-four hours after onset. It is extremely important to establish immediately whether urine is being formed or not after operation or trauma and to determine the etiology as soon as possible. A careful and detailed balance sheet is very useful in the proper management of such cases.

The basic principle of treatment of anuric patients is the supplying of just enough fluids to replace that lost from skin and lungs and any additional fluid lost by vomiting and diarrhea.[6,8,12] The following general rules apply to most cases of anuria:

1. Limit all fluids to 10 cc./kg. (not more than 750 cc./d.) plus whatever external losses occur.
2. Administer a high caloric protein-free feeding such as sugar and butter or a sauce of starch, sugar butter and flavoring substance,[6] or a commercial fat emulsion such as ediol® or lipo-mul® as much as tolerated within limits of total fluid allotment.
3. Allow for a weight loss of about one pound (or 400 to 500 Gm.) per day.
4. Do not administer streptomycin or dihydrostreptomycin. These are cumulative in the face of anuria and hence are seriously neurotoxic to vestibular and auditory nerves.
5. Keep a daily chart of fluid balance.
6. Determination of serum potassium, carbon dioxide content, and chloride, and blood urea nitrogen (or nonprotein nitrogen) on alternate days. Serum sodium determinations are also useful and included if available. If protein nitrogen) on alternate days. Serum sodium decardiogram every day is helpful and can detect evidences of serious hyperpotassemia.
7. When the serum potassium is 7.0 or higher, get daily determinations.
8. When potassium intoxication is established (8.0 mEq./l or higher) or unequivocal electrocardiographic evidences of toxicity) and especially if paralysis begins to develop, a constant slow 10 per cent glucose infusion with insulin (1 unit per 2 grams of glucose in the fluid) is indicated. This procedure is of benefit only during and shortly after the glucose and insulin infusion. Now more vigilance regarding the potassium level is necessary. Meroney and Herndon[8] have shown recently that calcium administration will abort symptoms of potassium intoxication for many days and avert the need for the artificial kidney in some instances. They recommend the following solution:

Calcium gluconate 10 per cent......	100 cc.
Sodium bicarbonate 7.5 per cent....	50 cc.
Dextrose 25 per cent in water....	400 cc.

(50 units of regular insulin)

This solution should be given intravenously by polyethylene tube in a large vein at a constant rate of 25 cc. per hour. The evidence presented is convincing. This will not correct the other chemical abnormalities of uremia and particularly the serious acidosis which develops, so that dialysis may be indicated for this reason.

When diuresis occurs (anytime up to the twentieth day) sodium chloride loss may be considerable. It is necessary to determine the sodium chloride loss and to continue to follow the serum chemistries closely for a while. At least one chemical determination for sodium and chloride during the diuresis is highly desirable. If determinations are not available, a crude estimate can be made on the basis of an average loss of 30 mEq./l. or about 2.0 Gm. of sodium chloride/l. of urine during this phase. Actually losses can be nearly twice this amount, but at least 30 mEq./l. is a safe minimum replacement.

Anuric patients are prone to develop pulmonary edema, so sodium chloride must be given only to replace losses. If no external losses occur, in general, it is better not to attempt to correct changes in serum sodium, chloride or carbon dioxide. Change in sodium concentration especially can occur from shift of sodium into cells.

By far the most important factor in the improved survival in recent years has resulted from strict limitations of fluids to approximately 10 cc./kg./day in excess of measured losses. It is a mistake not to allow a weight loss of one-half to one pound a day. The indications and experiences with an artificial kidney are reviewed by Swan and Merrill[12] and by Kolff.[6]

Chronic Renal Disease

Parenteral fluid therapy is seldom necessary, but it is worthwhile to emphasize certain features relative to fluids and electrolytes. In the nephrotic syndrome the most important measure is restriction of sodium chloride and other sodium salts. In the phase of extensive edema these patients regularly excrete an excess of salt-retaining hormone.[7] When azotemia develops, variable electrolyte patterns also develop. Then the kidney fails to conserve (or excrete excessive amounts) of sodium salts and sometimes excretes excessive amounts of potassium too. A serious lowering of sodium concentration can occur unless adequate amounts of sodium salts are ingested. For this reason it is unwise to restrict dietary sodium in uremic patients unless laboratory studies are readily available and it has been demonstrated that the patient does not lose salt. This statement does not apply to the edematous non-uremic patient with renal disease. Potassium deficiency may become manifest, so that extra potassium as potassium citrate or acetate in doses of 20 to 60 mEq. per day is needed.

Congestive Heart Failure Complicated by Electrolyte Disturbances

The need for salt restriction and the great benefit derived by relief from edema by use of diuretic agents are well-established clinical observations. Diuretic agents must cause a loss of sodium chloride in addition to water in order to be effective. There are several important sequellae which may occur after an intensive therapeutic program with mercurial diuretics:[10] (1) hypochloremic alkalosis with or without potassium depletion, (2) hyponatremia ("low salt syndrome") and (3) ammonium chloride intoxication with acidosis. Usually only one of these conditions exists at one time, but varying combinations of conditions do occur. The patient usually remains edematous. Often the initial clue that something is wrong is a failure to respond to mercurial diuretics after an initially good response. At other times lethargy, stupor or coma develop. Hypochloremic alkalosis can best be treated by the use of ammonium chloride in tablet form in dose of 8 to 12 grams a day. Failure to correct the abnormality may be due to faulty absorption or inadequate dosage. Ammonium chloride can be given by intravenous injection also as a 1 per cent solution with glucose. This has to be administered slowly (150 cc. per hour) or serious neurological symptoms develop (stupor, shock, coma, convulsions) or even death may occur.[10]

The routine intermittent administration of ammonium chloride (8.0 grams per day for three days with one or two days rest) whenever mercurial diuretics are used not only prevents hypochloremic alkalosis but also enhances diuresis.

If potassium depletion has been found to exist this must be corrected or the alkalosis will persist. Potassium chloride can be given orally. It is noteworthy that digitalis toxicity is made worse by potassium depletion and ameliorated by potassium therapy.

Hyponatremia with serum sodium concentrations of 120 mEq./l. may be due to low salt diet and diuretics but may also occur spontaneously. It must be considered to be a grave prognostic sign. If there has been a prolonged attempt to restrict salt and to use mercurial diuretics, the use of 5 per cent sodium chloride solution administered slowly in amounts from 200 to 300 cc. daily until the calculated deficit is corrected probably will be beneficial. Sodium chloride may actually be harmful or at least of no value in the type where there has been an apparent shift of sodium and chloride into cells without true salt depletion. One can calculate the sodium deficit in the extracellular fluid compartment by the following formula: 142—plasma sodium concentration x 0.6 x body weight in Kg. - mEq. 10.

The amount needed is often from 20 to 40 grams of sodium chloride in an average sized adult. Only one-third to one-half of the calculated amount can be given safely in one twenty-four-hour period.

Hyponatremia can occur in patients with hepatic cirrhosis with ascites. This is particularly true of patients who have just had an abdominal paracentesis. If fluids are not restricted at this time extreme thirst may cause the patient to take a volume of water equal to that lost by paracentesis with consequent dilution of the extracellular electrolytes. Hyponatremia is treated as noted above, but patients may not respond well. This is particularly true of those patients who have not had paracentesis or diuresis and still have hyponatremia. The strict limitation of fluids to 800 to 1000 ml. per day after paracentesis is a useful preventive measure. Paracentesis should not be performed unless there is interference with eating and locomotion because of tense ascites.

Summary and Conclusions

Some of the more serious disturbances of fluid and electrolyte balance which occur in medical practice have been discussed. The greatest possible emphasis has been placed on recording information including up-to-the-minute record of therapy and on preventive measures. Acute and subacute disturbances have been discussed almost exclusively. The administration of fluids parenterally over a long period of time involves many variables; however, the same general principle applies, namely, the prevention of depletion or accumulation of ions. In addition more attention has to be given to vitamins, calories, magnesium, and the mechanical aspects of venoclysis.

References

1. Burnett, C. H., Burrows, B. A., and Commons, R. R.: Studies of alkalosis. I. Renal function during and following alkalosis resulting from pyloric obstruction. J. Clin. Investigation, 29:169-174 (Feb.) 1950.
2. Butler, A. M., Talbott, N. B., Burnett, C. H., Stanbury, J. B., Mac Lachlan, E. A.: Metabolic studies in diabetic coma. Tr. Assoc. Am. Phys., 60:103-109, 1947.
3. Darrow, D. C., and Pratt, E. L.: Fluid therapy, J.A.M.A., 143:365-373 and 432-439, 1950.
4. Flink, E. B., and Zimmerman, B.: Fluid and electrolyte balance. I. Basic considerations. Minnesota Med., 37:545-557, 1954.
5. Holmes, J. H., and Montgomery, A. V.: Thirst as a symptom. Am. J. Med. Sci., 225:281-286.
6. Kolff, W. J.: Forced high calorie, low protein diet and the treatment of uremia. Am. J. Med., 12:667-679 (June) 1952.
7. Luetscher, J. A., Jr., and Johnson, B. B.: Chromatographic separation of the sodium retaining corticoid from urine of children with nephrosis, compared with observations on normal children. J. Clin. Investig., 33:276-286, 1954.
8. Meroney, W. H., and Herndon, R. F.: The management of acute renal insufficiency. J.A.M.A., 155:877-883 (July) 1954.
9. Randall, H. T.: Water and electrolyte balance in surgery. Surg. Clin. No. America, 445, 1952.
10. Schwartz, W. B., and Relman, A. S.: Electrolyte disturbances in congestive heart failure. J.A.M.A., 154:1237-1241, 1954.
11. Sprague, R. G., and Power, M. H.: Electrolyte metabolism in diabetic acidosis. J.A.M.A., 151:970-976 (March) 1953.
12. Swann, R. C., and Merrill, J. P.: The clinical course of acute renal failure. Medicine, 32:215-292 (May) 1953.
13. Worthen, H. G. and Raile, R. B.: Fluid and electrolyte balance II. Parenteral fluid therapy in infants and childhood. Minnesota Med., 37:558-564, 1954.
14. Weisberg, H. E.: Water, electrolyte and Acid-base Balance. Baltimore: Williams and Wilkins Co., 1953.
15. Zieve, L., and Hill, E.: Prognosis in moderate or severe diabetic acidosis. A.M.A. Arch. Int. Med., 92:63-74, 1953.

STATE NURSE SCHOLARSHIPS

J. G. Neal, state supervisor of health and physical education and chairman of the state scholarship committee, has announced that the Minnesota State Board of Education has approved 153 nurse scholarships, the first to be granted for the fiscal year July 1954—July 1955. Of the $72,500 available under the law $24,700 remains. Other applications are pending completion.

The awarding of these scholarships marks the beginning of the fourth fiscal year the state nurse scholarship program has been in existence and brings the total number of scholarships awarded to 795. The Minnesota State Legislature in 1951 passed the first nurse scholarship law which provided *needed* financial assistance to students who had the ability to complete an accredited nursing program. The maximum amount of a scholarship for the professional nursing candidate is $600, a practical nursing student $300. In return for this scholarship, the student must practice nursing in Minnesota for one year.

Broken homes, one or both parents deceased, large families, illness and mother on ADC (aid to dependent children) are some of the reasons given and substantiated by the applicants applying for assistance. Many of the students have also worked their way through high school.

FLUID AND ELECTROLYTE BALANCE

Part IV: Surgical Considerations

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IN THIS fourth and last section of a symposium on problems in fluid and electrolyte management are to be included the aspects of the problem which seem to be of most frequent interest to the surgeon. It is perfectly obvious, however, that there are no problems in this field which are, strictly speaking, peculiar to the surgical patient. The maintenance of a postoperative patient does not differ in a qualitative way from the maintenance of any other individual. He can not take fluid and nutrition by mouth. The losses sustained through tubes and fistulae require the same type of replacement as those incurred through vomiting or diarrhea. Furthermore the non-specific physiologic consequences of a surgical operation are only a special case in the larger category of physiological derangements resulting from disease and trauma. Much of this paper will therefore consist in amplification of principles that have been introduced in the foregoing papers.[6,7,23]

One of the most significant features of patients who have undergone major surgery is that rather rapid changes occur from one day to the next in fluid and solute losses, metabolic requirements and overall physiologic status. For this reason the complex ready-made solutions designed for routine day to day maintenance of patients by parenteral route are not applicable for use in surgery. Neither are formulae of much practical value. The requirements of a surgical patient may be far in excess of or far less than that predicted for the maintenance of a healthy individual. From the practical standpoint, therefore, the flexibility of treatment necessary for ideal management of patients before and after major surgery is best achieved by the use of simple basic solutions to which requisite ions can be readily added. In the University of Minnesota Hospitals the most frequently used basic solutions are 9 per cent saline solution, 5 per cent glucose solution (plus the combination of these two), and 10 per cent glucose solution. In addition, sterile concentrated solutions of electrolytes are purchased or prepared. These concentrates, which are now commercially available, include sodium chloride, potassium chloride, sodium bicarbonate, sodium lactate, ammonium chloride, calcium and magnesium salts and others. They are added to the basic solutions in accordance with the daily needs. It should be emphasized, furthermore, that it is well to think of daily requirements not in terms of volume of a given solution such as cubic centimeters of "physiologic" saline, but rather as expressed separately in the amount of water, on the one hand, and the quantity of each specific ion (preferably in milliequivalents) on the other.

Valuable Measurements

Despite the achievements which have been made during the recent years in methods of assay of the electrolyte concentrations of body fluids and the *in vivo* determination of fluid volume, it is still true that the most valuable data are those which can be obtained with the least elaborate methods. Although it is a truism to emphasize the importance of the clinical appearance of the patient, it is nonetheless remarkable how much information can be learned from clinical data such as turgor of the skin, the facies, the mucous membranes, the existence or absence of abnormal neuromuscular phenomena and the presence or absence of changes in the psyche or sensorium. Moyer, in his book on fluid balance, has done an admirable job of describing in detail the physical findings associated with various categories of fluid and electrolyte disturbance, and has pointed out the degree of accuracy with which diagnosis in this field can be made from clinical findings alone, as well as the frequency with which chemical data may be misinterpreted when the clinical picture is not taken into consideration.[16] One of the most obvious possibilities for such errors was touched upon earlier where it was pointed out that in the absence of a pH determination the interpretation of an elevated CO_2 combining power must depend on the clinician's judgment as to whether the respiratory or metabolic mechanisms are disturbed.[7] Profound derangements of central nervous system function such as coma and seizures are now recognized to be frequently related in a very pre-

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dictable manner to alterations in the state of hydration and tonicity of the extracellular fluid.

Exact measurements and recording of intake and output data are necessary for assessment of fluid needs. This frequently necessitates the use of an inlying urethral catheter purely to assure quantitative urine collection. Similar collection must be made of all external losses through tubes and fistulae, and output determinations may occasionally necessitate the weighing of dressings which cover fistulae when no other method of quantitative recovery is possible.

The daily determination of the body weight as applied to fluid administration in surgical patients by Wangensteen has become an invaluable technique for assaying the state of hydration.[22] The interpretation of the body weight depends on the valid assumption that significant changes occurring over short time intervals are mainly due to gains or losses of water. Overall protoplasmic depletion in patients maintained on inadequate nutrition will, of course, over longer periods of time cause reduction of body weight which must not be misinterpreted as the result of negative water balance. Such depletion, however, can be corrected for so that the body weight as an index of hydration can still be useful even in patients carried on parenteral alimentation over periods of many days. There are situations in which the body weight change gives crucial information obtainable in no other way. The most prominent among these is oliguria. Here the body weight is the only reliable basis for deciding whether failure of urine output has resulted from primary renal deficiency or merely inadequate fluid administration. For obvious reasons, treatment in this situation based on an improper assumption as to the mechanism can be disastrous.

Chemical determinations of blood ionic concentrations are of unquestioned importance and the specific syndromes associated with their alterations will be discussed. Again it should be pointed out that, whereas an abnormality in the serum concentrations may of itself be important, the serum level alone is no index of the overall state of excess or depletion of a solute in question. It is feasible, of course, to measure total amounts of electrolytes either by using isotopic tracer technique or by utilizing the concentration in conjunction with various measurements of the extracellular space. These methods, however, are rarely available for use in routine clinical situations. It is hoped that the use of stable substances such as rubidium which can be utilized for the measurement of the total body potassium will in the future make total assays of body constituents feasible in the solution of clinical problems.[2]

Body fluid chemistry need not be limited to determinations applied to the serum or plasma and, it is frequently necessary to determine the composition of secretions abnormally lost from the body. Similarly, urinary concentrations particularly of sodium, chloride and bicarbonate, although frequently misleading, can, when properly interpreted, be of value. Bedside methods are available for determination of these quantities.[19]

The fluid management of surgical patients can be divided for purposes of discussion into four phases: (1) maintenance of a patient who for reason of the surgery or disease is unable to take food or water by mouth; (2) replacement of external losses; (3) non-specific effects related to the trauma incident to surgery; and (4) recognition and treatment of specific syndromes of electrolyte distortion.

Basal Maintenance of Patient in the Absence of the Oral Route

The initial consideration for fluid replacement must be the basal requirement for the human being who for reasons of disease or surgery is denied the oral route of alimentation. The minimal requirements for an adult then must take account of an insensible water loss ranging from 600 to 900 cc. and an anticipated urine output of 1000 to 1500 cc. Insensible losses may be far greater than this particularly under circumstances of fever, prolonged surgery under heavy drapes, hyperventilation, and so forth. The factor of insensible loss is therefore not only variable but highly unpredictable and the ignorance of this quantity is the basic reason for dependence on the body weight in assessing states of hydration. Accurate studies on patients with anuria in whom the control of water administration must be extremely precise have indicated that insensible losses may drop considerably lower than the above-mentioned limits. As little as 400 to 500 cc. of exogenous water may be required.

Large amounts of sodium chloride are not required for maintenance of patients who do not suffer from external losses. The normal kidney can restrict sodium excretion to less than that contained in 1 gram of sodium chloride (17 mEq.) and the insensible water loss (excluding frank sweat) contain only traces of sodium salt. One to 3 grams is therefore adequate for ordinary maintenance. As has already been mentioned, however, this ability of the kidney to conserve in the absence of intake does not hold for potassium ion.[7] Since 30 to 40 mEq. of potassium may be excreted by patients receiving none by mouth or by parenteral route, it is necessary to include in a parenteral regimen at least 2 to 3 grams daily of a salt such as potassium chloride. These losses may be much greater following the stress of major surgery, burns and trauma. A detailed analysis of these requirements is to be found in the paper by Elman on the subject.[11]

Replacement of External Losses

In addition to maintenance amounts of fluid, parenteral administration must include volume-for-volume replacement of water lost through abnormal routes such as tubes and fistulae. The replacement of solutes lost through these channels must take into account the origin of the secretion. In most instances average values can be used for such replacements although occasionally where losses are large, it is necessary to send samples to the laboratory for more accurate analysis.

This is particularly important in fistulae wherein the secretion may be of mixed or undetermined origin. Since the ratio of sodium to chloride in various gastrointestinal fluids varies greatly from that of plasma, their loss will produce not only over-all electrolyte depletion but also profound disturbances in acid-base equilibrium. Thus loss of normal gastric juice which contains more chloride than plasma and very little sodium produces alkalosis within a very short time. Conversely, pancreatic juice possesses sodium concentration which is similiar to that of plasma, but a very small amount of chloride, the remainder of the anions being made up by bicarbonate. It is essentially a solution of sodium bicarbonate. External pancreatic fistulae, therefore, produce acidosis. Bile is also more alkaline than plasma though its sodium-to-chloride ratio is not nearly so high as that of pancreatic juice. Intestinal fistulae, except for high duodenal ones, tend also to produce acidosis because of a relatively greater sodium loss. This we have found to be particularly the case in the occasional instance of a patient who has undergone a previous subtotal gastrectomy which eliminates a major source of chloride coming into the intestinal tract.

Lockwood and Randall have provided us with data based on many more cases than had been previously studied on sodium chloride, potassium on gastrointestinal secretions of all types obtained through tubes or fistulae.[11,18] Their table, which is included in Dr. Flink's paper, is a good one to have available for reference.[6]

To replace the acid secretion from a normal stomach it is usually only necessary to use sodium chloride solutions even though this entails giving an excess of sodium ion. A normal kidney excretes the sodium and acid salts such as ammonium chloride are rarely necessary. In the management, however, of pancreatic, biliary and intestinal fistulae where sodium loss predominates, the administration of sodium chloride is usually not adequate and alkaline replacement solution such as sodium bicarbonate or sodium lactate must be administered. In this connection it must be remembered that the plasma itself, being of a buffered solution of sodium chloride and sodium bicarbonate, is normally alkaline. The administration of sodium chloride is therefore basically acidosis-producing though the normal kidney can compensate for this effect.

Non-specific Responses to Major Surgery

Although an exhaustive treatment of the subject of physiologic response to surgery is beyond the realm of this discussion, a few basic considerations in this field are relevant to a consideration of the common electrolyte abnormalities seen in surgery. It is widely appreciated that following trauma of a degree represented by major surgical procedures a series of metabolic changes uniformly take place which qualitatively are independent of the type of operation. These changes which became apparent with the work of Cuthbertson on experimental fractures have been studied extensively in many clinical investigations beginning with those of Howard and of Albright.[1,5,8] The counterpart of these reactions as seen in experimental animals is found in the experiments of Selye and others who have emphasized the role which the endocrine system plays in their production.[20] Moore has elaborated these investigations and has attempted to put the degree of metabolic response on a quantitative basis.[13] Among these phenomena is the tendency for patients throughout a variable number of days after surgery to retain sodium ions to an abnormal degree. This means that excessive amounts of salt administered to the patient will not be excreted at a rate even approximating that observable in normal individuals. Consequently, whereas it is almost impossible to produce edema by excessive sodium administration in the healthy human, this will occur in postoperative patients and particularly in those who have had massive procedures or who suffer infection or other complications resulting in a protracted postoperative course.

Concommitant with retention of sodium an abnormally high potassium excretion occurs. This is partly associated with a breakdown of intracellular substance, as demonstrable by a large output of nitrogen in the urine, but balance studies clearly indicate that urinary loss of potassium under these circumstances is greater than that which can be explained by protein breakdown alone. Though it is generally believed that these two phenomena are the consequence of increased adrenal activity, the observations of Ingle on adrenalectomized rats and some observations of our own in adrenalectomized patients undergoing surgery suggest that they can take place even in the absence of alterations in adrenal function.[9] It is our feeling, therefore, that excessive adrenal activity is perhaps only one of several factors in the mediation of postoperative metabolic responses.

A seeming paradox exists in the fact that, whereas sodium and to a slightly lesser extent chloride are avidly retained by the postoperative kidney, the serum levels of these two ions tend to be lowered during the immediate period following surgery. This dilution of the major extracellular electrolytes is illustrated by the course of extracellular electrolyte concentrations in uncomplicated cases, for which mean values are shown in Figure 1. The question we then ask ourselves is where have the sodium and chloride ions gone if they are depressed in the plasma and not excreted in the urine. To some extent this can be answered in light of the additional information that the postoperative kidney also tends to retain water and that there is, therefore, a dilution of the extracellular ionic concentration as a result of water retention alone. Studies of body weight changes, however, and of extracellular space measurements indicate that this by itself is not adequate completely to explain the reduction in extracellular concentration of sodium and chloride. It has been suggested, therefore, that sodium moves into the cell following surgery. Although such a shift almost surely does occur concommitanly with the loss of potassium from the body, there is still some question as to whether the entire picture can be explained on this basis. Since the reduction of sodium and of chloride are roughly equivalent, this explanation would require that chloride also moved into the cell. It is more difficult to find evidence to support the latter movement. One must admit, therefore, that the apparent translocation of these ions which results from the stress of surgery has not been completely elucidated. It has been pointed out that the bone constitutes a reservoir of sodium constituting perhaps 30 per cent of the total amount of this ion in the body.[3] Changes in the composition of this reservoir have not been studied but it would seem likely that a more thorough knowledge of this aspect of sodium metabolism is in order.[14]

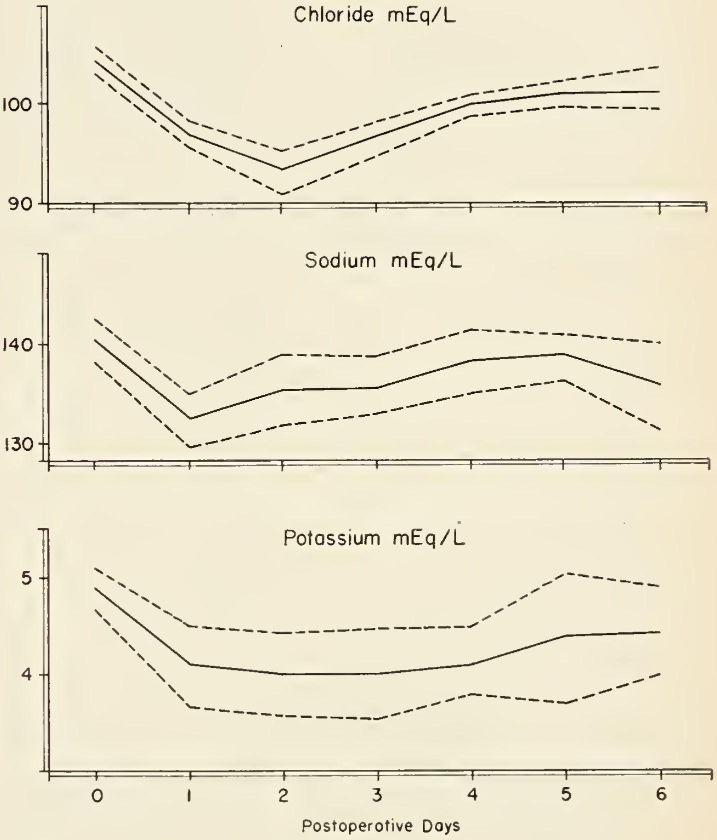


Fig. 1. The course of sodium, chloride and potassium concentrations in the plasma following uncomplicated major surgery (mean values in forty cases).

As is shown in Figure 1 the serum level of potassium is consistently reduced throughout the period following surgery when all nutrition is withheld. This situation was emphasized by Randall and his associates who pointed out that the postoperative course of serum potassium could be significantly elevated by the administration of potassium salts.[17] Whereas temporary depressions of the potassium such as shown here are perhaps not harmful, the level continues to fall and eventually leads to the characteristic picture of potassium deficiency if administration of this ion is withheld for more than a few days. In order, therefore, to obviate the necessity for giving very large amounts of potassium when the depletion syndrome becomes imminent it is well to give maintenance amounts within a day or two after operation or when adequate urine output has been assured.

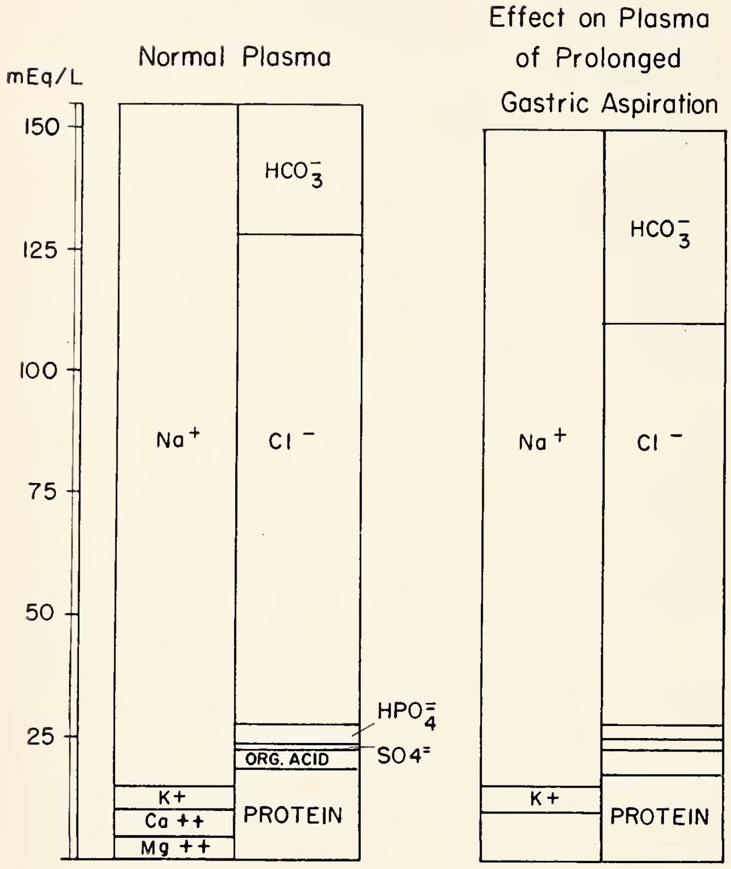


Fig. 2. Hypochloremic alkalosis. Effect on extracellular electrolyte concentrations of several days of nasal-tube suction without adequate chloride replacement. Observe reduction of chloride with expansion of bicarbonate and essentially normal sodium.

Even in the absence of a complete explanation for the deficit in sodium after operation the two facts (1) that the sodium is withheld from the urine while potassium is lost, and, (2) that the serum levels tend to be low, are of great importance in explaining some of the electrolyte complications to which these individuals are subject.

Most Frequent Syndromes of Electrolyte Derangement of Interest to the Surgeon

Since many of the common derangements of fluid and electrolyte balance have already been discussed in the sections dealing with pediatrics and internal medicine, a complete description of these entities is not pertinent at this point. Brief mention, however, should be made of those disturbances which are more commonly seen in connection with surgery and the ways in which the physiological processes associated with stress are related to their occurrence.

Dehydration.—Though dehydration in a precise sense refers to the effects on the body of water loss alone, it most frequently is used to describe the deficiency of water in combination with electrolytes. Pure water dehydration is actually rare. The familiar signs of dehydration are loss of turgor of the skin, sunken eyes and dryness of the mucous membranes. Where controlled observations are possible, the change in body weight, of course, is the best index and the best guide to therapy. It is important that under the circumstances of dehydration the serum and electrolyte concentrations can be elevated or depressed or normal and therefore may give no indication of the over-all deficit in the major ionic constituents. In the absence of fluid intake, water loss almost invariably exceeds electrolyte loss so that it is best to begin hydration with solutions that are dilute with respect to ionic constituents. Subsequent chemical determinations frequently reveal the presence of electrolyte deficit as a more normal state of hydration is approached.

Edema.—Edema as a complication of surgery was frequently seen when "physiological" saline was used rather routinely as a hydrating solution.[10] Following the recognition by Coller and his associates of the tendency for patients to retain sodium in the early postoperative period, the excessive use of sodium chloride was almost universally discontinued and postoperative edema became far less common.[4] As with over-all extracellular dehydration so with extracellular fluid accumulation, the serum levels cannot be expected to give any index of excess of ions such as sodium which may actually be the basis for fluid retention in the extracellular compartment. Here again the familiar clinical signs are of the greatest value though body weight determinations where possible indicate beginning fluid retention long before clinical edema is demonstrable.

Hypochloremic Alkalosis.—One of the most commonly seen derangements of acid-base balance is the alkalosis which is caused by continuous drainage from the stomach of the fluid high in chloride concentration. The serum chlorides are low, bicarbonate is elevated and the sodium may be normal or slightly depressed. It is of interest in these circumstances that the alkaline urine which one might expect as a result of an attempt on the part of the kidney to compensate for an elevated plasma pH is frequently not seen. The urine pH is, therefore, useless as a guide to the effectiveness of treatment in this situation.[21] The mechanism of this "paradoxical aciduria" is partially obscure but is probably related to the concommitant retention of sodium, the majority of which is being transported into the cells while large quantities of potassium are being removed. As was mentioned above, simple chloride-deficiency alkalosis can usually be treated with sodium chloride without the necessity for the use of acid salts such as ammonium chloride. Such alkalosis is, however, frequently not on the basis of chloride loss alone but is a result in whole or part of potassium deficiency. Figure 2 illustrates the composition of the normal plasma and the distortion produced by pure chloride-loss alkalosis resulting from the use of an inlying gastric tube without adequate chloride replacement.

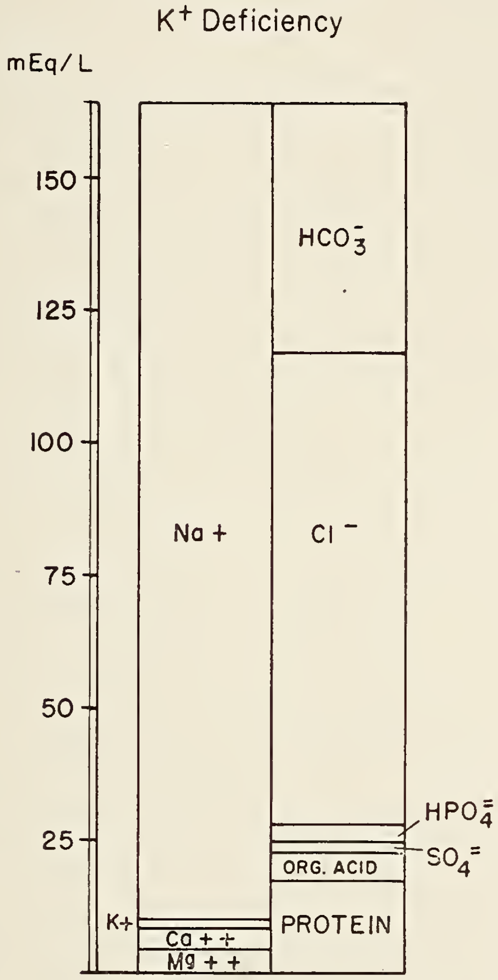


Fig. 3. Potassium deficiency. High duodenal obstruction treated by nasal tube drainage and parenteral therapy including adequate sodium chloride but no potassium salts. Serious hypochloremic alkalosis with depressed serum potassium level.

Potassium Deficiency.—The depletion of potassium ions is common in surgical patients because gastrointestinal secretions contain roughly twice the potassium concentration of plasma but a more important factor is the massive loss of potassium which appears in the urine as a consequence of surgical trauma or acute surgical diseases. A frequent plasma electrolyte picture is that of hypochloremic metabolic alkalosis which is refractory to treatment with sodium chloride or even ammonium chloride. Serum levels of potassium are usually depressed but may not be so and so-called "refractory alkalosis" is adequate indication for intensive treatment with potassium salts. The clinical signs are weakness or paralysis and frequently paresis of intestinally motility can be shown to be on this basis. The tetany (Trousseau sign, Chvostek sign, carpopedal spasm) which accompanies severe akalosis is occasionally seen. Disorientation is frequent. It is important that in early stages of potassium depletion hyperactive speech and irrelevant conversation may indicate the necessity for investigating potassium depletion. Electrocardiographic changes have been described in the previous sections as have the types and routes of treatment.[6] The electrocardiogram and CO_2 combining power in our experience are the best guides to effectiveness of therapy. Figure 3 shows the fluid and electrolyte picture in a young woman with high intestinal obstruction who had been treated for a period of over two weeks with massive amounts of sodium chloride and ammonium chloride without potassium ion. Vigorous administration of potassium chloride was all

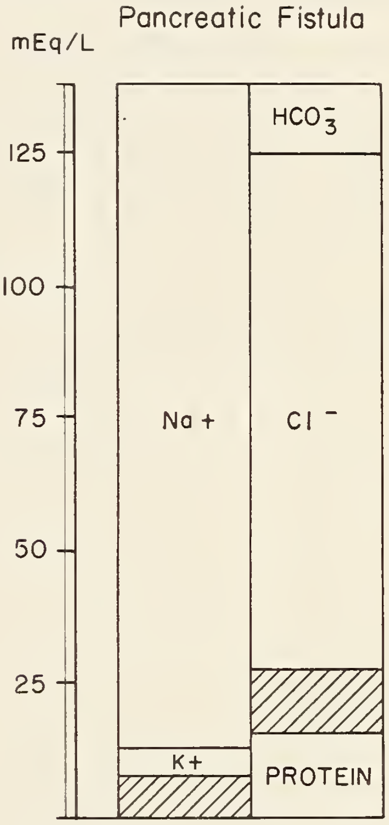


Fig. 4. Hyponatremic acidosis. The result of sodium loss through pancreatic fistula which resulted from drainage of pancreatic cyst. Readily corrected by parenteral administration of sodium lactate or ingestion of sodium carbonate. Note reduction of sodium with corresponding contraction of bicarbonate. Crosshatch = normal values assumed (cations) or determined by subtraction (anions).

that was needed to return her acid-base balance to normal. In postoperative patients we have most frequently seen the potassium depletion syndrome in those patients who have suffered prolonged debilitation or interference with gastrointestinal function prior to surgery. As one might anticipate, it is also particularly common in patients undergoing a second operation within two to three weeks of the first during which interval the negative potassium balance resulting from the initial surgery has not been completely restored.

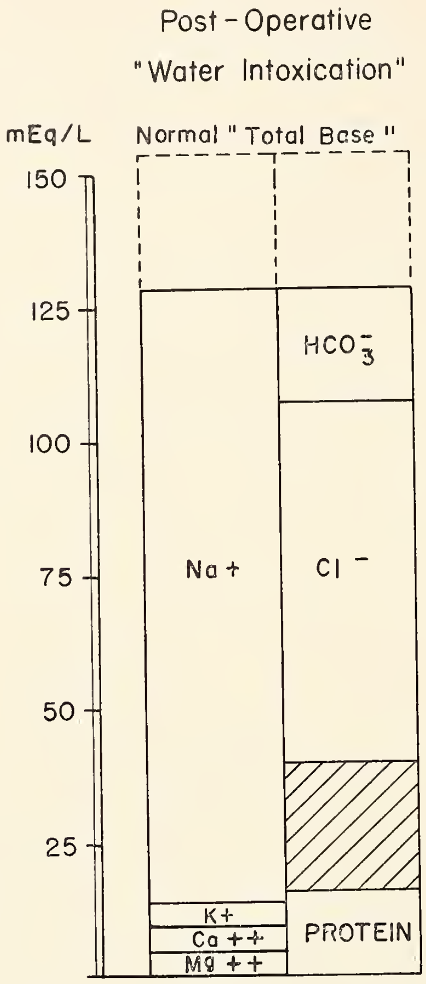


Fig. 5. Water intoxication. Seventy-one-year-old hypertensive woman treated with low salt diet prior to right colectomy; 3000 cc. 5 per cent glucose on operation day and 3500 cc. 5 per cent glucose without sodium chloride on first post-operative day. Seizures occurred 30 hours after operation. Diagram shows results of blood chemical determinations at time of seizures. Observe reduction of sodium and chloride, i.e. over-all electrolyte "dilution."

Hyponatremic Acidosis.—Figure 4 illustrates the metabolic acidosis which can result from pure sodium loss. These measurements were made on a man who developed external pancreatic fistula following operation for pancreatic cyst. Daily administration of sodium lactate is adequate to prevent acidosis under circumstances such as these and in patients who do not have fistulae involving the intestinal tract itself, oral administration of sodium bicarbonate is frequently feasible.

Dilution: Water Intoxication and Low Sodium Syndrome.—Among the most dramatic clinical emergencies associated with abnormalities of electrolyte metabolism are those which have as their basis a severe depression of extracellular sodium concentration.[24] In Part I of this symposium the hypothesis of Darrow and Yannet was mentioned and it was pointed out that the relationship between intracellular and extracellular water depended on the concentration of extracellular sodium. Since water moves into the cell in the face of lowered sodium concentration of the extracellular fluid, the effect on the intracellular volume is the same whether the dilution is produced by addition of water or by abstraction of sodium from the extracellular compartment. The cells of the central nervous system are particularly sensitive to this type of volume change. The clinical picture is characterized by profound central nervous system disturbances and frequently by seizures. In the cases of "water intoxication" which have been studied in this hospital, the symptoms were nearly always between twelve and thirty-six hours after surgery. Reference to Figure 1 shows that this is the period when the usual postoperative drop in serum sodium concentration is maximal. These instances represent then a sort of exaggeration of a normal postoperative phenomenon. Many of the cases studied received more salt-free glucose solutions than they should have. Others were given only usual or even small amounts. In any case, none of the patients in the series received amounts of water which would produce symptoms in normal individuals. Surgery, therefore, renders patients highly susceptible to this complication. Serum sodium levels when drawn at the time of symptoms are nearly always below 125 mEq./L and in many instances can be measured below 115 mEq./L. Because of the possibility of this complication, we feel now that it is not wise ever to give nothing but salt-free glucose solutions during the first two days after operation and even when no external losses occur it is advisable to give 4.5 to 6 grams of sodium chloride per day during this period. A similar picture associated with profound serum sodium depression has frequently been noted in patients undergoing surgery for acquired heart disease particularly following the operation of mitral valvulotomy.[15] These patients have characteristic impairment in water excretion and are usually prepared for surgery with long periods of sodium restriction. In view of the frequency of this complication then, it appears inadvisable to maintain patients on such severe sodium restriction when mitral surgery is contemplated. Treatment of these conditions requires strict limitation of administered water and administration of hypertonic solutions and 2 per cent saline is most frequently used. Figure 5 illustrates the usual electrolyte picture in water intoxication, the only consistent findings being a reduction in the concentrations of all the major extracellular ions.

Hypernatremia.—A relatively rare but serious complication has been recognized in a few patients who demonstrate a pathologic tendency to retention of sodium in the face of otherwise normal renal function.

It occurs in two general types of conditions; certain kinds of renal injury, and occasionally lesions of the central nervous system involving either the frontal lobes or the hypothalamus. It was first described by Luetscher as a complication of sulfathiazol poisoning of the kidneys. It is now recognized that it can occur following any type of renal damage. Generally it appears to be a stage in the recovery from renal injury and thus hypernatremia is occasionally seen during the diuresis phase following instances of so-called "lower nephron syndrome." The cases in which the central nervous system has been implicated have usually been seen following neurosurgery particularly pre-frontal lobotomy. Serum sodium levels of over 190 are not uncommon. Clinical signs include profound central nervous system depression and occasionally neuromuscular phenomena such as athetoid and choreiform movements of the extremities. Death is likely to occur. Little is known about the treatment except for the obvious procedure of withholding of sodium salt and administering as large amounts of salt-free glucose solution as can be tolerated. Even with such a regimen it may be many days before the serum sodium level comes down to normal and the scant sodium excretion despite seriously elevated sodium levels can be truly remarkable.

The conditions which have been described above are, of course, pure situations but in clinical practice frequently occur in combination. By no means all the possible electrolyte complications have been described. Unfortunately the surgeon must frequently be concerned with the delicate problem of managing patients with anuria. This subject has been dealt with in Dr. Flink's discussion of the problem as it arises in internal medicine, and the method of management is in no way different when the problem arises in surgical patients.

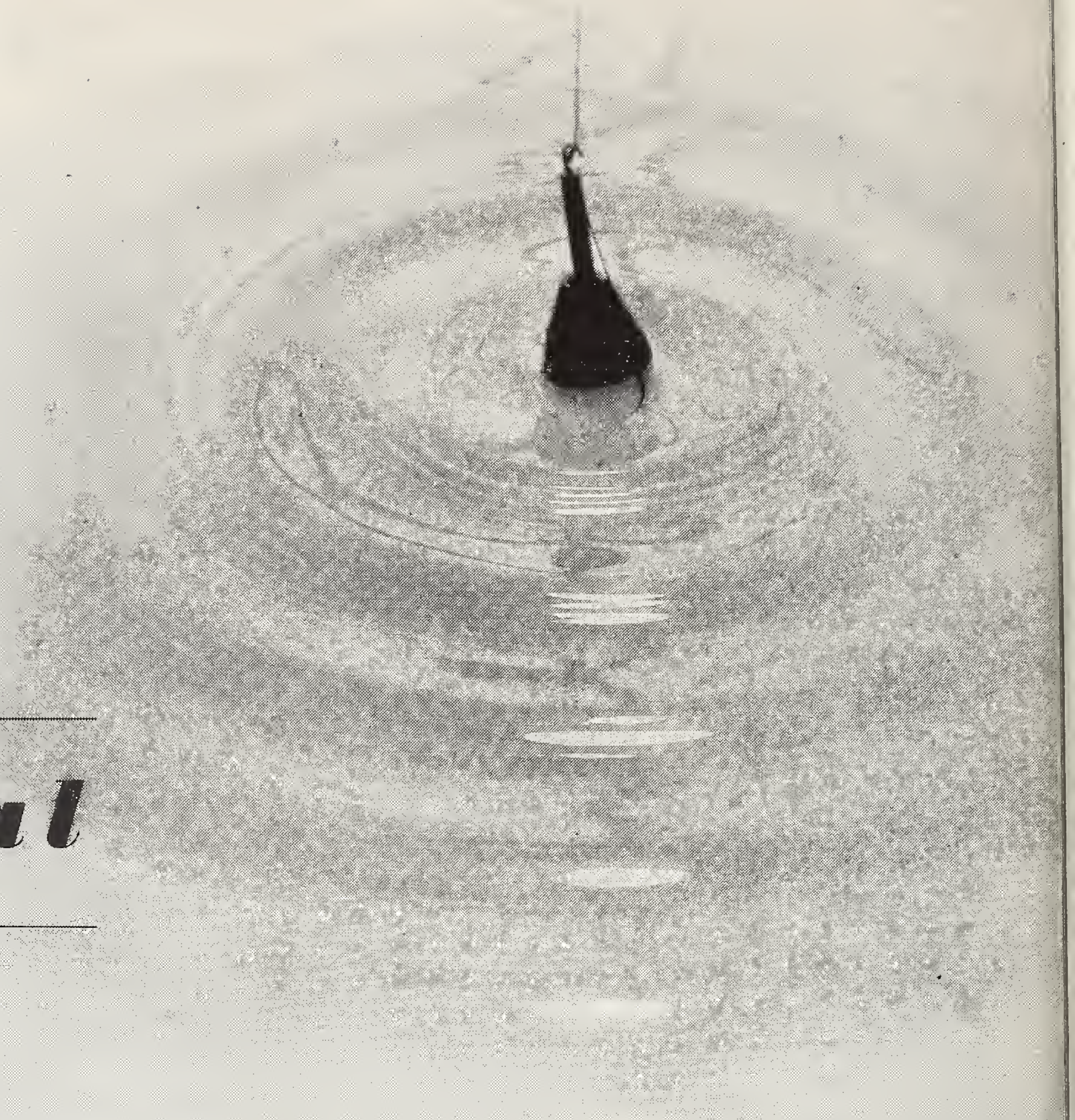
Summary

Problems of maintaining and correcting fluid and electrolyte balance are essentially the same whether encountered in the practice of Internal Medicine, Pediatrics or Surgery. Because of the highly variable and rapidly-changing physiologic status of the postoperative patients, however, it is our feeling that complex or "ready-made" polyionic solutions are not as useful in this field as are simple basic solutions to which ions in concentrated form can be added.

Certain syndromes of electrolyte distortion are rather commonly seen in surgical patients and the manner in which their development is related to the altered physiological processes which characterize the response to surgery has been discussed.

References

1. Albright, F.: "Cushing Syndrome," its pathological physiology, its relationship to the adreno-genital syndrome and its connection with the problem of the reaction of the body to injurious agents. Harvey Lectures, 38:123, 1942-43.
2. Ansell, J. S., and Zimmerman, B.: The use of stable rubidum for the determination of total body potassium. To be published in Surgical Forum, 1954.
3. Bergstrom, W. H., and Wallace, W. M.: Bone as sodium and potassium reservoir. J. Clin. Invest., 33:867, 1954.
4. Coller, F. A., Campbell, K. N. V., Vaughn, H. H., Iob, L. V., and Moyer, C. A.: Postoperative salt intolerance. Ann. Surg., 119:533, 1944.
5. Cuthbertson, D. P.: Post-shock metabolic response. Lancet, 242:453, 1942.
6. Flink, E. B.: Fluid and Electrolyte Balance. III: Problem in Internal Medicine. Minnesota Med. (Sept.) 1954.
7. Flink, E. B., and Zimmermann, B.: Fluid and Electrolyte Balance. I: Basic Considerations. Minnesota Med., 37:545, 1954.
8. Howard, J. E., and Bigham, R. S., Jr.: Tr. of Tenth Conference on Metabolic Aspects of Convalescence. Josiah Macy, Jr. Foundation (1945).
9. Ingle, D. J., Meeks, R. C., and Thomas, K. E.: The effect of fractures upon urinary electrolytes in non-adrenalectomized rats and in adrenalectomized rats treated with adrenal cortex extract. Endocrinology, 49:703, 1951.
10. Jones, C. M., and Eaton, F. B.: Postoperative nutritional edema. Arch. Surg., 27:659, 1933.
11. Lockwood, J. S., and Randall, H. T.: The place of electrolyte studies in surgical patients. Bull. N. Y. Acad. Med., 25:228, 1949.
12. Luetscher, J. H., Jr., and Blackman, S. S., Jr.: Severe injury to kidneys and brain following sulfathiazol administration: high serum levels and persistent brain damage. Ann. Int. Med., 18:741, 1943.
13. Moore, F. D., and Ball, M. R.: The Metabolic Response to Surgery. Springfield, Illinois: Charles C Thomas, 1952.
14. Moore, F. D.: Bone sodium (editorial). Ann. Surg., 139:253, 1954.
15. Moore, F. D.: The low sodium syndromes of surgery. J.A.M.A., 154:377, 1954.
16. Moyer, C. A.: Fluid balance, a Clinical Manual. Chicago: The Year Book Publishers Inc.
17. Randall, H. T., Habif, D. V., and Lockwood, J. S., and Werner, S. C.: Potassium deficiency in surgical patients. Surgery, 26:341, 1949.
18. Randall, H. T.: Water and electrolyte balance in surgery. The Surgical Clinics of North America. Philadelphia: W. B. Saunders Co., 1952.
19. Scribner, B. H., Power, M. H., and Rynearson, E. H.: Bedside management of problems of fluid balance. J.A.M.A., 144:1167, 1950.
20. Selye, H.: Stress. Montreal: Acta, Inc., 1950.
21. Van Slyke, K. K., and Evans, E. I.: The paradox of acidura in the presence of alkalosis caused by hypochloremia. Ann. Surg., 545, 1947.
22. Wangensteen, O. H.: Controlled administration of fluid to surgical patients including description of gravimetric methods of determining status of hydration and blood loss during operation. Minnesota Med., 25:783, 1942.
23. Worthen, H. G., and Raile, R. B.: Fluid and Electrolyte Balance. II: Parenteral Fliud Therapy in Infants and Children. Minnesota Med., 37:558, 1954.
24. Zimmermann, B., and Wangensteen, O. H.: Observations on water intoxication in surgical patients. Surgery, 31:654-669, 1952.
25. Zimmermann, B., and Freier, E. F.: The occurrence in surgical patients of severe hypernatremia without exogenous dehydration. Surgery, 31:373-384, 1952.



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Seminar

BRUCELLOSIS

JAMES D. TOWNSEND, M.D.
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Although clinical descriptions believed to be compatible with brucellosis are recorded as far back as Hippocrates in 450 B.C. actual discovery of the organism was made by David Bruce in 1886. Because the disease was so prevalent on the island of Malta, it caused marked morbidity among the British troops stationed there. To meet this problem, the Mediterranean Fever Commission, headed by Bruce, was formed in 1904 to investigate its cause, and possible means of prevention. During the next two years the work of this commission was monumental. A member of the commission, Dr. T. Zammit, accidentally discovered that goats' milk was heavily infected with Brucella organisms. Following this the incidence of the disease was greatly reduced by forbidding the use of raw goats' milk on the island.[16,20]

The first authentic case of brucellosis originating in the United States was reported from Washington, D. C., in 1905.[20] Minnesota did not report its first case of human brucellosis until 1927. There was a steady increase in the number of cases reported each year until 1947. This incidence has since declined.[8,13]

Etiology

There are three species of the genus Brucella, all of which may cause the disease in man. They are Brucella abortus, Brucella suis, and Brucella melitensis, their primary habitat being cattle, hogs and goats respectively. Morphologically they are very small, non-motile, Gram-negative bacilli or coccobacilli. The melitensis variety is most invasive and causes the most severe infection in man, while abortus causes the least severe infection. Suis lies between the other two in pathogenicity.[16,33]

Epidemiology

There are only three important means by which Brucella infection is transmitted to human beings. These are:

1. Ingestion of infected raw milk or other unpasteurized products.
2. Contact with infected animals, or their flesh or products of conception.
3. Contact with live cultures during the course of scientific work.[16,32,33]

In the United States brucellosis is the most common milk-borne disease, and it is the most common disease transmitted from animals to man.[3,16]

Raw milk is the major source of infection in man except in those occupational groups exposed to the above means. It is interesting that there is evidence that the normal skin is a more vulnerable portal of entry for the organism in man than is the digestive tract. However, the disease is primarily an occupational one as indicated by these figures reported by Jordan.[21]

Annual Infection Rate per 100,000 (Iowa 1942-1945)

1.	Packing-house workers	271.5
2.	Veterinarians	250.0
3.	Farm workers	43.0
4.	Merchant, professional	3.3
5.	Farm wives	2.2
6.	Housewives	1.4

Because of these occupational factors most cases occur in men. In Minnesota and Iowa about three-fourths of the cases occur in adult males.[3,33,37] Brucella abortus is responsible for about three-fourths of the cases in this state and we are fortunate in this respect since it is the least pathogenic strain. Within recent years however, the incidence of both melitensis and suis infections have been increasing here. Presumably they are acquired from Iowa. Brucella suis is the most common strain encountered in human cases in Iowa.[13] It is interesting to note that Brucella melitensis is being harbored by hogs, instead of goats or sheep.[22] In addition Brucella suis occasionally infects cows and when it does, rather severe milk-borne epidemics may result because of its more invasive nature. It is unusual for a milk-borne epidemic to occur due to Brucella abortus.[3]

Children are apparently more resistant to the disease than adults.[32,37] Some authors point out that the apparent low incidence in infants and children may be due to failure to consider the disease in the diagnosis of many unexplained illnesses in childhood.

The geographic distribution of the disease in man roughly parallels infection in goats, swine, cattle and sheep throughout the world, but ac-

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curate statistics are not available. Some areas show an extremely high incidence, for example, the rural area about Cordoba, Argentina, in which 77.9 per cent of persons were found to be positive reactors. In Minnesota 20 per cent of the rural population has been reported to give positive intradermal tests for brucellosis.[33] In the United States, Iowa has the highest incidence of reported cases with 5.9 per 100,000 per year. Minnesota ranks seventh with 3.4 cases per 100,000.

In 1951, Iowa, Wisconsin and Minnesota reported about one-half of the total number of cases in this country. The predominent strain involved depends upon the livestock population of the area. In Iowa hogs are the most numerous farm animal and Brucella suis is isolated from 63 per cent of human cases; cattle are more abundant in Minnesota and Brucella abortus is found in 86 per cent here; in Mexico where goats are plentiful, Brucella melitensis is recovered from 95 per cent of the cases.

The disease is arbitrarily classified as acute from onset to three months, subacute from three months to one year, and chronic when the duration of illness is one year or more. There is some variation in opinion regarding the frequency with which the disease becomes chronic. Harris believes that at least 90 per cent of the cases in the United States are chronic. Of sixty-five patients followed by Spink, 46 per cent had subjective complaints longer than one year, but only 26 per cent had objective evidence of continuing disease. In addition only 12 per cent were disabled for work longer than one year.[33]

A critical analysis of the chronicity of this disease yields the figures shown in Table I taken from Feig.[8]

The well documented tendency for these patients to have vague symptoms persisting long after the acute disease subsides, plus the difficulty of making a conclusive diagnosis at this stage, combine to make this problem difficult to evaluate.

TABLE I.

	Jordon & Borts (1947) 108 cases		Spink et al (1951) 48 cases	
Duration	Cases	%	Cases	%
3 mo. or less	54	50	33	69
3-6 months	14	13	11	23
7-12 months	19	18		
1 year and more	21	20	4	8

Clinical Manifestations of Acute Brucellosis

Simpson has said that "no disease, not excepting syphilis and tuberculosis, is more protean in its manifestations."[29] Harris, in his volume on brucellosis, devotes 131 pages to its symptomatology. The disease has been proved capable of infecting almost every tissue in the body. From the foregoing it will be seen that an exhaustive discussion of the symptoms of brucellosis is impossible here.

However, it is our purpose to describe the typical clinical course of acute brucellosis. Howe and his co-workers[19] had an excellent opportunity to observe the acute disease in seventeen laboratory workers accidentally infected with brucella suis and melitensis. Almost all of these cases were confirmed by actual cultivation of the organism. All of these patients had been given "a Brucella vaccine" prior to exposure, but whether this altered the course of the illness could not be stated. In these patients the incubation period varied from twenty-four hours to seven days, in all but one. This one patient's symptoms were characterized by a gradual increase in severity for a period of one month. The symptoms were not pathognomonic. Fatigue was the chief symptom in all cases. Joint pain and aching muscles were prominent as was headache. One-half of the patients had at least one severe, shaking chill. Nonproductive cough, nausea, vomiting and photophobia were common. In four cases diarrhea occurred. Fever in these patients was extremely variable. The highest temperature elevations were usually found in the late afternoon and the severity of the disease seemed to vary directly with the degree of fever. With the exception of fever, there were no physical signs in these patients. The disease process did not localize in any organ system.

The acute disease due to Brucella abortus does not vary significantly from this in its symptomatology. Regardless of the species of the causative organism, the physical examination may be negative or it may reveal splenomegaly (one-third of cases), cervical (one-half of cases), and axillary lymphadenopathy. The duration of the symptoms is, in general, less than six months, and it may terminate in a state of well-being in a few weeks.[32]

An analysis of the signs and symptoms in thirty-five culturally proven cases due to Brucella abortus was carried out by Hall and these are listed in the accompanying tables.[14]

TABLE II. INCIDENCE OF VARIOUS SYMPTOMS IN CASES STUDIED

	Per cent
Weakness	94
Sweats	89
Malaise	77
Anorexia	77
Chilliness	74
Headache	74
Backache	63
Chills	63
Pain in Neck	51
Joint Pain	31
Nervousness	31
Insomnia	31
Cough	23
Diarrhea	14
Abdominal Pain	11
Constipation	6
Neuralgia	6
Pleural Pain	3
Dysuria	3
Testicular Pain	3

Because of the extreme variability of the disease certain manifestations deserve further comment.

1. *Fever* may at times be as high as 106-107°. A peculiar thing about the fever of brucellosis is that frequently the patient will not complain of fever or look feverish but will be found to have a temperature of 101 to 103°. Sweating may be drenching in character and often has a peculiar fetid or "mousy" odor.[16,29]

2. *Arthralgia* and muscular pains are prominent features of the acute form. Aching pain in the lumbar spine and long bones is common. Suppurative osteomyelitis occurs but is more likely to do so in the chronic phase of the disease.

3. *Central nervous system* symptoms may dominate the clinical picture from the onset. Practically all possible central nervous system signs and symptoms have been described. Emphasis has been placed on the predominance of meningeal symptoms in the majority and on the frequency of cranial nerve involvement, especially the 8th cranial nerve.[25]

De Jong[6] states that "uncomplicated brucellosis practically always shows some nervous system symptoms, apparently due to the action of a bacterial toxin which may have a special affinity for the central and peripheral nervous system."

4. *Gastrointestinal* symptoms frequently include anorexia and constipation. Simpson states that the latter is "practically invariable and parallels the severity of infection." Abdominal pain is often troublesome. In 125 cases reported by Hardy, forty had abdominal pain and in ten it was the chief complaint.

TABLE III. INCIDENCE OF PHYSICAL SIGNS IN CASES STUDIED

	Per cent
Fever	100
Lymphadenopathy	68
Weight Loss	51
Splenomegaly	43
Hepatomegaly	34
Abdominal Tenderness	11
Tender Spine	6
Neurologic Changes	6
Rash	3
Orchitis	3
Rales	3
Conjunctivitis	3
Rhinitis	3
Dicrotic Pulse	3

5. *Respiratory* symptoms may be an outstanding feature of the disease. Cough with mucoid or muco-purulent sputum is frequent in the first few weeks and may persist for many months. Chest x-ray may be negative or reveal peribronchial infiltration, hilar infiltration and patchy pneumonic areas. Hemoptysis is occasionally encountered.

It should be pointed out that not all persons whose tissues are invaded by Brucella develop the illness described above. In fact a great many of them apparently suffer no recognizable illness at all. This is supported by the fact that about 20 per cent of persons in rural Minnesota are found to have positive intradermal tests, yet there is no history of acute brucellosis.[31] It is probable, then, that a considerable number of patients have the chronic form of the disease that is not readily detected.

Although there is a greater tendency for the chronic disease to produce localized complications, the majority of them do not. Even in this phase brucellosis is marked by a multitude of symptoms in comparison to the paucity of physical findings. The rather vague symptoms include weakness, generalized aches and pains, anorexia, constipation and nervousness. The nature of these symptoms makes it apparent that confusion of this disease with psychoneurosis is likely. Complications which do occur, however, include spondylitis, orchitis, meningitis, encephalitis and endocarditis.[30,31]

Pathology

Although the pathological features of brucellosis have not been completely elucidated, certain facts stand out:

1. The organisms seem to localize and multiply intracellularly in phagocytes of the reticulo-endothelial system. Accordingly, the liver and spleen are often the site of most marked changes.

2. The most frequently occurring lesion is a granuloma which is not specific but bears much resemblance to the tubercle. Caseation does not usually occur. Microscopically the lesions show central areas of epithelioid cells surrounded by lymphoid cells. Although giant cells may occasionally be seen, they are not so prevalent as in the typical tubercle.[40]

3. As might be expected in this disease, the pathological findings are extremely variable. At times there may be only slight stimulation of the reticulo-endothelial system, or there may be no demonstrable lesions. On the other hand, extensive changes attributed to brucellosis have been described in every organ in the body.[16]

Diagnosis

In a disease with such protean manifestations it is unfortunate that no uniformly satisfactory means of establishing the diagnosis exists. Harris states that "the diagnosis of brucellosis may be among the most difficult tasks in medicine."

The history can do no more than suggest the possibility of this disease. A source of exposure is important, however, although it is not always obtained. Suspicion should be multiplied when dealing with a packing house worker, veterinarian or farmer. The physical examination is often surprisingly normal. Anterior cervical lymphadenopathy is observed in approximately one-half of the cases and splenomegaly in one-third.[34] Other findings may be noted depending upon the particular localization of the disease. They are less common in the acute form. Since the history and physical examination are inconclusive, further information must be sought from laboratory tests.

1. Culture of the organisms from blood or other sources offers the only absolute proof of the diagnosis. However, this procedure presents many difficulties. First of all, Brucella is one of the most difficult of all bacteria to isolate. Special media is required and incubation should be carried out under 10 per cent CO_2 concentration. Bacto-tryptose agar is the media now widely employed with considerable success. Although the melitensis and suis strains grow readily under aerobic conditions, abortus requires 10 per cent CO_2 and is most difficult to isolate. Cultures should be incubated for at least one month. In addition to this difficulty, bacteremia is often fleeting and unpredictable, so that the success of a blood culture may depend on pure chance. Cultures are more successful in the acute disease because bacteremia is more frequent and persistent. Nevertheless, positive blood cultures have been obtained from persons who are clinically well and have no frank history of a previous acute episode of brucellosis.[42] A case in point is furnished by Hall in a survey of families of patients with proven brucellosis. One fifteen-year-old girl, with no recognizable illness, was found to have a titer of 1:60 for brucella and the abortus strain was isolated from her blood.[14]

Cultures of spinal fluid, bile, feces and prostatic fluid may be undertaken if the clinical situation seems to warrant it. Positive cultures from sternal marrow have been reported while venous blood was negative. This simple procedure offers the additional advantage that histologic study may reveal the nonspecific granulomatous lesion of Brucella infection.

It should be pointed out that if routine rather than special cultural methods are used in the search for Brucella, the chances of isolating the organism are slight.

2. There is general agreement that next to culture of the organism, the blood agglutination reaction is the most important laboratory test. A titer of 1:100 or over is usually regarded as significant and indicative of past or present infection with Brucella. There is no titer of agglutinins that is specific for the active disease. The higher the titer, the more likely it is that the organisms can be isolated from the blood.[5] Usually the agglutination titer in culturally proven cases is 1:160 or over and in 90 per cent it is 1:320 or more. During the first week or two of acute brucellosis, agglutinins may be absent.[8] They may appear in the blood at any time from a few days to a few weeks after the onset of illness.[29,42] After recovery from the disease, agglutinins may persist for years or they may disappear completely. In performing the test it is necessary to carry the dilution of serum far enough so that the prozone phenomenon is avoided. If this occurs, agglutination may not begin until a titer of 1:160 or 1:320 is reached. In rare instances blocking antibodies may be present and the usual agglutination test will be negative.[12,30]

If this is suspected it can be detected by centrifugation of the serum-antigen mixture, or other special tests.[15] Reports of positive cultures in the presence of negative agglutination tests are frequent in the literature.[5,11,16] However, Spink states that a negative agglutination test in bacteriologically proven brucellosis is unusual.[8] Another pitfall is that although the agglutination reaction may become negative upon recovery, a totally unrelated illness may cause an anamnestic reaction and agglutinins are again discovered. Furthermore, the presence of Brucella agglutinins is not specific and cross agglutination occurs in serum from patients with tularemia, cholera and those having received cholera vaccine. The frequency of cross reactions in other conditions is probably low.[9,30] If the intradermal test is to be carried out it should be deferred until all information from the agglutination reaction has been secured, because agglutinins may be stimulated to develop. However, some authors have reported that when intradermal testing does provoke a rise in titer above 1:80 this indicates previous or current infection.[2,23]

3. According to Harris, "the significance of the intradermal reaction is, in general, comparable to that of the tuberculin test." Two preparations are in common use for the performance of this test. Bacterin (and similar preparations) is a heat killed suspension of Brucella abortus organisms. It is a more sensitive antigen although its use entails the risk of an occasional violent reaction with skin necrosis. Brucellergen is a protein nucleate fraction of Brucella organisms and it is less sensitive as well as less likely to cause a severe skin reaction.

A positive skin reaction cannot be used as evidence of Brucella infection without clinical and other laboratory evidence. A negative reaction can occur in the presence of a positive culture and therefore cannot be used to rule out brucellosis. This occurs in cases of Brucella endocarditis and its mechanism is perhaps similar to the anergy found in cases of miliary tuberculosis.

Because of the inconclusive nature of this test, plus the fact that 20 per cent of patients from rural Minnesota give positive skin reactions, the intradermal test has been abandoned as a diagnostic procedure at the University of Minnesota Hospitals.[32]

Most authors especially discourage the use of the skin test to "confirm" the diagnosis of chronic brucellosis in patients who have a history of exposure plus vague symptoms compatible with psychoneurosis.

4. The opsonocytophagic test, which measures the phagocytic power of the leukocytes against Brucella organisms, has also been abandoned by most authors as having no particular usefulness in the diagnosis of brucellosis.

5. The routine laboratory studies offer only ancillary aid in the diagnosis of this disease. The white blood count is usually normal or below normal. Rarely it may be increased. A relative lymphocytosis is usual. The sedimentation rate is normal or elevated and is of no diagnostic value.

Differential Diagnosis

Harris lists thirty-nine diseases in his discussion of the differential diagnosis of brucellosis. These range from psychoneurosis to African trypanosomiasis and Filariasis bancrofti. Perhaps the most commonly encountered problems are in the differentiation of brucellosis from typhoid fever, infectious mononucleosis, and infectious hepatitis.[10,34]

The similarity between infectious mononucleosis and acute brucellosis can be quite striking and merits further discussion. Fever is common to both diseases, as is lymphadenopathy and splenomegaly. A normal or decreased white blood count may be encountered in both conditions together with a relative lymphocytosis. Atypical lymphocytes may occur in brucellosis as well as infectious mononucleosis. In addition, patients with acute brucellosis may have a positive heterophile test. However, the antigens causing this reaction in brucellosis may be absorbed out with guinea pig kidney, and this of course provides a means of differentiating between the two conditions.[8]

Prognosis

Writing about acute brucellosis caused by Brucella abortus, Spink stated that "the duration of the disease, in general, is less than six months and some cases terminate in a state of well-being in a few weeks."[32] Harris, referring to brucellosis in general, believes that it may not be a self limited disease and that it is impossible to estimate the percentage who fail to recover completely, and are subject to repeated relapses. He believes that "the duration of the infection may be limited only by the life span of the individual."

The great majority of cases of acute brucellosis survive as indicated by the case fatality rate for the U.S. from 1942-1949 which was 1.5 per cent. For the same period the rate in Minnesota was 0.2 per cent.[8] The mortality rate in culturally proven cases at the University of Minnesota is about 3 per cent.[32]

Treatment

Prior to the advent of the newer antibiotics, the therapy of brucellosis was eminently unsatisfactory. As each new drug becomes available its usefulness in brucellosis is evaluated.[24,27,35,38,41] For a time the combination of streptomycin and sulfadiazine was found to be most effective.[39] Aureomycin, terramycin and chloromycetin have all been found equally effective in promoting prompt relief of acute symptoms. However, the combined clinical and bacterial relapse rate of 69 per cent makes it obvious that these medications are not curative.[24]

At the present time combined treatment with 2 gms. of dihydrostreptomycin and 3 gms. of aureomycin daily for twelve to fourteen days has given results far superior to any previous method of treatment.[17] In cases with localizing complications the therapy is continued for twenty-four days except that dihydrostreptomycin is reduced to one gram daily. Of sixty cases treated in this manner by Herrell and Barber, there was only one symptomatic relapse and two bacterial relapses. The follow-up period was from three to twenty-four months. No toxic effects of the drugs were encountered.[36] Other investigators have not had such favorable results with this combination.[14]

The fever and toxicity of brucellosis can be controlled quickly with ACTH, but bacteremia persists. Chronic cases are unaffected and granulomas in the liver and bone marrow remain unchanged. The only indication for ACTH in brucellosis is felt to be for control of serious toxic symptoms while antibiotic therapy gets under way.[18]

In the past treatment of the more chronic cases of brucellosis with vaccine therapy has been common. Some investigators believe that this involves a process of desensitization and others contend that a state of immunity is produced which promotes destruction of viable Brucella. In any case, results of this therapy have been extremely variable.[30] It is still advocated in selected cases of chronic brucellosis when response to antibiotics has been disappointing.[28]

Prevention

Vaccination of human beings does not appear to be effective. Pasteurization prevents the milk-borne transmission of brucellosis to human beings. The majority of infections at present, however, result from direct contact with infected animals or their tissues. The eradication of this means of infection is more difficult and is based upon the elimination of infected animals. To accomplish this these animals must be detected and slaughtered. If such a program could be carried out, brucellosis would soon cease to be a problem. Two major obstacles stand in the way. The first is that practical tests for the diagnosis of brucellosis in animals are not capable of detecting the infection in every instance. The second, and more important, is that no uniform nation-wide program of test and slaughter has been instituted. Until these obstacles are overcome we shall continue to be confronted with the problem of brucellosis in clinical medicine.

References

1. Apter, N. S., Halstead, W. C., Eisele, C. W., McCullough, N. B.: Impaired cerebral functions in chronic brucellosis. Am. J. Psych., 105:361, 1948.
2. Barrett, G. M., and Richards, O. G.: Chronic brucellosis. Qrt. J. Med., 22:23-42 (Jan.) 1953.
3. Borts, I. H.: Some observations regarding the epidemiology, spread and diagnosis of brucellosis. J. Kansas Med. Soc. (Dec.) 1945.
4. Braude, A. I.: Spink, W. W.: Studies in pathology and pathogenesis of experimental brucellosis. J. Infect. Dis., 89:272-276 (Nov.-Dec.) 1951.
5. Dalyrmple-Champneys, W.: An epidemiologist's reflections on some laboratory tests in brucellosis. Am. J. Pub. Health, 41:152-57, 1951.
6. De Jong, R. N.: Central nervous system involvement in undulant fever, with the report of a case and a survey of the literature. J. Nerv. & Mental Dis., 83:430-42, 1936.
7. Eisle, C. W., McCullough, N. B.: The treatment of brucellosis. Illinois M. J., 96:241-244 (Oct.) 1949.
8. Feig, M.: Some epidemiologic aspects of brucellosis in the midwest. Am. J. Pub. Health, 42:1253-66 (Oct.) 1952.
9. Feinberg, R. J., Wright, G.: Factors influencing the agglutination titration in human brucellosis. J. Immunol., 67:115-122 (Aug.) 1951.
10. Finch, G. H.: Streptomycin therapy in undulant fever. Am. J. Med., 2:485-490 (May) 1947.
11. Foshay, L.: The laboratory diagnosis of undulant fever. Am. J. Clin. Path., 10:176-185, 1940.
12. Griffits, J. J.: Agglutination and an agglutinin "blocking" property in serum from known cases of brucellosis. Pub. Health Report, 62:865, 1947.
13. Hall, W. H.: Public health aspects of brucellosis. J. Am. Dietetic A., 27:6 (June) 1951.
14. Hall, W. H.: Brucellosis in man—A study of thirty-five cases due to brucella abortus. Minn. Med., 36:440-465, May, 1953.
15. Hall, W. H., and Manion, R. E.: Comparison of the Coombs test with other methods for brucella agglutinins in human serum. J. Clin. Invest., 32: 96-106 (Jan.) 1953.

16. Harris, H. J.: Brucellosis. 2nd ed. New York: Paul B. Hoeber, Inc., 1950.
17. Herrell, W. E., and Barber, T. E.: A new method for treatment of brucellosis. J.A.M.A., 144:519 (Oct.) 1950.
18. Herrell, W. E., and Barber, T. E.: Treatment of brucellosis with aureomycin or terramycin combined with dihydrostreptomycin. Postgrad. Med., 11:476-486 (June) 1952.
19. Howe, C., Miller, E. S., Kelly, E. H., Brookwalter, H. L., and Ellingson, H. V.: Acute brucellosis among laboratory workers. New England J. Med., 236-741, 1947.
20. Huddleson, I. F.: Brucellosis in Man and Animals. New York: The Commonwealth Fund, 1943.
21. Huddleson, I. F.: The relation of brucellosis to human welfare. Ann. New York Acad. Sc., 78:415 (April 10) 1947.
22. Jordon, C. F., and Borts, I. H.: Brucellosis and infection caused by three species of brucella. Am. J. Med., 2:156-167 (Feb.) 1947.
23. Krakauer, H. S., Rockman, R., and Neter, E. J.: The effects of brucellergen skin test on brucella agglutinin titer in patients with brucellosis and other diseases. J. Infect. Dis., 89:56-58, 1951.
24. Killough, J. H., Magill, G. B., and Smith, R. C.: Terramycin, chloromycetin and aureomycin in acute brucellosis. J.A.M.A., 145:553-556, 1951.
25. Nichols, E.: Meningo-encephalitis due to brucellosis with the report of a case in which Br. Abortus was recovered from the cerebrospinal fluid and a review of the literature. Ann. Int. Med., 35:673-693 (Sept.) 1951.
26. Ruiz-Casteneda, M.: Studies on the pathogenesis of brucellosis. Proc. Soc. Exp. Biol. & Med., 64:298-302, 1947.
27. Schaffer, J. M., Kucera, C. J., and Spink, W. W.: Evaluation of prolonged antibiotic therapy in mice with chronic brucella infection due to brucella melitensis. J. Immunol., 70:31-38 (Jan.) 1953.
28. Simpson, W. M.: Brucellosis, advances in therapy. U. S. Armed Forces M. J., 4:337-349, 1953.
29. Simpson W. M.: Brucellosis, In Tice's practice of medicine, 4:99-107, 1952.
30. Spink, W. W.: Diagnosis and management of brucellosis (undulant fever). Vet. Admn. Tech. Bull., TB 10-49, 1949.
31. Spink, W. W.: Human brucellosis. Proc. U. S. Livestock Sanitary Association, Fifteenth Annual Meeting (Dec.) 1946.
32. Spink, W. W.: Studies on brucellosis in Minnesota. Minnesota Med., 33:333-38 (April) 1950.
33. Spink, W. W.: What is chronic brucellosis? Ann. Int. Med., 35:358-373, 1951.
34. Spink, W. W., and (by invitation) Anderson, D.: Studies relating to the differential diagnosis of brucellosis and infectious mononucleosis: clinical, hematologic and serologic observations. Tr. A. Am. Physicians, 64:428-434, 1951.
35. Spink, W. W., Braude, A. I., Casteneda, M. R., and Goytia, R. S.: Aureomycin therapy in human brucellosis due to brucella melitensis. J.A.M.A., 138: 1145 (Dec.) 1948.
36. Spink, W. W., and Hall, W. H.: The influence of cortisone and adrenocorticotropic hormone on brucellosis-II. Adrenocortiocotropic hormone (ACTH) in acute and chronic human brucellosis. J. Clin. Invest., 31:958 (Nov.) 1952.
37. Spink, W. W., Hall, W. H., and Braude, A. I.: Studies on the pathogenesis of human brucellosis. Tr. A. Am. Physicians, 9:126, 1947.
38. Spink, W. W., Hall, W. H., and Magoffin, R.: Follow-up study of therapy in forty-eight culturally proved cases of brucellosis. A.M.A. Arch. Int. Med., 88:419-432 (Oct.) 1951.
39. Spink, W. W., Hall, W. H., Shafer, J. M., and Braude, A. I.: Human brucellosis—its specific treatment with a combination of streptomycin and sulfadiazine. J.A.M.A., 136:382, 1948.
40. Sundberg, R. D., Spink, W. W.: The histopathology of lesions in the bone marrow of patients having active brucellosis. Blood (Supp. No. 1) 1947.
41. Urschel, D. L.: Para-aminobenzoic acid in the treatment of acute brucellosis—a report of four cases. Antibiot. & Chemother., 2:11 (Nov.) 1952.
42. Wise, B.: Acute brucellosis—clinical, bacteriologic and serologic studies of three patients. Arch. Int. Med., 72:346, 1943.

PRESCRIPTION IS WRITTEN FOR "PHYSICIAN-DOLDRUMS"

A prescription has been written by a Philadelphia radiologist which points toward a cure for that virus sometimes contracted by physicians at their many scientific meetings—a disease called "The Doldrums."

The virus is caught from hearing too many long-winded speakers read dry, poorly prepared medical papers.

Offering the suggested therapy is Dr. Robert P. Barden. His prescription is presented in an editorial in the current issue of *Radiology*. The title is, "So You Are Going To Present A Scientific Paper?"

When a doctor arises to read his pet scientific paper, writes Dr. Barden," . . . . from the standpoint of education, entertainment and personal promotion, this may be a catastrophe."

Here are some ingredients in Dr. Barden's prescription:

1. Finish the paper before the deadline. If allotted 20 minutes, speak for 17.
2. Never say all you know, and retire from the podium while your listeners are hoping for more.
3. Talk in headlines. Explain just enough to make the main thought clear.
4. Be simple in concept; terse in expression; logical in progression from one idea to the next.

Concluding, Dr. Barden writes: "After rehearsing the presentation alone several times, stop-watch in hand, try it on one of your colleagues—or even your wife."

Laboratory Aids to Medical Practice

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NECROPSY: ITS RELIGIOUS IMPLICATIONS

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PHYSICIANS occasionally encounter a situation wherein the family of a deceased person objects to necropsy on religious grounds. To meet this challenge and secure permission for necropsy, the physician must be armed with convincing arguments. In view of the sparse literature on the subject and the many misconceptions that exist, the information herein was compiled. The problems will be discussed under the various churches.

Catholic

Necropsy is fully accepted by the Catholic Church, the procedure being mentioned in its writings. In the "Ethical and Religious Directives for Catholic Hospitals," the postmortem examination is sanctioned when done under proper safeguards, which means that proper respect and no desecration be shown to the body. The examination should be made under supervision and to promote science.

Catholics, according to Canon Law 1203, are opposed to cremation. This in no way has reference to necropsy but was mainly intended as a rebuff to certain groups and factions which, at one time, advocated cremation as a means of completely destroying the body and soul so as to avoid payment for mortal sins by such destruction.

In "Moral Limits of Medical Research," Pope Pius XII has expressed indirectly his acceptance of necropsy.

Jewish (Orthodox and Conservative)

The Orthodox and Conservative branches of the Jewish Church are fairly uniform in opposition to necropsy. The basis for their reasoning lies in volume Baba Bathra of the Talmud, which is the book of interpretation of old Biblical laws used extensively by the Jewish people. This volume states that all mutilation of the corpse is prohibited because it involves disrespect to the dead. When buried according to Jewish law, every part of the body must be interred, including even the smallest piece of tissue.

However, there are some exceptions to this general dictum opposing necropsy. These have been summarized from the classic work of Rabbi Leopold Greenwald* as follows:

1. It is most positively prohibited to perform necropsy on bodies of Jewish people or to hand these bodies over to students to dissect the same into sections; these sections will not receive Jewish burial. (This also applies to limited necropsy.) Even if one slew himself, or in the case of sinners who did not repent their sins, this still holds true. However, great authorities have ruled that if one makes the declaration during life that he desires a postmortem examination of his body after demise, then that is permissible.
2. When a governmental law states that all must yield to postmortem examination and the lives of Jews are endangered because of such dictatorship, then necropsy is permissible.
3. If one dies of an unusual disease and physicians desire dissection of the body in order to find some cure for those following him, this is also forbidden.
4. However, if there is before us a diseased person with the same disease of which one died and the physicians make the postmortem examination for the sake of finding a cure for the living one, then the Hachtam Sofer permits it.
5. If the sick one consented, before his demise, to re-

This is the twentieth in a series of editorial reports sponsored by the Minnesota Society of Clinical Pathologists and designed to foster closer relationships between clinicians and pathologists. This paper results from work done by Dr. Tweeddale as a resident in pathology, Lincoln General Hospital, Lincoln, Nebraska.

*Personal thanks and gratitude are extended to Rabbi A. Aaron Segal of Rochester, Minnesota, for translation and summarization.

ceive a postmortem examination or sold his body for such a purpose, it is permissible.

6. If a person died of a hereditary disease and if competent physicians declare that there is hope to save the children of the dead person if a postmortem examination is performed, and if they would examine the cause of such death and would bury all the organs after such examination, then this is permitted.

7. When one is killed and it is desired by means of postmortem examination to find evidence, it is permitted.

Jewish (Reform or Liberal)

In sharp contrast to the beliefs just presented, this faction of the Jewish Church is in favor of necropsy. Their reasoning is as follows:

1. In volume Ketubot of the Talmud a dictum states that any law of the Talmud may be violated for the sake of saving human life except in case of incest, idolatry and murder. Members of this branch of the Jewish Church consider this dictum apropos to the question of necropsy, whereas the Orthodox and Conservative branches do not.

2. The Talmud also states that if one is sick and fails to call a doctor he deserves to die. The Reform group applies this to anything serving to promote well-being and cure the ill, even to necropsy.

3. This group tends to be more liberal in thoughts and ideas, as emphasized by recent Jewish scholars. Dr. Jacob Z. Lauterbach, one of the outstanding authorities on the Talmud, has written in favor of necropsy.

Christian Science

Opposition to necropsy among Christian Scientists is as basic as is their belief. However, one circumstance occurs under which a postmortem examination is sanctioned. Mary Baker Eddy stated, "If a member of The Mother Church shall decease suddenly, without previous injury or illness, and the cause thereof be unknown, an autopsy shall be made by qualified experts."

Under other circumstances necropsy is not recommended. The reason for this stems back to the very nature of the Christian Science belief. In Genesis are found two accounts of man's creation. The first creation was wholly a spiritual one; in the second, man arose from the dust into which God breathed the breath of life. Thus, in the first account man is a true spiritual being; in the second account, accepted by the other religious faiths, man has both a material and a spiritual component. The belief of Christian Science lies in the account of the first creation, in which matter does not exist. Sin, as well as all forms of disease, is considered evil and is nonexistent, according to these concepts. Therefore, necropsy would do no good as it is based on material causes that the Christian Scientists consider as illusions.

Other Protestant Religions

Among the major Protestant Churches one finds a relative unanimity of opinion in the acceptance of necropsy. The Bible in no place mentions necropsy, postmortem examination or the equivalent. Modern church writings usually do not mention necropsy and the attitude of the various churches is that of acceptance. They consider that the indestructibility of the soul militates against any harm that could possibly result from necropsy. One must consider, however, that it is the moral obligation of the physician to perform necropsy in a careful manner, with proper respect exhibited at all times during the procedure. All churches insist on this.

In conclusion, it is apparent that necropsy is usually permitted by most religions in the interest of the advancement of science and for the betterment of mankind. Exceptions to this have been noted.

AMERICAN MEDICAL EDUCATION FOUNDATION

Several large contributions from state medical associations have boosted the American Medical Education Foundation nearer to its goal of two million dollars in 1954. The California Medical Association recently contributed $100,000. Another large contribution came from the Arizona Medical Association in the amount of $7,230, which represents a $10 per member dues increase voted for AMEF by the association. Both Arizona and Utah have followed Illinois physicians by voting a dues increase as a method of increasing contributions from their states.

Since January 1, 1954, the Foundation has received a total of $968,000 and expects to reach the one million mark this fall.

Case Report

SUPERIOR MESENTERIC VEIN THROMBOSIS

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The diagnosis of this catastrophic abdominal emergency is difficult and definitive treatment is often delayed too long. Intra-abdominal venous thrombosis produces a bizarre clinical picture and the diagnosis is often missed or erroneously interpreted as its arterial counterpart.[1,14,15]

We wish to report an interesting case of thrombosis of the superior mesenteric vein.

Case Report

This 70-year-old white female was first seen in consultation at a nearby hospital and admitted to this hospital on October 3, 1953, with complaints of nausea, vomiting, and abdominal distress of four days' duration. These symptoms at first were mild in character but on October 2, 1953, the patient vomited everything she ate and experienced an episode of diarrhea with passage of small amounts of gas which was followed by abdominal distention and labored respirations. There was no remarkable abdominal pain but she did complain of pain in the substernal region. There had been a 25-pound weight loss during the past six months with no history of any similar episode or previous abdominal surgery. Further questioning revealed a history of a posterior myocardial infarction in the winter of 1952, a phlebectomy in August, 1953, at another hospital, and a severe penicillin reaction.

The patient was dehydrated with a pulse rate of 70 and respiratory rate of 24 per minute. The temperature was normal. The blood pressure was 140/90 and the heart was enlarged to the left with a normal rhythm, good tones and no murmurs. The chest was clear. The abdomen was distended with audible high pitched but "distant" peristaltic sounds. There was no tenderness or mass. Pelvic and rectal examinations were negative. Stasis dermatitis of the lower extremities was evident, and there was an old phlebectomy scar on the right.

The red blood cell count was 7,090,000 with 16 grams of hemoglobin. The white blood cell count was 13,100 with 85 per cent neutrophiles. The urine gave a three plus reaction for albumin. An EKG was interpreted as showing an old posterior myocardial infarction. A flat plate of the abdomen at the nearby hospital revealed a distention of the stomach and small bowel and no gas in the large bowel. When repeated on admission to this hospital, after gastric decompression with a Miller-Abbott tube had been accomplished, the findings were essentially unchanged. The tube failed to pass through the pylorus. A chest film showed only a left ventricular enlargement of the heart; lung fields were clear.

An exploratory celiotomy was performed on October 3, 1953. Resection of 55 cm. of small bowel and an ileo-ileostomy were carried out because of multiple scattered areas of dark blue-black discoloration. No evidence of any obstruction was found.

The resected specimen showed patchy areas of blue-black discoloration which were easily visible through the serosa and were more marked in the proximal portion. The lumen contained serosanguinous fluid and soft fecal material. There was slight edema of the walls. The mucosa was intact but raised, due to the underlying linear, oval, and wedge-shaped areas of blue-black discoloration. On microscopic examination no inflammatory cell infiltration or ulceration of the mucosa was apparent. The vessels were engorged with red blood cells, associated with mild intravascular neutrophilic infiltration and beginning vascular organization. In the submucosa, muscularis, and serosa there were areas of recent and old hemorrhage with mild perivascular neutrophilic infiltration.

The patient tolerated the surgical procedure rather well. The carbon dioxide combining power on October 4 was 27 volumes per cent (12.2 mEq. per liter). The hematocrit was 44 per cent and the plasma chloride measured as sodium chloride was 440 mg. per cent (75.2 mEq. per liter). Heparin and dicumarol were begun. On October 5 the temperature had risen to 101.8 degrees. There was loose mucus in the tracheo-bronchial tree but the patient was coughing fairly well. The following day there were coarse râles in the chest, abdominal tenderness, and no evident peristalsis. The temperature rose to 105.2 degrees on October 6 and abdominal tenderness increased. The patient's condition deteriorated rapidly and she died that same evening.

The essential necropsy findings, in addition to a recent intact ileo-ileostomy and bronchopneumonia, were: arteriosclerotic heart disease with cardiac hypertrophy; (a) calcification of mitral ring, grade III, (b) calcific aortic nodular disease with aortic stenosis, grade II, and (c) thrombosis of superior mesenteric vein. The thrombotic process did not involve the portal or lineal vein. There was no demonstrable phlebothrombosis or thrombophlebitis in the veins of the lower extremities. The segment of remaining small bowel was normal. There was no evidence of old or recent myocardial fibrosis or infarction.

Sections from the superior mesenteric vein revealed no sclerosis or inflammatory cell infiltration of its wall. The lumen contained antemortem thrombus material.

Etiology

Venous thrombosis is usually secondary to intra-abdominal infections, neoplasms, surgical procedures, arteriosclerosis, cardiac decompensation, blood dyscrasia, polycythemia vera, trauma, incarcerated hernias, migrating thrombophlebitis, portal stasis, adhesions or bands, volvulus or intussusception. Occasionally it may be primary or of unknown etiology.[1,6,7,11,12,16] An instance has been reported following mumps.[8]

In Johnson and Baggenstoss' review[11] of ninety-nine cases studied in a period of almost thirty-four years, when those whose disease was secondary to strangulation by hernia, volvulus and intussusception were excluded, patients between the ages of forty and sixty-nine accounted for 70 per cent of the cases. Seventy-nine per cent were secondary to either intra-abdominal

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infection, neoplasm or abdominal surgical procedures, and only 8 per cent were primary or of unknown etiology. Berry and Bougas,[1] reported the occurrence of fifty-three cases in 54,000 surgical admissions over a twenty-two-year period. Only eight cases were of unknown etiology, an incidence of primary venous thrombosis of 0.02 per cent of the surgical admissions.

Pathology

The arterial type of thrombosis may be general or segmental and is due either to emboli or thrombi. The venous type is always thrombotic.[7] Since the process is slower, venous occlusion of the mesenteric vessels usually results in hemorrhagic infarction and not gangrene as is seen in the arterial occlusion.[9,14] These hemorrhagic infarcts usually involve the lower part of the jejunum and the ileum[9] but may involve the entire small bowel.[2,3]

Any part of the mesenteric venous system may be occluded. In Johnson and Baggenstoss' series[11] of ninety-nine cases, the superior mesenteric vein or its tributaries were involved in ninety-three or approximately 94 per cent of the cases. Simple thrombosis, both recent and old with organization, was the most common histopathologic process.

The walls of the involved segment are usually thickened, edematous and dark red in color. The lumen contains serosanguinous fluid, and the mucosa may show all stages of degeneration with edema and engorgement of dilated vessels. The other layers reveal a hemorrhagic reaction.[1,9]

Symptoms

The symptoms are those of any other acute abdominal condition with obstruction: pain, vomiting, diarrhea or constipation, abdominal rigidity with tenderness, distention, and borborygmi, fever, melena, shock, leukocytosis, and hemoconcentration.[1,9]

There is often a prodromal period of several days' or weeks' duration characterized by vague abdominal discomfort and occasionally by diffuse intermittent abdominal pain. The onset is usually slow. Mild to moderate colicky pain and tenderness in the lower abdomen on deep palpation are common. The pain is frequently out of proportion to the abdominal findings and is usually not easily controlled by opiates.[1,9] Distention is usually present but tympany is frequently absent because the loops of bowel are filled with blood and fluid. Vomiting occurs in more than one half of the cases and diarrhea or constipation may be prominent features. The latter is more common if the prodromal period is of several days' duration.[1] Hematemesis is a rare sign, but, if present, is very suggestive but not diagnostic. Gross or occult blood in the stool is not frequently seen. With an associated ileus the blood from the small bowel may not reach the large bowel. A clinical picture of shock is rarely seen. The temperature and white blood cell count may be normal or elevated.

Diagnosis

The lack of specific diagnostic findings is probably the reason for the frequent error in diagnosis. Despite the early lack of spasm and rebound tenderness, the condition should be considered an acute abdominal emergency.[9] The differential diagnostic features between arterial and venous thrombosis is that in the latter, the onset is slow with mild to moderate colicky pain, tenderness in the lower abdomen on deep palpation usually without any significant elevated temperature or leukocytosis.[9]

The preoperative diagnosis is frequently that of intestinal obstruction, acute pancreatitis, perforated peptic ulcer, acute cholecystitis and the other acute abdominal conditions. The following criteria may be helpful in making an accurate diagnosis:[9,10,15]

1. A prodromal period of vague abdominal discomfort or severe colicky pain out of proportion to the physical findings.
2. Constipation or diarrhea.
3. Marked abdominal tenderness with little spasm.
4. Normal or slightly elevated temperature.
5. Polycythemia and a high hematocrit.
6. A flat plate of the abdomen showing hoop-shaped, slightly dilated loops of jejunum with serrated margins and/or gas in the proximal half of the colon which stops rather abruptly in the mid transverse colon.

The prognosis is more favorable in thrombosis of the mesenteric arteries[4] and recovery from mesenteric venous thrombosis is rare.[5]

Treatment

The best treatment is early exploratory laporotomy with resection of bowel and mesentery well beyond the site of infarction and primary anastomosis.[13]

The use of anti-coagulants such as heparin and dicumarol are useful adjuncts. Fleming[9] reported a case of mesenteric venous thrombosis which was explored for diagnosis and recovered by use of anti-coagulant therapy alone.

Summary

We have reported a case of thrombosis of the superior mesenteric vein to illustrate that, as compared with arterial mesenteric thrombosis: the onset of venous thrombosis is slower; the abdominal tenderness is out of proportion to the muscular spasm; fever and leukocytosis are usually absent; and, the outcome is much more often fatal.

References

1. Berry, Frank B., and Bougas, J. A.: Agnogenic venous mesenteric thrombosis. Ann. Surg., 132:450-474 (Sept.) 1950.
2. Chodoff, R. J.: Mesenteric vascular occlusion. Ann. Surg., 131:440-444 (March) 1950.
3. Cogswell, H. D.: Massive resection of the small intestine. Ann. Surg., 127:377-382 (Feb.) 1948.
4. D'Abreu, F., and Humble, J. G.: Mesenteric venous thrombosis. Lancet, 1:534-536 (April 13) 1946.
5. Dempsey, W. S., and Jones, T. E.: Venous mesenteric thrombosis. Cleveland Clin. Quart., 15:140-142 (July) 1948.
6. Duncan, D.; Ferrell, T. E., and Hansbro, G. L.: Acute mesenteric venous thrombosis. Ann. Surg., 83:205-208 (Feb.) 1952.

(Continued on page 746)

President's Letter

TOO MUCH TROUBLE?

It is always discouraging to me to read the figures showing the percentage of qualified voters who do not go to the polls in this country.

And it is even more discouraging to feel obliged to urge people to vote on election day.

We, as Americans, should not need to be urged to vote. We should do it automatically, that is, we should consider it an unalterable routine to cast our ballots for the candidates of our choice.

Because we are a nation of free people, we often take for granted that very freedom. We find ourselves lax in the responsibilities of citizenship, when we ought to realize that the freedoms we enjoy carry with them the load of responsibility felt strongly by only a few. The right to choose the men who represent us is one of those rights which embodies the traditional principles of a free democratic people.

Voting is a job of the people. It is a task, to be sure, but it is a challenging one—and one we should be proud to undertake. But voting also has a certain satisfaction to it—it can give the same feeling known after a job well done. It makes us part of the general picture, and can make us feel as if we have done something to influence, in some small way, the trend of things nationally, and on a state and local level.

Now that the primaries are over, and candidates have squared off in campaigns we, as physicians and as citizens as well, must take the role of judges. We must weigh qualifications, study platforms, scrutinize past records, and cast our ballots. Whether we vote as just plain citizens, or as practitioners makes no difference—the important thing is to vote, to shoulder seriously the responsibility that is ours.

It is a well-known fact that the medical profession has not voted regularly; the percentage of those physicians who do vote is shamefully low. Minnesota physicians can help shatter that low record by taking a few minutes on November 2 to go to the polls and become part of the working government by exercising the right of free choice.

President, Minnesota State Medical Association

Editorial

Arthur H. Wells, M.D., *Editor;* Henry G. Moehring, M.D., and John F. Briggs, M.D.

BUILDING FOR THE FUTURE

As YOUR state chairman, it was again my privilege this year to attend the A.M.A. committee meeting of the American Medical Educational Foundation, which was held in Chicago January 24. I was impressed with the personal challenge that faces every doctor practicing medicine in this country. Too many times the AMEF is thought of as another organization on a national basis trying to get a donation from the doctor. He may feel that having supported other worthy medical enterprises on a state level that he will let this plea go unheeded. Perhaps he has not taken the necessary time to familiarize himself with the objectives of this fund and how it can affect him directly.

Briefly, the AMEF is attempting to raise funds from individual doctors, on a voluntary basis, in support of our seventy-nine medical schools who are in dire need of unrestricted funds. In 1953 the University of Minnesota received $28,036.00 from the fund. Such funds are urgently needed over and above those our school gets through legislature appropriations. This leaves the school with many financial deficits to cover such items as augmenting professors salaries, special equipment necessary for training students, residents and fellows, and research. There are also scholarships and student loans that must be considered.

We are living in a competitive world. Therefore, if we wish to maintain high standards of teaching and techniques, as well as the advancement of science through research, our medical schools must continue to be adequately financed. This can only be accomplished through private or government sources. Endowments and state funds cover less and less of the needs each year because of the rapidly increasing costs. Even now the government is furnishing certain funds to our medical school. Funds received from this source during the past year have been $25,000 for improved cancer teaching, $25,000 for improved teaching of cardiovascular diseases and $15,000 for improved teaching in psychiatry. These grants however are for specific purposes and on a year to year basis. Money received from AMEF was used for the following important purposes: salary for a medical artist and illustrator for teaching demonstrations and making charts and lantern slides; employing two animal caretakers for animal research projects; augmenting salaries for an instructor in physiological chemistry, and a teaching assistant in physiology, and salaries for two additional internists returned from military service, one to head the outpatient admissions clinic and the other as an instructor in internal medicine. The remainder of the fund will be used to help buy teaching equipment for the new Mayo Memorial building.

As practicing physicians, we are the recipients of the many new and startling advancements that we have been privileged to add to our armamentarium from year to year. Even if every doctor contributed to the best of his ability in support of this cause, we would not as a group succeed alone. However, we are not alone in this endeavor. Industry has also sensed that it has a large stake in medical education. It too is vitally concerned with industrial health and medicine. Support from this large source depends a lot upon the support the medical profession gives this fund. It has been indicated in a definite way that industry feels that the profession can do a better job than it has done.

It might be of interest to you who take the time to read this, that there are 4,288 doctors registered in Minnesota. In 1953 only 222 doctors in Minnesota contributed to AMEF. The breakdown on this reveals that of the four large medical centers, twenty-nine doctors in Hennepin county, six doctors in St. Paul, sixty-five doctors in Duluth, and twenty-one doctors in Rochester contributed $1,540.00, $170.00, $1,690.00 and $975.00 respectively. Of the remainder of the state, 101 doctors contributed $4,073.00 for a total of $8,449.00. A comparison of 1953 to 1952 contributions shows an increase of from .96 per cent to 5.18 per cent. Not very impressive, is it?

Finally permit me to point out that every physician owes something to his medical school. Aside from the tuition he paid when going through school he has paid nothing for his education. An alternative to the support of the AMEF program would be to cut down on educational standards, or to accept federal support for our medical school. I am sure you will agree that neither the public nor the profession will stand for that. Do you think enough of your profession to back it up with a contribution?

In the near future you will receive information on the American Medical Educational Fund, together with a request for a contribution from you. I sincerely urge every doctor in the state to support this worthy project.

H. E. Drill, M.D.

MEDICAL EDUCATION ON TAPE: PART II

An EDITORIAL in Minnesota Medicine* called attention to the Audio-Digest Foundation of California which is producing tape recordings of reviews of medical literature in various fields, of the proceedings of medical conventions, and of special lectures by renowned authorities. These recordings are then rented or sold to physicians on a non-profit basis.

There is little doubt that the Audio-Digest service can be of real aid to the busy practitioner in keeping up with important advances in medicine. We do, however, wish to take issue with the suggestion made in the editorial that this development will render obsolete and unnecessary the various postgraduate medical education programs available to today's physician. Although audio-visual aids of many types are being used with increasing frequency and effectiveness by educators in many fields, the student-teacher relationship still is—and will continue to be—the basic element in any educational program. This relationship must be close and personal for education is far more than the simple dissemination of factual information. It is a complex reaction for which the experienced, stimulating teacher performs a vital catalytic function. If this were not so, the printing press would long since have solved the problem of education; not only the phonograph but the teacher as well would be unnecessary. Spontaneous discussions and the question-and-answer sessions are essential features of any teaching program. Medical education, like the practice of medicine itself, cannot become an assembly line or "ready-to-wear" proposition.

It is particularly difficult for us to see how the physician could place sole reliance on recorded lectures for extending his knowledge in certain fields of which electrocardiography, endoscopy, radiology, and hematology are a few examples. These and many other fields require well-prepared demonstrations in addition to the opportunity for the student—undergraduate, graduate, or postgraduate—to exchange viewpoints with the instructor.

The editorial also asked the reader to choose whom he would prefer to hear discuss rheumatic heart disease, Paul D. White or Joe Doke of his own city. The answer it naturally evoked was "Why, Paul D. White, of course." The question should, however, have been stated as follows: "Would you prefer hearing Paul D. White on tape recording, or Joe Doke in person, where he will be available after his formal talk for answering questions and discussing particular points of interest?" The question thus stated, particularly if considered in light of the possibility that Joe Doke may himself be a pretty fair cardiologist and a good speaker, might bring about a less unanimous response.

We do not wish to convey the impression that we believe that the Audio-Digest is without value. As stated at the outset, we believe that is a really significant development and one which will be of real help to a great number of physicians. We do not believe, however, that physicians will be willing to use the Audio-Digest service to the exclusion of all other means of continuing their education. We do not believe that it spells doom for postgraduate medical education programs. For many years, excellent records which teach German and other foreign languages have been available, yet students still register for courses in these subjects in our colleges and universities. The availability on records of the lovely music from "South Pacific" with the voices of Ezio Pinza and Mary Martin did not, to our knowledge, make tickets to that production less sought after. If anything, these records interested more people in seeing the play. Perhaps Audio-Digest will in the long run have a comparable effect.

Robert B. Howard, M.D.

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*Medical education on tape. Minnesota Medicine, 37: 213, (March) 1954.

TOO MANY MEETINGS

THERE ARE too many medical meetings. This is especially true in the larger cities. The very purpose for which meetings were primarily instituted is defeated. They teach the profession less and less, and waste more and more of the already deficient time in the average physician's day.

Every hospital besides its general monthly meeting has meetings of various committees, specialties and laboratories. Some hospitals average a meeting a day. Many counties hold a meeting once a month. Besides the state Medical meeting usually held once a year, there are various medical national clubs, societies and organizations devoted to the specialties which also hold meetings monthly, quarterly, semi-annually or annually. Thus a physician placed on two or three hospital staffs and forced to attend the absolutely necessary meetings, to be in good standing both in the hospitals and his medical groups, may not find time for all the meetings.

Since there are so many meetings often the material presented is not clinical but historical in scope and sometimes boring by too-frequent repetitions. Every physician knows very well, that discoveries in medicine worthwhile do not occur daily, weekly or even monthly. The material at such meetings frequently falls below the average expectations. It is only on rare occasions that the physician finds something new and valuable. For want of well-rounded-out speakers at some meetings, as well as interesting topics to be presented, occasionally both speaker and material are also poorly prepared.

To remedy all the above conditions, only a few measures would be necessary. A reduction in the number of meetings would be most helpful. Then fewer speakers would be required and thus a better choice could be selected. The material too could be improved by omitting ancient historical discourses and devoting more time to recent discoveries, methods of treatment, drugs and helpful procedures. Moreover, if some methods could be improvised to expand the ego of many an ambitious physician without creating another medical society, there would not be a constantly growing list of superfluous medical organizations, unnecessary or useless journals and inevitably increasing number of medical meetings. If some of these medical committees, groups, societies or organizations could be eliminated, or amalgamated with others, there would also be a reduction in meetings.

Such measures would tend to lessen the strain on the physician, conserve his time, prolong his life, preserve his health and yet keep him well informed in medicine with some spare time for recreation. The patient too would receive better care for less expense.

J. A. Lepak, M.D.

We close this month's editorial page with an anonymous poem that should epitomize the feelings of many a Minnesota physician about this time of year:

THE DUCK HUNTER

The poor duck hunter in his blind
Is chilled in front and wet behind,
It's seven hours since he fed,
And twenty since he's been in bed.
It's cost him near a hundred bucks,
To hide here from the silly ducks,
Which presently, ere day dawns dim,
Will rise and hide themselves from him!

SUPERIOR MESENTERIC VEIN THROMBOSIS

(Continued from page 742)

7. Durand, B.: Mesenteric thrombosis. Am. J. Surg., 78:338-343 (Sept.) 1948.
8. Elkan, W.: Acute mesenteric vascular occlusion following mumps. J. Internat. Coll. Surgeons, 20:259-268 (Sept.) 1953.
9. Fleming, J. W., Jr.: Mesenteric vascular occlusion. J. Missouri M. A., 48:530-533 (July) 1951.
10. Harrington, L. A.: Mesenteric thrombosis. Am. J. Roent., 58:637-640 (Nov.) 1947.
11. Johnson, C. C., and Baggenstoss, A. H.: Mesenteric vascular occlusion; study of ninety-nine cases of occlusion of veins. Proc. Staff Meet. Mayo Clin., 24:628-636 (Dec. 7) 1949.
12. North, J. P., and Wollenman, O. J., Jr.: Venous mesenteric occlusion in the course of migrating thrombophlebitis. Surg., Gynec. and Obst., 95:665-671 (Dec.) 1952.
13. Seybold, W. D., and Musgrove, J.: Surgical aspects of mesenteric vascular occlusion. Proc. Staff Meet., Mayo Clin., 25:585-591 (Oct. 11) 1950.
14. Storer, J.; Kazdon, P., and Belton, J. L.: Venous mesenteric thrombosis. Am. J. Surg., 84:636-640 (Dec.) 1952.
15. Ross, J. R., and Kepkay, P. H.: Portal venous thrombosis. Lahey Clin. Bull., 8:85-88 (Jan.) 1953.
16. Warren, S., and Eberhard, T. P.: Mesenteric vascular thrombosis. Surg., Gynec. and Obst., 61:102-121 (July) 1935.

Public Health

MINNESOTA'S FACILITIES FOR CARE OF THE CHRONICALLY ILL AND THE AGED

The Minnesota Department of Health has a special interest in the aging population because chronic illness occurs most frequently in this group. It is, to a considerable extent, preventable, because of our responsibility for licensing institutions for the care of older people, and because of authority for the administration of the Hill-Burton hospital construction program vested in the State Board of Health. Since the program started in 1946, the health department has aided in the costs of constructing and equipping 43 facilities with the more than $13 million in Hill-Burton funds which has been made available in Minnesota Now the program is being expanded to include nursing homes, rehabilitation facilities, diagnostic or treatment centers, and places a greater emphasis on chronic disease hospitals.

Minnesota has a very urgent problem in planning for the care of its older people. We now have approximately 300,000 people who are sixty-five years of age or older. This represents 9 per cent of our total population. Only ten states have higher percentages of people in this age group. Many aged persons require some period of institutional care, the period varying from a few days to many years. It is estimated that 10 to 20 per cent of the beds in our general hospitals are now occupied by long-term, chronically ill persons. The general hospital is usually not equipped to provide optimum care for such patients, and their presence in large numbers in general hospitals restricts the number of beds available for acute cases.

The solution to this problem involves the provision of additional chronic disease and nursing home beds in connection with general hospitals. Proximity to the general hospital assures ready availability of good medical and nursing care, of diagnostic and treatment facilities, and also obviates the necessity of duplicating many services such as kitchen, laundry, and boiler plant. Chronic disease units need an adequate staff, a fully-equipped physical therapy department, occupational therapy, and other such services that will encourage maximum independence and rehabilitation. Rehabilitation should begin when the patient enters the hospital and should be based on a careful diagnosis and evaluation of the patient's capacities. The greatest problem in relation to this program is the present shortage of well-trained personnel.

St. Louis county has already provided 5.07 beds per thousand of its population and is supplying valuable information on the number of beds needed for the care of the chronically ill and aged. When this figure is applied to the state as a whole, it is found that Minnesota needs a total of 15,121 beds in this category. At present Minnesota has 338 institutions for the chronically ill and aged, with 10,947 beds. Many of these existing beds (5,694 or 52 per cent) are located in non-fire resistive quarters and should be replaced. The actual shortage of 4,174 beds does not reflect the increased growth and aging of our population; the chronically ill, long-term patients now in acute general hospitals; the proportion of patients in state mental hospitals that might be discharged to communities if proper facilities were available; and the number of aged and chronically ill persons now living in hotels, lodging houses, and similar places, who need care.

In co-operation with the Commission on Chronic Illness, the Minnesota Department of Health has joined eight other state health departments in making a thorough survey of long-term patients receiving nursing care in institutions. This survey will give us considerable information as to the characteristics of these patients and the kinds of care they require. It will also indicate the amount of medical and nursing care they receive and the source of payments for care.

The committee on health, chronic illness, and rehabilitation of the Governor's Commission on the Aging is recommending to the 1955 State Legislature that state aid be provided to assist counties and other non-profit groups in constructing and equipping modern, fire-resistive nursing homes. The amount of $500,000 for each year of the biennium will probably be requested to supplement the very nominal appropriation that is anticipated as Minnesota's share under the recently amended Hill-Burton law. In expending available funds during the next two years, it is proposed to give high priority to those nursing homes that are closely affiliated with general hospitals to assure adequate medical and nursing supervision as well as good programs of rehabilitation. This is not a new departure for Minnesota, since in 1913 the State Legislature appropriated funds to assist in the construction of county tuberculosis sanatoria. We have an even greater problem today with our aging population. The funds proposed above will not build many beds at present construction costs, but at least a start will be made toward the provision of good facilities at the local level.

Helen L. Knudsen, M.D.
Chief, Section of Hospital Services
Minnesota Department of Health

SCHOOL OF PUBLIC HEALTH IN NEW QUARTERS

When the University of Minnesota's school of public health moves to its new quarters in the newly-completed Mayo Memorial building this fall, it will also have embarked upon its second decade as an organized professional school for training medical and technical personnel in the several fields of public health and preventive medicine.

Organizationally the youngest of the schools in the University's college of medical sciences and one of ten such schools in the United States, it has already won an international reputation as a training center for public health practitioners, educators and administrators.

Students from nearly every state and territory of the United States and from the far corners of Asia, Africa, Europe and South America have flocked to Minnesota to participate in its program; and an average year's enrollment boasts representatives from thirty states and fifteen foreign countries.

The principal work of the school of public health is the training of professional specialists in the major phases of public health work, but its distinguished faculty, under the directorship of Dr. Gaylord Anderson, has also contributed significantly to the study and solution of problems of contagious disease, nutrition, metabolism, physiology, psychosomatics and many another medical question.

Members of the faculty too have found time to bring the message of public health to civic and professional groups in countless speaking engagements, to sponsor and participate in frequent health and medical conferences, and not the least to keep the people of Minnesota informed of modern health and safety practices through radio and television appearances.

Most widely known of these services are Dr. Stewart C. Thomson's weekly radio talks over the Minnesota School of the Air. Housewives, heeding his warnings of dangers in the home, have cajoled their husbands into repairing faulty household equipment.

To these gentlemen, public health work is more than a mere occupation; rather they count themselves as modern bearers of a Minnesota tradition which long antedates the foundation of their school. For well over a half century they and their predecessors have been in the forefront of a dedicated crusade among the state's medical and lay leaders to achieve a strong and progressive program of public health. In large measure too, this crusade and the movement which culminated in the foundation of the school of public health in 1944 goes back to the last quarter of the nineteenth century.

Public Health History

In those years the technique, even the concept, of large scale public health programs was novel. Medicine itself was just beginning to enter upon a new age of discovery and advancement, but disease was still able to spread almost unchecked. Every year 5,000 Minnesotans perished from the ravages of typhoid fever. Influenza, diphtheria, scarlet fever and tuberculosis killed and maimed additional thousands.

Such everyday modern health measures as control over sewage and waste was completely inadequate; and the idea of public filtration and control over water supplies was too revolutionary to win much public support. Even the city of Minneapolis did not begin to filter its water until 1908 and to chlorinate it until 1910.

The beginnings of the long fight for better public health services in Minnesota go back to the indefatigable Dr. Charles Hewitt of Red Wing—a man whose own life personified the inseparable union between public health training, general medical education and a publicly-supported, statewide public health program.

In his own personal war against disease, the good doctor took to manufacturing and distributing his own smallpox vaccine. And for many years, he taught courses in public health to University of Minnesota students without pay, leaving his private practice in Red Wing at regular intervals to perform what he considered to be a public duty.

It was Dr. Hewitt who first brought before the state legislature the project for creating a state health service and who was responsible in 1872 for Minnesota becoming one of the first three states in the nation to establish a state board of health. It was he also who drafted and presented to the University's board of regents in 1882 the plan for the creation of a medical department at Minnesota.

After the initial victories of Dr. Hewitt and his supporters, public health education and preventive medicine followed two major lines of development in the state. The state board working through the medical profession and through public and private health agencies, sought to encourage citizens and communities to adopt better health and sanitation procedures. And within the medical school, training in public health and preventive medicine became an integral part of every course of instruction.

This was the beginning. In later years as the scope of the medical sciences expanded and the demand grew for greater specialization in the medical profession, the idea of specialized instruction in public health began to take hold. It received further impetus both from the surge of medical research at the beginning of the present century and from the affiliation in 1915 of the medical school with the Mayo Foundation in Rochester.

But the crucial fact was the tremendous need for more effective and more widespread medical service for all the people of Minnesota. In 1915, it was still the destiny of one Minnesota mother in every fifty to perish in the throes of childbirth; and almost one baby in ten did not survive the first year of life. Contagion, despite conspicuous progress in the fight against smallpox, typhoid fever and other diseases, continued to take a heavy toll among children and young adults.

Organization Expands

Such was the backdrop for the next surge in the development of public health training in Minnesota. In 1918, the medical school, supported by a special legislative grant, began to offer a special three-month course in public health nursing. Four years later, this course, one of the first of its kind to be established in the United States, was expanded to nine months.

During that same year, the increasing demand for specialized health education and for trained leaders in public health led to the creation in the medical school of a separate department of preventive medicine and public health. One of the original instructors was Dr. Harold S. Diehl who was to become dean of the college of medical sciences in 1935.

The ensuing decade saw the gradual addition of new functions to the training and research program of the new department. In the mid 1930's the United States Public Health Service chose Minnesota as one of its centers for public health training; and in 1937, Dr. Anderson, who already had made a distinguished career of public health work in Massachusetts, came from Harvard to head the department.

The year 1944 witnessed the reorganization of the department as the school of public health; and the postwar period saw the addition of professional training courses in hospital administration, public health engineering, vital statistics, public health education and public health veterinary medicine. In 1946 also, Dr. Ancel Key's famous laboratory for physiological hygiene, which had been organized in 1937, was made a division of the school.

School Outgrows Facilities

With this multiplication of teaching activities and the accretion of other new responsibilities, the school had outgrown its facilities in Millard Hall and found itself compelled to resort to makeshift arrangements to carry on its work. The ultimate solution was found in the allotment of space in the newly constructed Mayo Memorial building; and when the school takes up its new quarters on the eleventh and twelfth floors of the building, it will bring together units which previously were scattered through five widely separated buildings.

As it is presently organized, the school of pub-

(Continued on page 752)

Medical Economics

Edited by the Committee on Medical Economics
of the
Minnesota State Medical Association
George Earl, M.D., Chairman

PR INSTITUTE WELL ATTENDED

The AMA Public Relations Institute, held in Chicago, September 1 and 2, had a record number of 250 registrants from all states. The meeting covered various aspects of public relations, from national, state and county medical organization levels, including such subjects as how direct mail should be handled, a television production seminar, public relations and the medical assistant, working with the allied professional fields, and the over-all fee situation.

One of the more interesting sessions of the meeting was entitled "What's the Score on Fees?" It included panelists Joseph Donovan, San Jose, executive secretary of the Santa Clara County (California) Medical Society; Dr. Walter L. Portteus, Franklin, president-elect, Indiana State Medical Society; and, Boyden Roseberry, White Plains, executive secretary, County of Westchester (New York) Medical Society.

Prior Agreement Necessary

Mr. Donovan described the "usual" fee plan of his county medical society, which was based on a survey of average and median fees of county physicians, which was tabulated to provide what he called an index of "usual" fees in the county. The county society has adopted this fee index, he said, but with the stipulation that the doctor has the freedom to charge more or less than the index *as long as there is prior agreement with the patient.*

Dr. Portteus stated that medical fees are one of the most intimate problems to the patient and his family. He stated that prior discussion of fees with the patient can obviate much of the misunderstanding and pave the way to payment. It also tends to eliminate cases of over-charging, he said. He distributed a pamphlet which he has prepared and gives to all his patients who are to be hospitalized. The pamphlet gives the patient a concise preview of what he should expect in medical care and hospital service, the medical team which will care for him, and the type of bill he will receive. It puts on paper what many doctors only discuss with their patients, and has an excellent public relations value for the doctor and his patient.

Mr. Roseberry discussed the medical social worker and the physicians' fees. He described his county society's program of using a medical social worker in conjunction with its Bureau of Medical Economics and its Mediation committee. The Bureau handles cases of inability to pay, dissatisfaction with care, dissatisfaction with fees. All these cases are referred to the medical social worker for investigation and report.

The Mediation committee handles all complaints that come to the society, through the social worker, but about 90 per cent are settled before referral to the committee, Mr. Roseberry reported. He stressed the value of the medical social worker in hearing the patient's complaints, stating that they do not, therefore, require that the complaint be put in writing. Any committee decisions adverse to the patient are handled by the social worker, who sees the patient and can explain personally why the decision was so made. The patient can then ask questions, and feels that he has had a better chance to have a complete hearing of his complaint.

BOARD SUGGESTS ORDINANCE TO PREVENT NURSING HOME FIRES

The National Board of Fire Underwriters announced recently a new edition of its "Suggested Ordinance on Nursing, Convalescent and Old Age Homes." The ordinance regulates safety measures for such homes, and is designed for adoption by municipalities and may also be used as a basis for state or county regulations.

Notes Lives Lost

Within the last ten years four of the more tragic fires in nursing and old age homes took the lives of eighty-eight persons, a bulletin from the Board states. "The record shows that the loss of life in those and many other fires in similar homes resulted from several causes, the most important of which were the use of highly combustible fibreboard on interior walls and ceilings, unenclosed stairs and dumbwaiters, lack of sufficient exits, inadequate fire separation for parts of buildings containing special fire hazards, and lack of automatic sprinkler protection."

Sounds Warning

The Board states that some nursing, convalescent and old age homes have modern buildings of fire-resistive construction, but a great many existing homes are in buildings of combustible construction. Many of them are in buildings originally built for use as private dwellings. The Board points out:

> "Large wooden frame dwellings of two or three stories in height provide reasonable fire safety when occupied by a single family, but when used for housing as many persons as can readily be contained in them when converted to nursing, convalescent or old age home occupancy, the hazard to life from fire is greatly increased. This is because of the larger number of persons subject at any one time to danger from any fire that occurs, and because the occupants of these homes frequently are not able to get out as readily as the members of a single family, and because the chances of fire increase as the number of occupants increases in any given space."

SOCIAL SECURITY PASSAGE CALLED "SEVERE SETBACK"

The Association of American Physicians and Surgeons has recently termed the passage of the Social Security expansion bill a severe setback for American liberty.

Because Section 106 was included in this bill, the Association feels that it is "unquestionably the biggest single step towards socialized medicine ever accomplished by any Administration." The Association's bulletin states: "The innocent sounding clause, 'to protect the benefit rights of disabled workers,' provides for doctors to certify permanently and totally disabled workers subject to rules and regulations of the Secretary of HEW (presently Mrs. Hobby) and to be paid out of the Social Security 'trust fund.' In other words, physicians who participate in this nefarious scheme to capture the American medical profession, will be government contract doctors—or participating slaves in socialized medicine as far as medical certification is concerned."

Asks Repeal of Section 106

Inclusion of Section 106, compared to exclusion of physicians under the law, is a small victory, the Association declares. Its bulletin states:

> "The small victory is the exclusion of physicians from compulsory Social Security. However, Washington reporters believe that the present Administration will 'get physicians' next year because President Eisenhower and Mrs. Hobby believe that they should have the 'protection of Social Security for their old age.'"

In urging physicians to work for repeal of Section 106, the AAPS states: "This open door to socialized medicine must be closed before the power-hungry and greedy bureaucrats within the Department of HEW are able to start the enslavement of physicians as government contract practitioners. . . When our lawmakers understand the full import of Section 106—its treachery, its definite long step to socialized medicine—they will rescind this horrible piece of legislation, because most of them are not in favor of imposing inferior medical care on the American people by way of socialized medicine. However, it is up to physicians and their friends to help the lawmakers to understand."

In conclusion, the Association says:

> "In the meantime, we recommend that physicians scorn the provisions of Section 106, continue to care for their own patients, refuse to participate or accept any federal pay in the same manner as many doctors did when EMIC was in effect during World War II."

REPORT DESCRIBES HEALTH BEHIND IRON CURTAIN

Communist boasts of good health and medical care are almost entirely untrue and hide the core of the problem—the "human aspect of medicine," a study by the Free Europe Committee, Inc., shows.

Walter H. Nelson, New York, director of magazine information for the American Heritage Foundation, reported on physicians behind the Iron Curtain in a recent issue of the *Journal of* *the American Medical Association* . His report is based on findings of the Free Europe Committee.

According to the report, the Iron Curtain doctor is a "new Soviet man" first, and a physician last. He must consider his worker-patients as "economic factors," precious only so far as their health advances the world-wide Soviet power policy. Nelson said:

> "Under Soviet-sphere state capitalism, plant physicians predetermine the number of persons entitled to be sick. Physicians are afraid to grant sick leave to too many persons, for this would tend to show that they are not taking sufficient care of the workers' health. Yet poor diet, constant nervous tension, and excessively high work norms make adequate care difficult."

Official Private Practice

Nelson reports that while physicians are officially allowed to practice privately, few do because of the required "exhausting" day's work at state institutions, because of exorbitant taxes on private income, and because few persons can afford private consultation. He notes that private practice probably is allowed only because "Communist bosses hesitate to patronize the state institutions, the conditions of which they know only too well."

Politics Interferes

Medical students are accepted by class origin and political reliability, the report says, and must pass not only medical courses but tests on politics and military principles. They are not allowed to choose their specialties—the Five-Year Plan determines what kind of physicians are needed and where, and a committee annually selects students for each field.

Nelson reports that medical science also has suffered by the outlawing of certain Western scientific principles. If a doctor gets good results with a "forbidden" treatment, he must credit some other cause, or pretend the treatment was just introduced, "with a gigantic 'Made in Russia' tag attached."

PUBLIC HEALTH

(Continued from page 749)

lic health offers professional instruction in seven fields of public health as well as numerous courses for non-professional and medical and nursing students. Behind this stands a broadly based program of research and public service, dedicated to the end of better and more widespread medical services for all the people of Minnesota and the nation.

In the larger sense, the years of growth which have seen public health education come of age in Minnesota have been years of great progress in medicine's long struggle against the hazards of accident and disease. Today less than one mother in 500 dies in child birth and communicable disease has ceased to be a dread menace to the young. More and more the problem of public health has become the problem of combatting accident, communicable disease and chronic infection among the middle aged—the men and women forty-five years and older—who today account for more than 80 per cent of the premature deaths in Minnesota.

That public health education should contribute equally and in full co-operation with the medical profession and the state public health service in the effort to solve this and other present-day problems of medicine, is the objective and the reason for the existence of the school of public health. These goals are in keeping with the aims of the new Mayo Memorial building which itself is dedicated to the provision of better medical services for all people through training, research and service in medicine.

MINNESOTA STATE BOARD OF MEDICAL EXAMINERS

230 Lowry Medical Arts Building
Saint Paul 2, Minnesota

F. H. Magney, M.D., Secretary

LICENSE OF HARRY C. BROWNE, JR., M.D., SUSPENDED FOR THREE YEARS

On July 16, 1954, the Minnesota State Board of Medical Examiners suspended for a period of three years the medical license held by Harry C. Browne, Jr., M.D., because of his personal use of liquor and the various barbital compounds. Dr. Browne had previously appeared before the Board for similar misconduct at which time he was permitted to retain his medical license by entering into a stipulation with the Board in which he agreed to restrain from the personal use of liquor, narcotics and barbiturates. However, when it was learned that Dr. Browne had violated the terms of his agreement with the Board his license was suspended.

Dr. Browne was born in Portland, Oregon, in 1910 and received an M. D. degree from the University of Oregon in 1935. He then served a one-year internship at University Hospitals in St. Louis, Mo. In 1937 Dr. Browne was licensed to practice medicine in Minnesota. At the time his license was suspended Dr. Browne was not in the private practice of medicine but was doing institutional work.

MINNEAPOLIS WAITRESS GIVEN FOUR-YEAR PROBATION TERM FOR ABORTION

Re: State of Minnesota vs. Lucille M. Burke.

On September 13, 1954, Lucille M. Burke, 1335 La Salle Avenue, Minneapolis, Minnesota, a thirty-three-year-old divorcee, was sentenced by the Hon. Thomas Tallakson, Judge of the District Court of Hennepin County, to a term of not to exceed four years in the State Reformatory for Women at Shakopee, Minnesota, on an abortion charge. The sentence was stayed however, and the defendant was placed on probation for a period of four years. On August 7, 1954, Mrs. Burke, a waitress at a Minneapolis night club, had entered a plea of guilty before Judge Tallakson to an information charging her with the crime of abortion.

The defendant, who has had no medical training and who holds no license to practice any form of healing, was arrested by a Minneapolis Police Officer at 2:30 A.M. on August 3, 1954, after she returned to a restaurant to claim a package containing her abortion instruments which she had left there by mistake a short time previously. Earlier that evening, Mrs. Burke had performed an abortion by using a catheter, upon a Minneapolis woman who is married and has a family. Only $90 of the agreed price of $150 for the abortion was paid to the defendant at that time, however, and the balance was to be paid within two weeks. The woman upon whom the abortion was performed did not become ill but was taken to General Hospital by police as a precautionary measure. According to the records, the defendant does not have a prior conviction for abortion.

"YOGI" HEALER SENTENCED TO JAIL FOR VIOLATION OF BASIC SCIENCE LAW

Re: State of Minnesota vs. Shelly Trimmer.

On September 7, 1954, Shelly Trimmer, thirty-six years of age, who lives on Woman Lake Road near Hackensack, Minnesota, entered a plea of guilty in the District Court of Cass County at Walker, Minnesota, to an information charging him with practicing healing without having a certificate of registration in the basic sciences. Following a statement of facts to the Court, Trimmer was sentenced to a term of one year in the Cass County Jail, by the Hon. Arnold C. Forbes, Judge of the District Court. Judge Forbes suspended the balance of the sentence after the defendant has served 30 days in jail, on condition that Trimmer refrains from violating the law and complies with the rules and regulations prescribed in connection with his probation. Trimmer must come back into court in the spring of 1956 at which time it will be determined whether or not his behavior in the meantime justifies a permanent suspension of the sentence.

A criminal complaint against Trimmer was signed by a representative of the Minnesota State Board of Medical Examiners after an investigation disclosed that on August 7, 1954, he had treated a four and one-half month-old baby and that the baby had died on the following day. Trimmer, who admitted in court that he is not registered in the basic sciences or licensed to practice any form of healing, furnished a green liquid medication and some pills for the infant but an analysis of these failed to show that they contained any harmful substances and when a postmortem examination of the baby was done it was found that the cause of death was interstitial pneumonia. Trimmer made a charge of $3.00 for his services in caring for the child.

In response to questioning by the Court prior to being sentenced, Trimmer said he was born in Erie, Pennsylvania, on October 27, 1917, and that he studied Yogi philosophy and healing for one year under Paramhansa Yogananda in Encinitas, California. When questioned concerning his method of healing, Trimmer stated that he determined through "psychic impressions," which he received in this case by running his fingers over the infant's spine, that the nerves from the brain to the rest of the body were not responding properly. Although Trimmer's attorney made a plea to the Court not to sentence Trimmer to jail on the grounds he was a spiritual healer, Judge Forbes told Trimmer that "if he had confined himself to spiritual means he would not have violated the law but people who defy laws must expect the punishment to be severe when it deals with the lives of babies."

History of Medicine In Minnesota

THE PIONEER DOCTORS OF CHIPPEWA COUNTY TO THE YEAR 1900

LEON G. SMITH, M.D.
Montevideo, Minnesota

CHIPPEWA COUNTY may well claim the distinction of being the residence of the first civilian Doctor of Medicine to practice in the state of Minnesota, namely, the **Rev. Thomas Smith Williamson, M.D.**

There were army surgeons located in the state before him, the first one probably being Surgeon Edward Purcell. The first army post was called Camp Coldwater, established at the site of what is now Mendota, in 1819. The following year, the construction of Fort Snelling was begun.

There are records of a devastating scourge of scurvy at Camp Coldwater that first winter of 1819-1820, during which almost half of the command perished. Mention is made of Dr. Purcell sending to the St. Croix River for a quantity of spruce twigs in order to prepare an infusion to combat the scurvy. Dr. Purcell is listed as serving at Camp Coldwater, Fort St. Anthony and Fort Snelling through 1823; his death is recorded in 1825.

The name of another doctor appears in 1832, when **Dr. Douglas Houghton** accompanied Henry A. Schoolcraft's expedition to explore the headwaters of the Mississippi River. Dr. Houghton acted as physician for the exploring force, but his most important task was to vaccinate the Indians against smallpox. His salary was reported to have been $3.00 per day.

The first exploration of western Minnesota appears to have been in 1823. At that time an expedition, under Major Stephen H. Long, proceeded by canoe (as all such expeditions at that time preferably traveled by water) up the Minnesota River as far as historic Traverse des Sioux. This was the ancient fording place of the river on the old trail between the upper and the lower Sioux villages, not far from the site of what is now St. Peter. Due to low water and a frequently obstructed river channel, the canoes were abandoned here, and the journey continued by land along the south bank of the river. Joseph Renville, about whom we shall learn more later, acted as guide and interpreter of this expedition, which continued up the river past Big Stone Lake, where it crossed to the east bank and continued north, down Lake Traverse and the Red River of the North as far as Pembina.

In the account of the expedition, no mention is made that any part of what is now Chippewa County was visited, but with the Minnesota River no wider a stream than it is at this point, with the water level as low as described, undoubtedly some members of the party crossed the river. Thus, 1823 probably is the date that Chippewa County was first visited by white men.

According to Folwell's "History of Minnesota," the Sioux (or Dakotah) nation was made up of seven tribes, three of which, the Teton, Yankton, and Yanktonai, lived upon the great plains to the west of Minnesota. The other four tribes had their homes in Minnesota, and were nicknamed by their western relatives "Isanti" (Knifemen). They, in turn, were made up of the Lower Sioux and the Upper

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The History Section will appear in quarterly installments from now on.

Sioux, so named from their geographical location. The Lower Sioux consisted of the Mdewakanton tribe, situated around Fort Snelling and down the Mississippi River as far as Winona, and the Wahpekute tribe, centered near what is now Faribault. The Upper Sioux consisted of the Wahpeton and Sisseton tribes, and it was these two tribes who occupied western Minnesota along the Minnesota River. Occasional bands of marauding Chippewas made their way down the Chippewa and Pomme de Terre Rivers from their wooded domain to the north, but in the main this was Sioux country.

On May 16, 1835, a band of missionaries, consisting of five adults and three children, arrived at Fort Snelling under the leadership of the Rev. Thomas S. Williamson. Dr. Williamson, at the time thirty-five-years-of-age, was born in Union County, South Carolina, March 8, 1800. He was a graduate of Jefferson College in Pennsylvania and of Yale Medical School. His wife, Margaret Poage, was born September 10, 1803, in Mason County, Kentucky. They were married at Ripley, Ohio, April 10, 1827, where Dr. Williamson had located, and where he had practiced medicine for ten years. To this marriage, three children, William Blair, Mary Poage and James G., were born.

Dr. Williamson's father was a Presbyterian minister and had tried to induce his son to become a medical missionary, but Dr. Williamson and his wife saw no reason for leaving the comfort of a settled home and community to face the rigors of the wilderness. In a short space of time, however, all three children died of malaria, and influenced by this, Dr. and Mrs. Williamson decided that "God had work for them to do elsewhere."

Accordingly, he returned to school, spent a year at a theological seminary and received his credentials as a medical missionary from the American Board of Missions. During this time, another child, Elizabeth Poage Williamson, was born. He then organized his mission party consisting of himself, his wife and child; his wife's sister, Sarah Poage; and an agricultural expert and teacher, Alexander Huggins, with his wife and two children. This party arrived, as before stated, at Fort Snelling, May 16, 1835.

It had been Dr. Williamson's plan to settle near Lake Calhoun, where the Pond brothers, Samuel and Gideon, had established a mission a year or two earlier. But, it so happened that, at just that time, Joseph Renville arrived at Mendota to sell his year's take of furs and buy his annual supplies of flour, salt, sugar, and coffee.

Renville was a person of great importance in western Minnesota, and he left the imprint of his personality upon much of the early history of the region.

He was a half-breed, the son of a French trader and a woman of the Kaposia band of Sioux. He had been educated in a convent school in Canada; had held a captaincy in the British army in the War of 1812; had served as an interpreter and *coureur de bois* for the North West Fur Company; had later transferred into the employ of John Jacob Astor's American Fur Company; and, finally, had established a trading post in the Wahpeton Sioux country, upon the northeast side of the Minnesota River, shortly below its exit from Lac qui Parle. Here he built a stockade and group of buildings called Fort Renville (in the fur company's records, Fort Adams) and in it he lived like an old-time baron, maintaining a small army of voyageurs and Indian and half-breed relatives. He taught them to plant and to keep corn, and to accumulate horses, cattle and sheep. He was extremely hospitable and, to the end of his life, among the western Minnesota Indians and breeds, his word was law.

Lawrence Taliaferro, the Indian agent at Mendota, had been having considerable trouble trying to keep peace between the Chippewas and Sioux. Thinking it might be a good idea to have a sub-agent in the Lac qui Parle area, he invited Renville to dinner, and suggested that he, Renville, might do well to invite Dr. Williamson and his party to locate near him. Since Renville readily perceived the advantages to him and his people of having close to him a combination of missionary, doctor and teacher, in the person of Dr. Williamson, and an agricultural adviser and teacher, as was Mr. Huggins, he readily agreed. He, in turn, quickly induced Dr. Williamson to change his mind about staying at Lake Calhoun. Instead, he and his group joined Renville's party to return to Lac qui Parle.

They left Fort Snelling June 23, in the fur company's large Mackinaw boats, well laden with trader's supplies. There was a good stage of water, and fair time was made by using oars, poles, even at times sails, and by pulling on willows along the banks. At Traverse des Sioux, they were met by wagons and Red River carts with oxen from Fort Renville, and the rest of the trip was made by land, leaving the Minnesota River near the mouth of Hawk Creek and cutting across overland by Black Oak Lake (four miles east of the present Montevideo), fording the Chippewa River near the present site of Watson, and reaching Fort Renville July 9, 1835, seventeen days' journey from Fort Snelling.

This date is important to this chronicle, as it marks the time of location of the first resident, civilian doctor in Chippewa County, and probably in the State of Minnesota as well.

Our history of Dr. Williamson in his new location, naturally is much more detailed regarding the missionary than the medical side of his activities, but there is no doubt that, in the succeeding years, he completely fulfilled Renville's hopes for success for him in his multiple role of preacher, teacher and doctor of the Indian people.

Dr. Folwell, in his "History of Minnesota," says:

> "Dr. Williamson was a man of intense but simple piety, possessed of strong common sense and indefatigable industry, absolutely devoted to the Indian life."

He was the first missionary to the Sioux people. In the short time he had been at Lake Calhoun, he had organized the first Christian church in Minnesota, June 12, 1835. It was named the "First Presbyterian Church of St. Peters" and its membership of twenty-two was made up mostly from the garrison at Fort Snelling.

Very soon after reaching Lac qui Parle, he organized a Presbyterian church there, with three members, which was shortly joined by Renville's wife and others of her family. Mrs. Renville was the first of pure Sioux blood to profess and die a Christian. Later, Renville himself, though raised and educated as a Roman Catholic, joined the church and became a ruling elder.

As Dr. Williamson was unable to speak the Dakotah language, he recognized the value of one who could; he, therefore, persuaded Gideon Pond, who could speak it, to leave Lake Calhoun and join him at Lac qui Parle.

The spring following Williamson's arrival, in 1836, work was started on building a large log mission close to Renville's stockade. It was within sight and hearing of Lac qui Parle, so named by the early French voyageurs as the "Lake that Talks." It is said that, when the wind is from the right quarter, the waves, breaking upon the stones, result in a distinct, musical note, thus giving the lake its melodious name.

Also, under Mr. Huggins' direction, the squaws developed and cultivated cornfields across the river along the flat, rich river bottoms.

The mission was about 200 miles from Fort Snelling. Mail came at intervals of three to five months, though at not such prohibitive postal rates as one would suppose, the rate being only 25 cents per letter. More urgent messages were dispatched by Indian runners. Once a year, at least one of the missionaries would go down to the Fort, accompanying Renville's party for supplies.

Three months after the Williamson's arrival at Lac qui Parle, another child, John Poage Williamson was born, October 27, 1835, the event occurring in a dirt floor cabin, in Renville's stockade. John undoubtedly was the first white child to be born in Chippewa County or, for that matter, in western Minnesota.

The Indian name for Dr. Williamson was Payjehoata Wechasta, meaning medicine man or, literally, grass-root man, namely doctor.

Following the completion of the mission, Dr. Williamson started the most important work of his life, the translation of a large part of the Scriptures into the Sioux language.

Again quoting Dr. Folwell:

> "In Renville and Gideon Pond, he found efficient guides and helpers. The work was carried on in the living room of Fort Renville, with members of the bodyguard seated on the surrounding benches, smoking their pipes. Williamson read the French version from the great family Bible of his host. Renville, accustomed to rapid interpretation, promptly dictated the Dakotah translation to Gideon Pond, who acted as scribe."

The following year, in September, 1837, a young minister, Stephen R. Riggs, joined the Williamson band, and from here on, most of our information comes from his writings, particularly his "Forty Years with the Sioux" and "Mary and I."

Joseph Renville died in 1846, and with his strong influence removed, there was much "backsliding" of the Indian converts. Shortly thereafter, Dr. Williamson was invited to move to the village of Little Crow at Kaposia, near the present site of South Saint Paul. This was probably due to the fact that the Lac qui Parle Indians had many relatives at Kaposia, so that the reports of his good work among them had reached Little Crow.

By this time, the Williamsons had several more children. They had probably recognized the disadvantages of living longer among the Wahpeton Sioux, who had been getting out of control since Renville's death, as well as the advantages of moving the family to a more civilized environment.

They moved to Kaposia, where he conducted a mission for several years; transferred back to the Minnesota Valley at a station near the Yellow Medicine Agency; remained there until after the Sioux Outbreak of 1862, after which, most of the Sioux were moved out of Minnesota. He and his wife then moved to St. Peter where they lived the rest of their lives, except for extensive traveling. Mrs. Williamson died July 21, 1872, and Dr. Williamson several years later, June 24, 1879. He continued work upon the translation of the Bible up to the last, even after he was confined to bed. Their home is still standing in St. Peter, or, at least it was in 1935. Both are buried in the old Traverse des Sioux cemetery, a few miles from St. Peter.

Hearing that a great-grandson of Dr. Williamson's is teaching music in Canby, Minnesota, I communicated with him and he graciously furnished me with the later history of the Williamson family, subsequent to its leaving Lac qui Parle.

There were seven children, in addition to the three who died as infants in Ripley, Ohio:

Elizabeth Poage, the child who accompanied her parents from Ohio, was married to Andrew Hunter in 1858 and died in St. Peter, March 11, 1863.

John Poage, the first child born in Chippewa County, was the grandfather of my informant. He became a missionary to the Sioux Indians and was stationed at the Redwood Agency at the time of the outbreak. Fortunately, he was visiting in Ohio at the time. Later he went with the Minnesota Sioux when they moved to the Fort Thompson Reservation in South Dakota. He continued to work as a missionary at the Yankton Reservation near Greenwood, South Dakota, where his son Gary and his grandson, Andrew, were born and where he died, October 9, 1917, after fifty years of missionary work with the Indians.

Andrew W. was born January 31, 1838, at Lac qui Parle. He became professor of mathematics at Gustavus Adolphus College in St. Peter and Augustana College in Rock Island, Illinois. He never married. He died in Portland, Oregon.

Nancy Jane was born at Lac qui Parle, July 28, 1840, and died at Greenwood, South Dakota, November 27, 1877.

Smith Burgess, born September 21, 1847, at Lac qui Parle, was dragged to death by an ox team at Yellow Medicine Agency, March 11, 1856, at the age of nine.

Martha, born October 5, 1844, at Lac qui Parle, died at Gresham, Oregon.

Henry Martin, born March 1, 1851, at Kaposia, Minnesota, died at Portland, Oregon.

After Joseph Renville's death, his trading post was taken over by Martin McLeod, a Scotchman who married a Sioux woman. He appears to have been a capable man, and in later years was elected to the state legislature and was prominent enough to have had a Minnesota county named for him.

The old mission at Lac qui Parle, after Williamson's departure, was conducted for at least part of the time by Stephen Riggs, but he was away working in other fields much of the time. In 1854, when the mission burned, the remaining badly depleted personnel was transferred to Dr. Williamson's station which was then at the Yellow Medicine Agency.

Several years ago, a replica of the mission was built upon its original site on the banks of the Minnesota River, about four miles west of Watson, where it was formally dedicated July 12, 1942.

There was, for all practical purposes, no government in Minnesota prior to 1836. At that time the Wisconsin Territory was organized, which included that part of Minnesota lying east of the Mississippi River. West of the river, whatever government existed was that administered by the Indian tribes, the Chippewas controlling that portion of the state lying in the wooded areas of the north and east, and the Sioux in charge of the prairies of the southwestern part.

In May, 1848, the Honorable John Catlin, as acting governor of Wisconsin Territory, called a special election in that part of the territory north and west of the St. Croix and Mississippi Rivers, and the Honorable H. H. Sibley was elected as delegate to the Congress of the United States to represent that area.

On March 3, 1849, the Territory of Minnesota was organized, in much its present form, with Saint Paul as the capital and the Honorable Alexander Ramsey as first territorial governor.

In 1851, three treaties were made with the Sioux and Chippewas, whereby large tracts of land were relinquished to the United States. The Treaty of Traverse des Sioux, with the Sisseton and Wahpeton tribes, July 18, 1851, acquired the land from Lake Traverse east to the Mississippi, except for a reservation extending ten miles from the Minnesota River upon either side, and running one hundred miles along its upper length. It was in this area that present Chippewa County lay. When first organized, it was split off from Renville County and included Swift County, and did not attain its present size until 1870, when Swift County was detached by act of the legislature. This action was ratified by the inhabitants of both counties at the next general election.

At the time the earliest settlers began to arrive, this was, of course, virgin country and a hunter's paradise: the habitat of buffalo, deer and elk; of fox, prairie wolf and occasional timber wolf; of badger, beaver, muskrat, mink and raccoon; of quail and prairie chicken; of plover, kill-deer and meadow-lark; and, of innumerable varieties of migratory water birds, snipe, duck, goose, swan and sand-hill crane. Many of these are still with us, but the buffalo, elk, timber wolf, prairie chicken, and sand-hill crane are seen no more in western Minnesota, and the prairie wolf, beaver, badger, quail and plover are rare.

In prehistoric times, it is thought that a tremendous body of water, Lake Agassiz, covered the entire Red River Valley region, and instead of draining north, as now, it drained to the south and east through the River Warren, and it was the erosion by this stream that formed the broad valley of the present Minnesota River. Roughly limiting this basin are two great terminal moraines, left by the glaciers of the Ice Age. Upon the southwest side is the line of hills running for many miles in a southeasterly direction from South Dakota into Minnesota near the Lac qui Parle-Yellow Medicine County line, well known to the early voyageurs as the "Coteau des Prairies." Forming the northeast side of the watershed is a similar conformation called the "Leaf Hills," running across Swift and Pope Counties, and the affluent streams that feed the upper Minnesota all rise in these hills.

Chippewa County lies wholly within the Minnesota basin. The shape of the county is roughly a triangle, with the hypotenuse its southwest border, lying along Lac qui Parle and the Minnesota River. The river's largest affluent stream from the north during its entire length is the Chippewa River, which bisects the county, and with its tributaries of Shakopee Creek and Dry Weather Creek, drains most of it. A portion of the more rolling western part drains to the Pomme de Terre, and some of the low, flat, slough country of the eastern part drains through Hawk creek into the Minnesota River below Granite Falls. A peculiar residual from the early River Warren action, is the Watson Sag, a low, marshy area cutting across from the Chippewa River to Lac qui Parle, whereby in high water times, the river may flow in that direction as well as in its normal course, thus making the elevated Watson Prairie an actual, large island.

Most of the land has been drained, so that now there are, in addition to Lac qui Parle, only two lakes in the county: shallow Buffalo (in early days, Shakopee) Lake in the northeast part of the county, and small, deep, rock-bordered and picturesque Carlton Lake, lying in the Minnesota Valley just below Montevideo. When settlers first came, however, there were many large sloughs, some with wooded shores, large enough to merit the name of lakes. The native trees were cottonwoods, willow, oak, elm, hackberry, black and burr oak and the thickets were of wild plum and hazel nuts.

Of these lakes, Black Oak was often mentioned in early history as a picnic spot and an overnight bivouac point upon the cut-off to Lac qui Parle from the regular Minnesota River trail. It lies four miles east of Montevideo, but for fifty years it has been drained and its bottom used as farm land. Other lakes mentioned in the early records were Willow, Dry Weather, and Lone Tree (also called Bad Water), all in the eastern part of the county.

As settlers first moved in (like the Indians, trappers and voyageurs before them), their route was usually along the water-courses. By 1857, the old Military Trail from Mendota, along the south bank of the Minnesota River, through the last settlement of any size, New Ulm, past the Lower Agency, the Upper Agency, and the Lac qui Parle Mission to Big Stone Lake, had been cleared and improved to the point where it was called a road. Some travel was still carried on by water, but most of it had been taken over by the Red River cart, a vehicle famous enough to warrant special mention. It was two-wheeled, very crude but very strong, made entirely of wood and leather, with not one particle of metal used in its construction. As no grease was used on the axles, and movement was wood upon wood, it is said that the groaning of these carts could be easily heard for miles.

At the time of the Sioux Rebellion, which erupted in August of 1862, the only settlement in Chippewa County consisted of two men, an Englishman and a Frenchman, living a short way down the Minnesota River from the mouth of the Chippewa, near present Carlton Lake. The Indians are supposed to have attacked without warning and killed one of the men, but the other, though wounded, managed to escape. In 1865, the claim was taken over by Walter Carlton, and, in the cellar of the log cabin, which was still standing, he found the remains of the man who had been killed.

We know that the Amos Huggins, who came as agricultural adviser to the Inians with Dr. Williamson, when Lac qui Parle Mission was first established, was still living near the mission at the time of the massacre, probably across the river beside his corn fields. He, with his wife and a Miss Le Fambois, acted as teachers to the Indians. Huggins still had a family of two children.

August 19, 1862, while he was working in the field, two Indians came to the cabin and waited for him. When he returned with a load of hay, without a word, they shot him down in cold blood. Why they killed him but spared the rest of the family, no one will ever know.

Mrs. Huggins was allowed to take a few small belongings, including Mr. Huggins' missionary Bible, and she (with her children and Miss Le Fambois), escaped, first to the home of a half-breed Chippewa named De Cota, who in turn took them to the lodge of a friendly Sioux, named Walking Spirit. He, in turn, helped them across the river to the home of a settler named Lagree. They reached his home the day of the attack upon Fort Ridgely, eighty miles down river, but still, so fast did news travel by word of mouth, that they heard all about the battle the same day.

From here Miss Le Fambois, joined and assisted by her brother, and disguised as a squaw, made her escape down to the settlement.

Mrs. Huggins and her children were soon asked to leave by Lagree, as he was afraid to shelter them longer. They returned to De Cota, but when he refused to take them in, they again made their way to the lodge of Walking Spirit. He and his wife befriended them and protected them from the other savages, until General Sibley and his military force pushed the Indians ahead of him up the valley and forced the release of all their prisoners at Camp Release between September 26 and 30. Reports of the number of prisoners vary, one estimate being 162 half-breeds and 107 whites, mostly women and children, among whom were included Mrs. Huggins and her two children.

After the backbone of the Rebellion was broken in 1862, settlers began to move into the country again, slowly at first, but soon in rapidly increasing numbers.

The first permanent settlement in the county was made, at what they called Chippewa City, by Walter and James Carlton and D. W. Wilkins. This was directly across the Chippewa River from what is now Montevideo. The first house was built in 1865. In 1868 the village was platted, probably attaining the size of a half dozen houses by 1870.

The first Board of County Commissioners was named by the Governor in 1869, which meant that the county must have had a population, by then, of twenty-five qualified voters, as that minimum was necessary in order to qualify as a county. The first county seat was located in Chippewa City.

However, by the following year, a new and larger village had sprung up on the east bank of the river and this was named Montevideo.

Various stories as to the derivation of the name are extant. A fallacious one is that it was named after Montevideo, Uruguay, but this theory has been advanced only since the development, during the last few years, of the Fiesta theme, and, with it, the good-will accord between the South American and the North American Montevideos. Our early settlers included several men of considerable learning, and, it is said that one of these Latin scholars, reputedly Lycurgus R. Moyer, stood—one day—upon the east bluff of the Chippewa River, looking down over the beautiful valley below him, and, possibly exaggerating the height of the hill on which he stood, brought forth the name, "I See from a Mountain," or in Latin, "Monte Video."

The real founder of the village was G. W. Frink. In 1866, he had made his first trip up the Minnesota River from the settlements, by ox-team. Again, that same fall, he came with his twelve-year-old son, probably Oscar, who was later to be the first boy from the county to study medicine. He described these early trips very graphically in a letter in the *Montevideo Leader* of June 21, 1895.

He told of their camping in the timber along the river, with a rifle always at hand, constantly on guard against possible attack by the savages.

His third trip, from his home in Morristown, Rice County, was with a party of thirteen, eight of whom were women and children. This was early enough in March so they encountered bad snow-storms, and much of their time was spent, so he said, in floundering, wallowing and shoveling in the snow, finding refuge some nights in settlers' cabins and some nights at deserted shacks. Finally, after seventeen days of struggling, they reached the G. W. Wilkins house in Chippewa City. All were exhausted and chilled, and some had frozen fingers and toes; consequently, even a sod house looked like a palace to them.

I quote from his letter: "Everything needed for existence had to be brought from New Ulm, 85 miles away. The prairie sod had to be broken. But what a delight it was to see the country settle up, to hear the lowing of cattle, the tinkle of the sheep bell, the sound of hammer and axe, and finally the puff of the Iron Horse. The Indian and buffalo are gone, replaced by civilization. The schoolhouse replaces the teepee, the church bell the savage war cry. A civilized and a prosperous community now occupies that splendid prairie region, and we feel an abiding interest in the town and country we helped build out of the wilderness."

(To be continued)

Minnesota Academy of Medicine

Meeting of January 13, 1954

The regular monthly meeting of the Minnesota Academy of Medicine was held at the Town and Country Club Wednesday, January 13, 1954. Dinner was served at 7 p.m. and the business meeting was called to order at 8:20 p.m. by the President, Dr. E. A. Regnier.

There were forty-one members and two guests present.

Minutes of the December meeting were read and approved.

The Secretary read the minutes of the Executive Committee meeting of December 9, 1953.

Votes were requested on the four new amendments to the Articles of incorporation and By-laws, and these were carried.

Dr. Regnier announced the death of Dr. Philip Donohue.

Joseph A. Maun of the firm of attorneys Bundlie, Kelley, Finley and Maun, St. Paul, was detained in the East, and his paper on "The Physician and his Taxes" was given by William R. Busch, an attorney of Mr. Maun's firm.

THE PHYSICIAN AND HIS TAXES

WILLIAM R. BUSCH

Saint Paul, Minnesota

Taxation is today such an integral part of our business and personal life that there is hardly a facet of our existence which is not affected, and to some degree regulated, by at least one of the many and diverse forms of revenue laws which the federal, state and local law making bodies have adopted. Thus regard for a moment the fact that the deed to your home and to your other real estate, the stocks and bonds in which you have invested, the safe deposit box in which you keep your valuables, the gasoline which you burn in your automobile, the tobacco you use, the telephone and cable service which you employ in your business and personal affairs, along with virtually countless other items, are all subject to federal taxation, and in addition, in most instances, to a state levy of substantial size.

The most important taxes, however, are the ones that are levied on the incomes we earn, the property we dispose of by gift, and the estate we leave at our death. Of these three, the income tax has beyond question the greatest significance. First of all, its rates are extremely high, and it is of never ending application. (Even death does not toll this tax, for immediately upon the taxpayer's death, a successor taxpayer comes into being in the form of his estate and it must return a tax on all income received after death which is not subject to income taxation in the hands of the taxpayer's heirs.) Secondly, and more importantly, the income tax is so widespread in application and complex in construction that a taxpayer simply cannot afford to proceed oblivious of its effects on his activities. With a graduated tax rate that starts at 20 per cent and ranges as high as 91 per cent, it seems clear that the unwitting loss of an allowable deduction, or the taxation as ordinary income of what is in reality a return of capital, or some other of the mis-steps which so frequently occur in the cases of unwary taxpayers, will at least unnecessarily dissipate the taxpayer's resources and may produce financial disaster where the amount involved in the lost deduction, the return of capital or whatever the item may be, is substantial in size.

If there is to be any meaning to the concept of equality in justice, remember that laws must be interpreted and applied by the courts in a consistent fashion and in accordance with the terms of the statute involved. Therefore, if you permit your transactions to be in such form that they fit within a provision of the law that was never intended to apply to them with the result that a larger tax than would otherwise be due is assessed against you, do not anticipate any relief from the courts. The courts have said again and again that although the taxpayer need not choose the most expensive way or form of operation from a tax point of view, having made his choice he will be bound to pay the tax consequences that follow.

It is the object of this discussion to cover some of the highlights of the vast body of law that has developed around the federal and state income tax statutes particularly as applied to the special problems confronting the medical practitioner.

1. *Adequate records are essential to a sound tax program.*

Perhaps the most fundamental need which the medical practitioner, or for that matter any business or professional man, is faced with in connection with his income tax matters, is the necessity for reasonably accurate accounting records. If these are not available in a form which will substantiate the correctness of the gross income which you report and the deductions which you claim, then you are needlessly prejudicing yourself and are paving the way for troublesome tax controversies. Incomplete or otherwise inadequate records give the

Commissioner of Internal Revenue the license to reconstruct your income and your deductions in accordance with his best judgment in the matter. Needless to say, his determinations will tend to favor the government.

One of the first things any examining revenue agent will do when checking the tax return of a doctor, is to compare the receipts reported in the return with the deposits disclosed by the doctor's bank statements. If these cannot be reconciled (and in the absence of detailed cash receipts records and a uniform procedure of depositing receipts in the bank, it is unlikely that it can be done) you will have a difficult job of explaining to do if you are to avoid being taxed for additional income.

The only really satisfactory approach to this record-keeping problem is to have a simple but complete accounting system installed in your office and then see to it that it is operated so as to produce a permanent record of your business receipts and disbursements with a minimum of accounting frills. Any costs incurred in this regard should be many times repaid by the elimination of the tax controversies you are otherwise almost certain to experience.

Among the standard steps that are normally involved in placing a medical practice on an efficient bookkeeping basis are two that are particularly significant from a tax point of view. The first consists of completely separating your personal and business checking accounts so that only business items clear through your office account. The purpose of this is to eliminate the possibility of personal expenses being mistakenly included in business deductions and at the same time to reduce the record-keeping chore of the person who handles the cash disbursements records for your practice.

The second step consists of establishing an inviolable rule that all business receipts must be deposited intact in your business bank account. The frequency with which the deposits are made will depend to some extent on your billing and collection methods. But in any event, they should be deposited at least once a week. Every dollar received as business income should find its way into your bank account and under no circumstances should any expenses, whether business or personal, be paid directly from receipts. If you follow this recommendation, you should experience no difficulty in explaining the relationship between your business income and the deposits to your bank account.

Historically, medical practitioners have determined and reported their income on the cash basis of accounting. In most instances, this is the only feasible method because of the uncertainty of collections. Having elected to report your income on this basis, all payments that you receive in any given year represent taxable income for that period without regard to when the services for which the payment was made were rendered. This means that it is possible for your taxable income to fluctuate substantially from year to year even though your total services rendered maintain a fairly constant level throughout the cycle. This is one of the inherent shortcomings of the cash basis of accounting, but fortunately it does not occur often enough or in a sufficiently pronounced form to make it a serious problem. If you have contemplated changing to the accrual method of accounting under which you pick up the income as the services are rendered, be certain to note that the Commissioner's approval must first be obtained.

To complete your bookkeeping requirements, it is necessary for you to have a record of all items of non-business income that you receive during the year. If your office staff is capable of handling it, this chore can be most painlessly carried on as an adjunct to your business books. Separate accounts can be established to record the non-business income and the receipts themselves can be deposited in your business checking account. If you cannot arrange your affairs in this pattern, be certain to maintain some permanent log so that when tax return time arrives you will have a correct record of your total non-business income.

One last point that should be noted in connection with your income tax records is that the best way to insure the full allowance of all deductible business expenses is to pay them by check. The same rule applies to personal items which the Internal Revenue Code permits you to deduct in computing your income tax liability. This is particularly so in the case of contributions. If they are supported by cancelled checks they will be beyond challenge; if they have been paid in cash, you will more often than not find it virtually impossible to obtain their full allowance where they are substantial in size.

2. Tax returns must be properly prepared and timely filed if penalties are to be avoided.

The object of your bookkeeping system is to produce an easily substantiated statement of your business receipts and expenses, so that when you prepare your income tax return you will be in a position to do so with the assurance that only adequate records can supply. The tax return itself is in reality nothing more than a specially designed statement of income and disbursements. Yet for most of you, it is probably as difficult to digest as is a medical chart to those of us who are uninitiated in your profession. For this reason, a good many doctors, particularly those who have been through the mill, as it were, with a revenue agent, have found it most economical in the long run to seek specialized assistance in preparing their returns. Whether or not this is necessary in your particular case will depend to a large extent upon the size and complexity of your income and the extent to which you are willing to risk potential tax difficulties.

Because of the specialized nature of a tax return, it will serve no good purpose to devote any time to considering the details of the various returns which you are required to file each year. However, because substantial penalties will attach if you fail to comply with the filing requirements, a word or two appears in order as to the rules covering the filing of returns.

You are all undoubtedly long since reconciled to the fact that by March 15* of each year your state and federal income tax returns covering the preceding calendar year must be filed. But this is only half the job, for you are also required to file a declaration of estimated tax on your income for the current year. Failure to file this declaration by March 15th will result in an immediate penalty of 5 per cent of each installment due but unpaid. This penalty increases to a maximum of 10 per cent depending on the length of time elapsing before your declaration is filed.**

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*The 1954 Internal Revenue Code changed this filing date to April 15.

Hand in hand with the requirement of a declaration is the statutory provision specifying that your estimated tax must be at least 80 per cent of your final tax liability for the year.† If it is not further penalties are encountered. In general, these amount to 6 per cent of the difference betwen the estimated tax paid and the actual tax liability.

Because of the magnitude of these penalties where the ultimate tax liability is substantial, a very real burden is placed on the practitioner whose total income is exempt from the withholding tax and hence entirely subject to the estimated tax provisions of the Code.

There are, however, two avenues of approach, either one of which if correctly followed should eliminate any chance of a penalty being imposed. The first, which is specifically provided for in the Internal Revenue Code and is by far the safer of the two, consists of determining your estimated tax liability for the current year on the basis of the immediately preceding year's income and deductions and the current year's tax rates, exemptions and dependency credits. If the installments of the tax estimate determined in this fashion are timely paid, no penalty can attach even though your actual net income for the year and your resulting tax liability far exceeds what has been estimated.

The second procedure designed to eliminate underestimation penalties consists of filing your declaration on or before March 15 disclosing a tax liability of a more or less arbitrarily chosen amount and then amending this estimate by January 15 of the next year to bring the total estimate to at least 80 per cent of your actual tax liability. Some people have used the so-called dollar estimate approach which consists of declaring an estimated tax of a nominal amount payable in four equal installments.§ So long as such a declaration is amended to increase it to 80 per cent or more of the year's actual tax liability by January 15 of the next year, a literal compliance with the Code requirements appears to exist. However, it would seem discrete to declare a more realistic amount than this lest the Commissioner attempt to penalize you for violating the substance of the Code requirements. Although it is perhaps doubtful that the Commissioner would be successful in making this penalty stick, the taxpayer is nonetheless risking its assertion and the expense incident to the resulting tax controversy.

A final point to be noted in connection with the estimated tax penalties is that the penalty for underestimation applies to the difference between your estimated tax and your tax liability as ultimately determined and not as disclosed in your tax return.‡ Thus, if a substantial portion of your deductions are disallowed, or if the examining agent succeeds in including additional income, you will be subject to an underestimation penalty if your estimate was set at the bare minimum of 80 per cent of your reported tax.

Before turning to the substantive aspects of the tax law, it seems worth while to briefly consider the matter of how long the records in support of your tax returns should be retained. To a large extent this is a function of the statute of limitations applicable to income tax matters. Ordinarily this limitation period is three years from the date the return is filed. However, if you omit income in excess of 25 per cent of the gross income disclosed in your return, the limitation period is automatically extended an additional two years.§§ For this reason it is best to retain your records for at least five years after the return for the year in question is filed.

Since the statute of limitations on contract indebtedness is six years in Minnesota, your cancelled checks covering the payment of your expenses should be retained for at least that period of time to protect you against the possibility of having to pay a debt twice. This is of course a matter of local law and does not concern your income taxes, but it should be kept in mind in determining when you can safely dispose of your old records.

3. *Certain types of receipts are not subject to income taxation.*

In considering the various substantive aspects of our income tax laws it seems logical to start with a summary of the principal types of receipts that are excluded from taxable income.

One of the most widely discussed of these is the interest which is paid by a state or local governmental body on its bonds. Such interest is specifically exempted from federal income taxation and for this reason the yield on these bonds is very measurably less than the yield on a comparable corporate bond. Thus to a person in the 50 per cent bracket, a 3 per cent municipal bond will produce as much net income after taxes as will a corporate security on which the cash income runs at the rate of 6 per cent a year.

In connection with these tax exempt municipals it should be borne in mind that the Minnesota income tax

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**This penalty is not found in the 1954 Code.

†The rules governing the penalty for substantially underestimating tax liability have been completely redrafted in the 1954 Code. Thus now the estimated tax must be only 70 per cent or more of the tax disclosed on the return filed for the period involved. In addition, the penalty which is at the rate of 6 per cent a year on the amount of each underestimation is not to be imposed if the estimate equals 70 per cent of the tax liability shown on the last year's tax return or 70 per cent of the tax that would be due at current tax rates and exemptions on the basis of the net income shown on last year's return.

§This is no longer permitted. The 1954 Code measures under estimations for each quarterly installment so that each payment seemingly must equal at least one-fourth of the minimum estimate tax payment required for the year.

‡This has been changed by the 1954 Code so that the tax shown on the return filed for the year is the measuring stick for the estimated tax requirements.

§§Under the 1954 Code, the extension is three years so that the total limitation period becomes six years in such cases.

law does not extend the exemption to bonds issued by other states or their subdivisions; hence, if you invest in outstate municipals you can properly exclude the interest from your income only in calculating your federal tax liability. For state tax purposes such interest is subject to the same rules as that received on ordinary securities. It should also be noted that the profit or loss which is realized on the sale of a municipal bond is not tax exempt and is includible in income in the same manner and under the same rules as gains or losses on the disposition of any other capital asset.

A second type of non-taxable income is the proceeds of a life insurance policy which matures by reason of the death of the insured. Where the right to receive the proceeds of the policy was acquired by purchase, a slightly different rule applies but this is a situation you are not likely to encounter.

If the insurance settlement provisions call for monthly installments instead of a lump sum payment, taxable income is not realized even though the aggregate of these installments exceeds the face of the policy at the time of the insured's death.* On the other hand, if the policy proceeds are retained by the insurance company at interest with a periodic payment of the interest to the beneficiaries, the total of these payments is taxable income as and when received, even though substantially the same monthly payments could be scheduled under the installment settlement provisions of the policy and be entirely non-taxable.

This divergency in tax treatment is well worth considering, particularly where the amount of insurance is large and the income tax bracket of the recipient significant. There are, of course, instances where good estate planning calls for the retention of the principal of the life insurance policy in order to adequately protect the secondary beneficiary. In such cases the tax disadvantages of this procedure cannot be avoided. However, ordinarily a periodic or installment settlement provision can be arranged which will produce approximately the same result as will retention of the policy proceeds at interest. In these situations the tax economy of the former method will normally dictate its use over the latter type of settlement option.

One further point, with reference to life insurance, concerns the so-called dividends which the mutual companies pay each year on their outstanding policies. These dividends are regarded for tax purposes as reductions in the policy premiums which is in reality what they are. Consequently, these dividends are not includible as taxable income regardless of whether they are received currently in cash or permitted to be accumulated and paid out when the policy matures.

Another important type of non-taxable income arises in connection with annuities and other insurance policies which mature for reasons other than the death of the insured. Payments received under such policies represent taxable income in part and a non-taxable return of capital to the extent of the remainder. The rule is that so much of the annual annuity as is not in excess of three per cent of the annuitant's cost basis in the policy is treated as taxable income. The balance of each year's receipts from the policy is exempted from tax until the full cost basis has been recovered. Thereafter, the entire amount received is taxable as ordinary income.** If the policy is surrendered for a lump sum payment, no income is realized except to the extent that the amount received exceeds the total net premiums which have been paid on the policy. Any excess is taxable as ordinary income and is not subject to being treated as capital gain regardless of how long the policy has been held.

Among the other types of non-taxable receipts that you are likely to encounter are: (1) damages or awards for personal injuries; (2) veteran's benefits; and (3) gifts and inheritances. The latter class of receipts is subject to special types of taxation which we will briefly review towards the end of this discussion.

4. *Capital gains and losses are subject to different treatment from ordinary income.*

The next phase of income taxation that appears to warrant consideration is the favorable treatment in the form of a substantially lower tax rate which is accorded gains or losses realized on the sale or exchange of capital assets.

In general, a capital asset is any property, whether or not used in a trade or business, which is not part of the taxpayer's stock in trade or his inventory and is not a literary, musical or artistic composition or a copywrite thereof which the taxpayer produced through his personal efforts. Governmental obligations which are issued at a discount without interest (e. g., the Treasury's E bonds) are specifically denied capital asset status.

Depreciable business property (such as your office equipment and your business automobile) is subject to a special rule. If it has been held for more than six months prior to the time of disposition, the taxpayer has the privilege of treating the gain from the disposition of such property as a capital gain, provided he similarly treats the gains and losses from all such depreciable property sales and any property involuntarily liquidated during the period. If a net loss is suffered on the sale of the depreciable assets, this loss is deductible as an ordinary business expense.

The procedure for handling capital gains for tax purposes has recently been modified so that now short term capital losses and long term capital gains are offset one against the other at one hundred cents on the dollar.

In addition, a net long term capital loss is no longer subject to any percentage reduction, and may be carried forward in its entirety against the capital gains and income of subsequent years to the extent that it exceeds the maximum capital loss deduction of $1,000 in the taxable year in which it occurs.

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*The 1954 Code has eliminated this tax saving possibility by subjecting to an income tax the excess of the aggregate installments over the lump sum value of the policy at death. An exception is allowed in the case of the surviving spouse of the insured who is permitted to receive annually without tax $1,000 plus that portion of the policy face value which is properly allocable to that year.

**The so-called "three per cent rule" has been eliminated by the 1954 Code and the tax free portion of each year's annuity payments is now calculated in effect by dividing the cost basis of the annuity policy by the remaining life expectancy of the annuitant at time of retirement or, in the case of a term policy, by the number of years in the term. This provides a much more equitable capital recovery pattern than was available under the old law.

In dealing with capital gains and losses, one of the more troublesome factors is determining the cost basis in the property disposed of. The only effective way to minimize this difficulty is to keep reasonably complete records of the original cost of the property, the amount, if any, of subsequent improvements or additions, and the amount of any allowable depreciation or other form of capital recovery. In addition, your records should show the time of acquisition and a notation of the serial number of the property if it is otherwise unidentifiable from other items of the same type which you own. If such records are not kept, you will be forced to guess at this information when you file your tax return for the year of sale. These estimates will be easy prey for an examining revenue agent and you will be hard-pressed to prove error in any changes which he proposes to make.

If you have acquired the property by gift, your basis is a substituted one that depends on the cost basis of the property in the hands of the person who gave the property to you. If the property is of any substantial value, it will behoove you to ask the donor his cost basis in the property. Perhaps you will be most commonly exposed to this problem in its reverse form—you will be the donor. In such case you can measurably increase the value of the gift by making available to the donee the information necessary to establish your cost basis in the property which then becomes his cost basis. Otherwise, the Commissioner of Internal Revenue is authorized to use his reasonable estimate in determining the donee's gain on disposition of the property and, as you can no doubt appreciate, the Commissioner's estimation will tend to favor the revenue.

Where property is acquired on the death of the owner, either under the provisions of his will or by virtue of the intestacy laws of the state, the fair market value of the property at the time of the owner's death becomes the basis for tax purposes of the recipient. This amount will generally be determined by the inventory value placed on the property in the Probate Court proceedings relating to the decedent's estate. However, if a federal estate tax return was filed and disclosed a higher value, or a higher value was placed on the property in determining the ultimate estate tax liability of the decedent, this amount becomes the basis.

5. *Joint tenancy, a boon or a boomerang.*†

At this point a comment or two seems appropriate on the value taxwise of the joint tenancy form of holding real or personal property.

As perhaps you have heard, from a property law point of view, the surviving joint tenant takes title to the whole of the property by virtue of the original conveyance of the property to the joint tenants. Title to the property is not considered to be acquired from the deceased joint tenant. However, this concept is not carried over into the estate tax branch of the law and valuable property interests are regarded as passing from the decedent on his death. As a result the fair market value of the property at the date of death is includible in hsi taxable estate except only to the extent that the consideration for the acquisition of the property was supplied by the surviving tenant. Therefore, where the deceased joint tenant supplied the total purchase price for the property, it is the same for estate tax purposes whether he held the property as sole owner at the time of his death or was a joint tenant. In either case, the full value of the property is includible for purposes of valuing his estate for estate tax purposes.

On the other hand, for income tax considerations, the property is viewed as being acquired by the surviving tenant in the fashion that the real property lawyer would look at it. As a consequence, the basis of the surviving tenant is the cost basis of the deceased tenant plus any consideration which the survivor may have supplied.

To illustrate this point, consider the husband and wife who purchased a home twenty years ago for a total price of $10,000. The husband supplied the entire consideration for the home and had it placed in joint tenancy in the hope of protecting his wife in the event of his death. On the husband's subsequent death, the wife, by virtue of the real property laws of the state, acquires full legal title to the house which is then, we will assume, worth $20,000. The full $20,000 is includible in the husband's estate for purposes of death taxes in the same manner as it would be had the husband owned it outright.

The disadvantage inherent in this type of property holding is immediately apparent when it is considered that the cost basis for income tax purposes to the surviving joint tenant in the example which we have used here is $10,000. Hence, if she is forced to sell the home she will have a $10,000 capital gain where the sale is made at the $20,000 fair market price that is used for state tax purposes. If the surviving widow has no other income, and this is generally not the case, and if she does not invest the proceeds of the sale of the house in a new residence, she will pay an income tax on the house sale of approximately $1,000. The amount of this tax will of course be measurably increased as the other income of the widow is multiplied.

The point to remember is that if the husband had held the property in his own name, the widow would have had no gain on its sale since her basis would be its fair market value. As a result she would have paid no income tax on account of its disposition. The tax that she does pay is the price exacted for using the joint tenancy method of title holding.

6. *Comments on business deductions.*

The mine run of business expenses present no problem at all. They are easily identifiable both by the taxpayer and by the examining revenue agent as being allowable deductions, and the amounts thereof are subject to fairly exact calculations. The important point to be remembered is that a failure to keep reasonably adequate and accurate records will render even the most standard expense item a difficult one to establish for the amount thereof will then be left to guesswork and the burden is on the taxpayer to establish the facts.

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†The following paragraphs are now of only historical concern since the 1954 Code removed the income tax inequity previously inherent in joint tenancy situations. The value at date of death rather than the decedent's cost basis is now prescribed as the surviving tenant's basis for gain or loss where a substituted basis in the property is called for.

In the practice of medicine, as in most other professional lines, the taxpayer encounters difficulty in only three or four areas of business deductions. For this reason we will confine our discussion of expenses to these items.

(a) Depreciation charges if fairly arrived at should no longer be subject to dispute by the Internal Revenue Service. The allowable depreciation charge on business furniture and equipment has long been recognized as a more or less annoying source of trivial adjustments in the tax liability of the practitioner. Depreciable life is at best an educated guess which has at times been imbued with a scientific aura by the application of mathematical formulae designed to divide the cost basis of the asset over its estimated life for recovery out of income. Most of you have undoubtedly had the experience of trying to establish that the life expectancy which you have assigned to a particular asset should be accepted instead of the longer life which the examining agent has like as not proposed. Usually the matter is resolved by your consenting to some small adjustment in your depreciation charge in order to wind up that year's tax liability. This you do even though convinced that your guess as to the life of the asset is much more realistic than that of the agent's—the cost of carrying the dispute further is usually more than the tax involved.

Since spring of 1953, this situation has been by and large eliminated. The Commissioner of Internal Revenue has directed his personnel to accept the taxpayer's depreciation figures unless they are obviously unreasonable. Therefore, if you are fairly realistic in your writeoff period and follow it consistently, you should have no trouble. If you assign a life of eight years to an asset which someone else might regard as having a life of nine or ten years, your calculations should be beyond change by the examining agent. On the other hand, if the life of the asset may reasonably be regarded as fifteen years you are treading on paper thin ice.*

(b) Automobile expenses should be no problem if you are realistic in your approach. The difficulties that may arise in connection with the deduction of automobile expenses can be minimized if your percentage assignment of the use of the automobile to your practice leaves some room for personal use where there is only one automobile in the family. If you have a two car family, then the allocation of 90 per cent or 100 per cent of your business car depreciation and operating costs as a deductible expense should meet with no substantial objection by the examining agent, unless your mileage indicates that you have been using the business car for vacation trips and other extended non-business purposes.

The use of charge accounts covering the operation and maintenance expense of your business automobile will eliminate the bulk of what can otherwise be a tedious record keeping procedure. It is well to have the family automobile serviced at a different garage or station if credit accounts are used. Otherwise you are running the risk of having your business expense records challenged as containing personal car expenditures.

(c) Business entertainment and promotion deductions require careful attention. The most prolific source of all administrative and judicial conflicts relating to the allowable business expenses of professional people is the entertainment and promotional expense category. Basically the problem is one of establishing that the expenditure which you claim is an ordinary and necessary one to the proper conduct of your practice. On occasion you will meet with an examining agent who claims that because it is unethical for a doctor to advertise, he is thereby precluded from any entertainment or promotional deduction. Fortunately, this is not the law, and there is no question that such a deduction is allowable if you can establish that the claimed expenditures were reasonably designed to advance or maintain your practice.

The crucial factor is that of proof and this will be complied with only if you maintain a fairly comprehensive log of the principal entertainment expenditures that you make, including the occasion, the persons entertained and the amount expended. The use of club memberships and courtesy accounts at the restaurants where you do the bulk of your entertaining will do much to minimize the record keeping burden. However, it is essential, particularly in view of the current attitude of the Internal Revenue Service toward this type of deduction, that you preserve with your club and restaurant expense statements a notation as to the identity of the groups entertained. Without this information it is very difficult to establish the requisite association between any given expenditure and your business; and without this association the deduction will not stand.

A good case in point is one involving a Dr. Richard A. Sutter, which was decided by the Tax Court of the United States on October 30, 1953. In that case, the doctor had claimed deductions for the following types of promotional and entertainment expenditures: (1) gifts to elevator operators, parking lot attendants, hospital employees and others; (2) the costs incurred on a hunting trip; (3) the gifts which he had made to medical associates; (4) the expenses incurred in connection with publishing an article dealing with his phase of the practice of medicine; (5) the cost of luncheons attended in connection with Chamber of Commerce and hospital association work; (6) expenditures incurred in business entertainment; and (7) the depreciation and cost of maintenance of a cabin cruiser used in conjunction with his business promotion activities.

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*The 1954 Code liberalizes depreciation charges by permitting accelerated rates to be applied to new business property acquired after December 31, 1953. Depreciation as to such property may now be computed by the sum of the digits method, the declining balance method utilizing a rate not more than twice the permissible straight line rate, or any other recognized procedure which writes off the asset during the first two-thirds of its life no faster than would be permitted under the declining balance method.

Of these seven categories of expenses, but two were allowed, and these only to the extent of 25 per cent of the amounts claimed. As to the first four classes of expenses, the Court affirmed disallowance on the ground that there was no adequate proof of any direct connection with the taxpayer's business. As to the cost of the luncheons which consisted almost entirely of the taxpayer's own meals, the Court concluded that there was no proof that the amount expended was in excess of what the taxpayer would have paid for his food if he had not attended these luncheons (the expenditure was thus reduced to a personal one which is expressly disallowed as a deduction by the taxing statutes).

Insofar as the entertainment expense deduction was concerned as well as that relating to the cabin cruiser, the Court arrived at the 25 per cent allowance figure on the theory that the rest of the expenses involved represented the amount reasonably allocable to the entertainment of a purely social nature and that which was designed to establish good will for the taxpayer's business.

The doctor in question was an industrial surgeon whose clinic serviced the needs of various industries and their insurance carriers. By the very nature of his practice the doctor was in a position to fairly easily establish the relationship between the entertainment of various employers and insurance representatives and the profitable practice of his specialty. His misfortune in having a substantial part of his claimed deductions disallowed is attributable largely to a failure of proof that you can avoid in your own case if you are willing to maintain the proper records to substantiate the deductions which you claim for this class of expense.

If the bulk of your practice or at least the significant part of it consists of referral cases, the cost of entertaining the doctors who actually refer patients to you or who are in a position to do so, should be fairly easily established as being closely related to the effective conduct of your practice and hence deductible to the full extent. The same would apply to entertaining patients where the relationship betwen the income produced and the outlay incurred is not unreasonable.

Unfortunately, there is and probably can be no hard and fast rule by which you can gauge the amount which will be ultimately allowed as a deduction for this entertainment and promotional type of expenditure. However, you can be certain that as the aggregate of your expenditures reaches substantial proportions when related to your gross revenue and the type of practice you engage in, the need for supporting records becomes more and more significant since the deduction will almost certainly be challenged by the examining agent.

7. *A comment or two on gift and estate taxes.*

Although the principal subject of this discussion has been income taxation, it seems appropriate by way of conclusion to comment briefly on the two other types of taxes which are likely to have a substantial impact on your affairs. The first is the gift tax, which is a levy on the right to gratuitously transfer property during life. The other is the death tax (either in the form of the federal estate tax or the state inheritance tax), which is applied to transfers of property at or by reason of death.

These two transfer taxes are so geared that the gift tax's rate is approximately three-quarters that of the death tax on any given value of property. In addition, both taxes are of the graduated type so that as the amount given increases the tax rate percentage climbs sharply. Because of these two factors it is possible in many cases to realize considerable savings in the total transfer tax burden by disposing of a portion of one's property to one's beneficiaries during life rather than retaining the whole estate so that it passes entirely at death.

Since 1948, when Congress authorized the joint return for income tax purposes, a so-called marital deduction is available for gift and estate tax purposes. The allowance of this deduction has opened up new vistas insofar as estate planning is concerned. The tax disadvantages formerly attendant on the transfer of a substantial portion of one's estate to his wife have been removed and the law now encourages the equalization of the estates of the husband and wife so that disproportionate tax burdens formerly occasioned by the husband's having the bulk of the family property can be avoided. To facilitate this, Congress has provided for the treatment of one-half of all qualified gifts to the donor's spouse as tax-free regardless of whether the gift is made during life or occurs at death. The only pre-requisite is that the relatively simple conditions surrounding the allowance of the marital deduction be complied with.

The satisfaction of these pre-requisites to the use of the marital deduction is one of the jobs to be fulfilled in the planning of your estate. How it will be handled in any particular case is of course subject to no set rule since it will depend upon a number of variables including the special objectives of the person involved, the size of his estate in relationship to that of his wife, and the number and age of his children. The significant point is that the tax savings occasioned by the use of the marital deduction can be assured if one but takes the time to plan his affairs.

It is not feasible here to list or discuss the various other factors which must be given due regard in planning the disposition of your property. It must suffice to say that they will probably include such fundamental procedures as the establishment of trusts to handle property which passes at death to minor children and the preparation and execution of appropriate buy and sell agreements covering your professional practice, where it is carried on in conjunction with others. The important thing both from the point of view of assuring peace of mind to you and your survivors as well as of assuring that no unnecessary gift or death taxes are

(Continued from page 773)

In Memoriam

WILLIAM AARON BESSESEN

Dr. William A. Bessesen, dean of the International College of Surgeons and widely known for many years as a Minneapolis surgeon, died at his home at 2222 Pillsbury Avenue, August 14, at the age of seventy-four years.

After six years of practice in Albert Lea and two years as assistant in surgery to Dr. William J. Mayo at Rochester, he came to Minneapolis nearly thirty-five years ago and remained in practice there. He had specialized in heart disease as well as in surgery and had also engaged in general practice. He had traveled widely to attend medical meetings in many parts of the world and had enjoyed an extensive acquaintance among physicians in many countries.

Dr. Bessesen was born in Freeborn county and attended school in Albert Lea. He received a B.A. degree from the University of Minnesota, and a master's degree in science from Northwestern University. He was graduated in medicine from Northwestern in 1905 and served his internship at St. John's Hospital in Fargo, with further postgraduate training at St. Mary's Hospital and at the Mayo Clinic in Rochester.

In 1915 Dr. Bessesen was married to the late Beatrice Gjertson, an opera singer, and both were prominent in social, art and musical circles in Minneapolis for many years.

Dr. Bessesen was a life member of the Hennepin County Medical Society, the Minnesota State Medical Association and the American Medical Association. He was a member of the staff of Eitel Hospital and had served as president of the Cosmopolitan Arts Club.

Survivors are, four sons, William Aaron, Washington, D. C.; Henry Adrian, Chicago; John Truman, Minneapolis, and Paul Stanford, Los Angeles, and two daughters, Mrs. Monica Bjornnes and Mrs. Delna Arnold, both of Minneapolis.

OTTO EMIL HUBBARD

Dr. O. E. Hubbard, well-known Brainerd physician, died suddenly at the home of his daughter, Mrs. Julius Twist, in Des Moines, Iowa, July 21, 1954. He was fifty-six years old.

Dr. Hubbard was born in Aurora, Illinois, and had lived in Brainerd for forty-five years. He attended high school there and was graduated in medicine from the University of Minnesota. He served in the Armed Forces during World War I and returned to Brainerd to practice medicine and surgery after graduation from medical school.

He was a member of the Upper Mississippi Medical Society, the Minnesota State Medical Association and the American Medical Association, also the Elks lodge and the Masons.

Survivors include his wife, Mrs. Nella K. Hubbard; one daughter, Mrs. Julius Twist of Des Moines; one brother, Fred Lyscio of Brainerd and, one grandson, Steven Twist, also of Des Moines.

WILLIAM HENRY HENNEY

Dr. William H. Henney died at the age of seventy-six at his home in McIntosh after thirty-five years in general practice there.

He had been health officer for McIntosh and nearby Winger, Erskine and Mentor for thirty years. He had also served as mayor of McIntosh for twenty-three years. When he retired from active practice because of ill health last spring, "Dr. Henney Appreciation Day" was proclaimed by the town and friends came from a wide area to honor the veteran physician.

Born in Cedar Rapids, Iowa, Dr. Henney attended high school in Redfield, South Dakota, and then taught school for a year before entering the University of Nebraska medical school. He succeeded in working his way through medical school, graduating in seven years. After that he practiced briefly in Thedford, Nebraska, and later in Beltrami county and at Fertile in Polk county before settling permanently in McIntosh. He was a member of the Red River Valley Medical Society, the Minnesota State Medical Association and the American Medical Association and of Our Saviour's Lutheran Church.

Surviving are his wife, Lydia; two sons, two daughters, three stepchildren, and twelve grandchildren.

WILLIS STORRS LEMON

Dr. Willis S. Lemon, former chief of the chest department of the Mayo Clinic, died August 19, 1954, of a heart ailment in Roanoke, Virginia. He was seventy-six years old and had retired from the Clinic in 1947.

Dr. Lemon was born at Villa Nova, Ontario, and was graduated from Aylmer Collegiate Institute at Aylmer, Ontario in 1896. He received his medical degree from the University of Toronto Medical Faculty and served his internship at the Toronto General Hospital with three years' postgraduate work at the Ontario Sanitorium for Tuberculosis. He practiced for eight years at LaGrange, Illinois, before joining the Mayo Clinic in 1917.

Surviving are his wife, Ethel Haines Lemon; a son, Dr. Willis E. Lemon of White Sulphur Springs, West Virginia; a daughter, Mrs. Katherine Lord of Hanover, New Hampshire; and two brothers, Dr. W. M. Lemon, LaGrange, and Dr. A. Ray Lemon of Aylmer, Ontario.

◆ Reports and Announcements ◆

MEDICAL MEETINGS

State

Physicians and Schools, First conference, University of Minnesota Center for Continuation Study, Minneapolis, December 2-3, 1954.

Sectional

Minnesota State Medical Association, Annual Meeting, Minneapolis, May 23, 24, 25, 1955.

American College of Surgeons, sectional meeting, The Fort Harry, Winnipeg, Manitoba, Canada, April 25-26, 1955.

National

American College of Chest Physicians, Seventh annual postgraduate course, Hotel New Yorker, New York City, November 8-12, 1954.

American College of Surgeons, Fourth clinical session, Atlantic City, New Jersey, November 15-19, 1954.

American Medical Association, Clinical Session, Miami, Florida, Nov. 29-Dec. 2, 1954.

American Medical Association, Annual meeting, Atlantic City, New Jersey, June 6-10, 1955.

National Society for Crippled Children and Adults, Statler Hotel, Boston, Massachusetts, November 2-5, 1954.

PR INSTITUTE

The AMA's Public Relations Institute in Chicago, September 1 and 2, attracted almost 300 state and county medical society representatives. Among those attending the "crackerbarrel Institute" from Minnesota were Harold W. Brunn, Assistant Executive Secretary, and Joyce Pearson, Director of Public Relations, both from Minnesota State Medical Association, and Naomi M. Peterson, Assistant Professor, School of Business Administration, University of Minnesota, Minneapolis.

The Institute, planned primarily for lay executive and PR personnel, M.D. chairmen of PR committees, and Auxiliary PR committee women, was the most successful ever held. The two-day meeting featured experts in medical television production, direct mail promotion, AMA services, medical fees, the role of medical assistants, medical motion pictures, and inter-organizational co-operation.

Because of the large and enthusiastic attendance, almost every problem confronting medical societies was discussed, and the meeting provided many suggestions and approaches to a successful and thorough medical public relations program for each society.

This co-operative spirit at the Institute displayed the united front of nationwide medical public relations, and the reported "success stories" were convincing proof that the programs of medical societies were winning the increased understanding and friendship of the public.

Another meeting, keyed to the public relations needs of individual physicians, will be the AMA's Seventh National Medical Public Relations Conference in Miami at the McAllister Hotel, Sunday, November 28—the day preceding the opening of the Clinical Session. All physicians are invited to participate and to learn how their colleagues have improved medical public relations in their home communities.

DEGREES NOW OFFERED IN MEDICAL JOURNALISM

According to an announcement from the American Medical Writers' Association, the first four-year collegiate courses in medical journalism and writing leading to a bachelor's degree will be inaugurated next September at the University of Illinois, Urbana, and the University of Missouri, Columbia. Several partial scholarships ($500 each), sponsored by the American Medical Writers' Association, will be available. Dr. Harold Swanberg, Quincy, Illinois, secretary of the Association, has initiated the fund with a $1,000 contribution to establish one scholarship at each of the schools. Checks for the fund should be made payable to the Association and marked for the Medical Journalism Scholarship fund. Descriptive literature concerning the academic courses may be obtained from Earl F. English, Ph.D., dean, School of Journalism, University of Missouri, Columbia, and I. W. Cole, School of Journalism and Communications, University of Illinois, Urbana. (JAMA, 155:499, May 29, 1954.)

VAN METER PRIZE AWARD

The American Goiter Association again offers the Van Meter Prize Award of $300 and two honorable mentions for the best essays submitted concerning original work on problems related to the thyroid gland. The award will be made at the annual meeting of the Association which will be held in Oklahoma City, Oklahoma, April 28, 29 and 30, 1955, providing essays of sufficient merit are presented in competition.

The competing essays may cover either clinical or research investigations; should not exceed three thousand words in length; must be presented in English, and a typewritten double space copy in duplicate sent to the Secretary, John C. McClintock, M. D., 149½ Washington Avenue, Albany, New York, not later than January 15, 1955.

AMERICAN BOARD OF OBSTETRICS AND GYNECOLOGY

The next scheduled examination of the American Board of Obstetrics and Gynecology (Part I), written examination and review of case histories, for all candidates will be held in various cities of the United States, Canada, and military centers outside the continental United States, on Friday, February 4, 1955. For information, candidates may contact Robert L. Faulkner, M. D., secretary, 2105 Adelbert Road, Cleveland 6, Ohio.

NATIONAL SOCIETY FOR CRIPPLED CHILDREN

Keynoting the 31st convention of the National Society for Crippled Children and Adults will be a speech by John F. Kennedy, junior U. S. Senator from Massachusetts, on the topic "Rehabilitation, Your Job and Mine." The convention will be held November 2-5 at the Statler Hotel in Boston, Massachusetts.

The convention theme, "Rehabilitation for Independence," will feature objectives of the Easter Seal Society campaign. Presiding will be National Society president J. Raymond Tiffany. A. L. M. Wiggins, Atlantic Coast Line Railroad chairman of the board, will talk to Easter Seal volunteers at a special luncheon.

Featured on the program will be: Mrs. Ivy Baker Priest, treasurer of the United States; Margaret Mead, Ph.D., noted anthropologist; Romaine Mackie, Ph.D., U. S. Office of Education, chief of the section on exceptional children and youth; Dr. Howard A. Rusk, medical director of the Institute of Physical Medicine and Rehabilitation, New York University—Bellevue Medical Center; Dr. William C. Menninger, general secretary of the Menninger Foundation; Basil O'Connor, president of the National Foundation for Infantile Paralysis; Dr. Augustus Thorndike, president of the Bay State Medical Rehabilitation Clinic; and, Dr. William T. Green, orthopedic surgeon and chairman of the Joint Committee on the Public Care of Crippled Children, Boston.

SECTIONAL MEETINGS—ACS

Winnipeg, Manitoba, will be the site for the Sectional meeting of the American College of Surgeons, April 25 and 26, 1955. Dr. P. H. T. Thorlakson and committee have announced that the program will include panels, papers and symposia dealing with gastric surgery, cardiac surgery, plasma substitutes, ureteral transplants, surgical lesions of the pancreas, biliary tract surgery, surgical diseases of the spleen, trauma and cancer.

Winnipeg speakers, as well as the following out-of-town authorities, will be featured: Earl A. Connolly, M. D., Omaha, Nebraska; Richard L. Varco and W. Robert Schmidt, Minneapolis; J. C. Evans, Rochester; Stanley O. Hoerr, Cleveland, Ohio; Thomas S. Wilson and Walter MacKenzie, Edmonton, Canada; R. A. Mustard, Toronto, Canada; and, Clayton H. Crosby, Regina, Canada.

For further information, physicians may contact Dr. H. Prather Saunders, Associate Director, American College of Surgeons, 40 East Erie Street, Chicago 11, Illinois.

CHEST RADIOGRAPHS AVAILABLE FROM HEALTH DEPARTMENT FILES

Dr. L. G. Rigler has recently pointed out that chest x-ray pictures taken even years before ultimate diagnosis of lung cancer in a patient may frequently show suggestive shadows when studied in retrospect. Many doctors have emphasized the value of comparison of previous chest radiographs in deciding on the significance of more recently discovered shadows.

The Minnesota Department of Health has available in its Tuberculosis Control Unit, approximately 1,400,000 chest x-ray pictures of Minnesota residents who had examinations made by the three mobile x-ray units operated by the Department from 1947 through 1953. These people were examined in surveys carried on in sixty-seven counties in all parts of the state, not including Hennepin or St. Louis Counties. In these two counties, mobile units operated by the respective county Christmas Seal organizations x-rayed many additional thousands of residents.

The registration cards which identify the x-ray films available from the Department have been arranged by county of survey and alphabetically by first letter of the name. The films are 70 millimeter photofluorograms in rolls of 350 pictures. Each satisfactory film bears a survey and x-ray number which is duplicated on the registration card, where additional data as to name, age, sex, address, date of film, and name of personal physician are recorded. Persons of school age and up were routinely included in the surveys. The great majority of the films were interpreted as negative. For any persons whose 70 millimeter films suggested pulmonary shadows, 14x17 inch chest x-rays were routinely done by the mobile unit; these larger radiographs customarily were returned to the county at the end of the survey for local disposition.

The Department is glad to make these radiographs available to physicians for their use in studying chest conditions in their patients. The figures suggest that over half of the state's population were x-rayed by the units, and that a radiograph might be available on one of every two persons, other than very young children, who might consult a doctor.

When requesting radiographs, as much information as possible should be sent as to name, age, sex, and place and date of the x-ray, if known. The Department will do its best to locate the radiograph and send it to the doctor.

PHYSICIANS-SCHOOLS MEETING SET

Plans for better school health programs for Minnesota will be discussed by physicians, dentists and school administrators at the first conference on Physicians and Schools, to be held December 2 and 3, 1954. The meeting, a follow-up of the American Medical Association's national conference on Physicians and Schools, will be held at the University of Minnesota Center for Continuation Study, Minneapolis.

The program will be sponsored by the Minnesota State Medical and Dental Associations, the School Administrators' Association and the Minnesota State Departments of Health and Education.

All physicians have been urged to attend the sessions. Tuition will be paid by the Department of Health.

STATE GP ACADEMY

The fourth annual scientific assembly of the Minnesota Academy of General Practice will be held in Rochester, October 20, with about 500 physicians expected to attend.

Thirteen Mayo Clinic staff men will present scientific lectures, and thirty-five scientific exhibits will be displayed. The meeting will be held in Mayo Civic auditorium.

Guest speaker at the noon luncheon meeting will be Dr. Albert M. Snell, former head of a section of internal medicine at the Mayo Clinic, who now is practicing at the Palo Alto, California, clinic. Dr. C. W. Mayo will be master of ceremonies.

Members of the clinic staff who will present scientific papers during the day are Drs. L. A. Brunsting, W. F. Kvale, T. T. Myers, E. A. Banner, P. R. Lipscomb, M. B. Coventry, C. H. Watkins, W. H. ReMine, O. H. Beahrs, J. C. Ivins, L. E. Ward, W. O. Osborne and J. L. Emmett.

General chairman of the meeting is Dr. R. L. Page of St. Charles. Dr. H. A. Wente of Rochester, who is secretary-treasurer of the Minnesota Academy of General Practice, is in charge of local arrangements. President of MAGP is Dr. James A. Cosgriff of Olivia.

MINNESOTA NURSING GROUP

Minnesota League for Nursing announces two regional workshops for instructors in the Twin Cities area. The first at the American Red Cross headquarters, Minneapolis, will be on November 29 and 30; the second at St. John's Hospital, Saint Paul, will be on December 2 and 3. If these are successful, others will be held in other areas of the state. A five-day teacher-training workshop was held November 8-12 with ten teachers from the Twin Cities hospitals in attendance.

NORTHERN MINNESOTA MEDICAL ASSOCIATION

A Bertha physician, Dr. W. W. Will, was elected president of the Northern Minnesota Medical Association at its thirty-fourth annual meeting last month. Other officers include: Dr. Leslie Lundsten of Bemidji, vice-president, and Dr. C. L. Oppegaard of Crookston, secretary-treasurer

Arrangements for the two-day meeting in Willmar were made by a committee headed by Dr. D. L. Jacobs, convention chairman.

Guest speaker at the convention banquet was Dr. Paul de Kruif of Holland, Michigan, noted scientist-author. Dr. de Kruif talked on hypertension.

Dr. L. H. Rutledge, Detroit Lakes, is retiring president.

SOUTHERN MINNESOTA MEDICAL ASSOCIATION

Highlighting the program of the Southern Minnesota Medical Association's meeting last month at Winona was a speech by Dr. W. C. Bernstein of St. Paul. Dr. Bernstein, retiring president and a member of the association for 25 years, outlined the group's 62 years of medical achievement in a talk at the convention banquet.

Officers elected for 1955 are: Dr. David Anderson, Austin, president; Dr. Walter J. Halloran, Jackson, first vice-president; Dr. Charles Stroebel, Rochester, second vice-president; and, Dr. G. R. Dressner, Rochester, secretary-treasurer.

Medals were awarded Dr. W. O. Finkelnburg, Winona, and Dr. J. D. Sjoding, Mankato, for submitting the best case report and scientific paper, respectively.

Working with Dr. Lewis I. Younger on the arrangements committee were: Dr. Philip Heise, Dr. George Loomis and Dr. Roger Hartwich, all of Winona.

MENTAL HEALTH MEETING HELD IN ROCHESTER

More than 250 legislators, physicians, welfare workers, mental hospital committee members, judges, county commissioners and other representatives of organizations interested in the state mental health program attended a special meeting last month at Rochester State Hospital.

The meeting, which stressed the importance of medical and surgical services and research, was the first in a series of five such regional programs planned to acquaint Minnesotans with the progress and problems in the mental health program. A new research project in Huntington's chorea was also discussed.

Dr. Magnus C. Petersen, superintendent, presided. Speakers included Drs. J. C. Ivins, H. W. Dodge, Jr., R. Royer and O. H. Beahrs of the Mayo Clinic, and Drs. Jorge A. Lazarte and A. H. Sanford and J. S. Pearson, Ph.D., of the state hospital staff: Jarle Leirfallom, commissioner of the department of public welfare, also spoke. The meeting was sponsored jointly by the welfare department and the various state hospitals.

CITY DIABETES SURVEY

Minneapolis, first city to use the Dreypak paper slip method in a citywide diabetes detection survey, will have its fourth mass diabetes check in five years.

The detection drive will be conducted during National Diabetes week—November 14-20.

Dr. Moses Barron is chairman of the Diabetes Association of Greater Minneapolis.

The Dreypak method, which does away with cumbersome bottles or urine specimens, will be even more simple this year than it was in 1952 when Minneapolis used it in a citywide survey.

Agencies co-operating in this year's survey include the Diabetes Association of Greater Minneapolis, Minneapolis health department, Hennepin County Medical society, Minneapolis Jaycees, Hennepin County Tuberculosis association and Twin City Retail Druggists association.

PSYCHOLOGY COURSE SET

Two psychologists from North Carolina and California will return to their alma mater in October to be guest speakers at a University of Minnesota psychology short course in the University's Center for Continuation Study.

The psychologists are George Welsh, associate professor of clinical psychology at the University of North Carolina, Chapel Hill, and Harrison G. Gough, assistant professor of psychology at the University of California at Berkeley. Both studied clinical psychology at the University of Minnesota.

The course, on the use of the Minnesota Multiphasic Personality Inventory, is planned for Thursday through Saturday, October 14-16, under the direction of Starke R. Hathaway, director of the University's division of clinical psychology and co-deviser of the MMPI. The course is open to psychiatrists and other physicians.

CONTINUATION COURSES

Fractures will be the subject of a continuation course to be presented by the University of Minnesota next November 22 to 24 at the Center for Continuation Study. Intended primarily for physicians engaged in general practice, the program will stress the practical management of the types of fractures most commonly met. Registrants will be invited to bring their own films to a "consultation session." The program will be presented under the direction of Dr. Wallace H. Cole, Professor, Department of Surgery, and Director, Division of Orthopedic Surgery.

NINE HEALTH FACILITIES TO GET HILL-BURTON FUNDS

An estimated $3,415,540 for nine health facilities to be built under the Hill-Burton Hospital Construction Act, was approved by the State Board of Health at its meeting Friday, October 8. Of this amount, $1,464,165 was committed from funds immediately available under Hill-Burton funds. An additional $200,000 was provisionally allotted to three of these projects from money provided under the amendments to the Act, Public Law 482, passed by the 83rd Congress. The amendments provide for the construction of specialized and related hospital facilities, including chronic hospitals, diagnostic or treatment centers, rehabilitation facilities and nursing homes.

Total cost of the nine facilities is estimated to be $8,350,394.

Two of the projects are for Minneapolis facilities, including the Minneapolis Public Health Center, for which the estimated federal portion will be $970,202 from Hill-Burton and amendment money to be split over a three-year period. The other project is for a combined chronic and psychiatric unit to be added to Fairview Hospital, totalling $423,675, split over a two-year period.

This is the first time that Hill-Burton money has been used to assist in the construction of any Minneapolis project, with the exception of three projects on the University of Minnesota campus, according to Dr. R. N. Barr, deputy officer of health, and Dr. Helen Knudsen, chief of the section of hospital services.

Other projects include a Public Health Center for St. Paul, for which the total federal share is estimated to be $405,891, from Hill-Burton and amendment money spread over the next two or three years.

Hospitals for four communities in the general hospital category "Priority A" group were approved. These include Long Prairie, $204,545; Paynesville, $163,620; Cambridge, $180,000; Two Harbors, $270,000.

Included in the allotment is $346,000 of Hill-Burton money for a regional hospital in Fergus Falls, the remainder of a split project from last year. It is estimated that the total cost of this hospital will be slightly more than one million dollars, with the total federal share $450,405.

A second regional hospital to be located at Willmar was approved by the Board. The total cost of the Willmar Hospital is an estimated $771,560, with the federal share of $347,202. Only $150,000 will be available this year on the split-project basis. Sponsoring agents of split projects must underwrite the federal portion, which is not currently available.

The review of projects under construction or scheduled over the next three-year period in Minnesota indicates that total construction for health facilities will exceed 65 million dollars.

The projects authorized by the State Board of Health had been previously recommended by the State Advisory Council on Hospital Construction. Amounts received from federal funds represent 45 per cent of the total cost of construction and equipment. The cost of the building site for public health centers is eligible for federal participation.

At the present time, amendment money for assistance in the construction of the specialized facilities is not available. As in the original Hill-Burton plan, states must survey needs and develop a state plan before funds can be specifically appropriated for chronic disease hospitals, diagnostic or treatment centers, rehabilitation facilities and nursing homes, according to Dr. Knudsen. It is expected that this preliminary work will be completed sometime after January 1, 1955.

THE PHYSICIAN AND HIS TAXES

(Continued from page 768)

incurred, is the virtually absolute necessity for integrating your program of property disposition and future protection for your family into what is known as an estate plan.

This estate plan may be relatively simple or it may be fairly detailed and complex depending on the size of your property holdings and the number and station in life of those whom you intend to receive your property. But whether simple or complex, the plan cannot have life unless you take time to create it and then assure its completion by executing a Will that is tailor-made to your particular needs.

The meeting was adjourned.

Robert E. Priest, m.d.,
Secretary

Woman's Auxiliary

RAMSEY COUNTY WOMEN ACTIVE

The Philanthropic Fund of the Woman's Auxiliary to the Ramsey County Medical Society benefited substantially from the annual Fall Fashion Festival which highlighted early fall activities of the Ramsey group. The festival was presented by Schuneman's and held at the Hotel Saint Paul. Mrs. M. Dudley Hilker of Saint Paul was general chairman for the affair.

All activities of the auxiliary for the 1954-1955 season were discussed and plans outlined at a Board meeting held August 23 at the home of Mrs. W. P. Gardner, president. Committee chairmen were briefed for the year at this meeting.

A large representation from the Ramsey Auxiliary attended the annual School of Instruction of the Woman's Auxiliary to the Minnesota State Medical Association also. This program of instruction for all auxiliary members was held October 1 at the Hotel Curtis in Minneapolis with Mrs. George Turner of El Paso, Texas, president of the Woman's Auxiliary to the American Medical Association, as one of the principal speakers.

STATE AUXILIARY ANNUAL MEETING

Doctors' wives from all over Minnesota attended the 32nd Annual Meeting of the Woman's Auxiliary to the Minnesota State Medical Association which which opened with the usual pre-convention Board meeting and luncheon, Monday, June 7, at the Kitchi Gammi Club. Tea for all visiting women followed in the afternoon at the Tweed Art Galleries, courtesy of the University of Minnesota Art Department, Duluth Branch, with the St. Louis County Auxiliary as host.

Among important actions taken at the annual meeting and luncheon, Tuesday, June 8, at the Hotel Duluth, were the following:

A gift of $100 was voted for the American Medical Education Foundation.

Purchase of $700 worth of films on health was voted. The films will be presented to the Minnesota Department of Health for distribution.

Mrs. Ernest M. Hammes, Sr., of St. Paul reported the purchase of two conference tables and a bronze plaque, all to be inscribed as gifts of the Woman's Auxiliary and all to be placed in the William A. O'Brien Memorial Seminar Room in the Mayo Memorial Medical Center at the University of Minnesota.

The program included a talk by Mrs. C. N. Pearson, Baraboo, Wisconsin, president of the Woman's Auxiliary to the State Medical Society of Wisconsin, on "Nurse Recruitment" and an address by Dr. Edward B. Shaw of San Francisco on "Community Responsibility for the Immunization Program." An impressive In Memoriam Service for deceased members was conducted by Mrs. John S. Milton, Minneapolis.

A rising vote of thanks to Mrs. Henry W. Quist of Minneapolis, retiring president, accompanied installation of new officers for 1954-55. They are:

President—Mrs. Peter S. Rudie, Duluth
President-elect—Mrs. H. H. Fesler, Saint Paul
First Vice President—Mrs. John Dordal, Sacred Heart
Second Vice President—Mrs. Virgil J. Schwartz, Minneapolis
Third Vice President—Mrs. L. P. Howell, Rochester
Fourth Vice President—Mrs. Justus Ohage, Saint Paul
Recording Secretary—Mrs. David J. Halpern, Brewster
Corresponding Secretary—Mrs. S. N. Litman, Duluth
Treasurer—Mrs. Charles W. Froats, Saint Paul
Auditor—Mrs. G. H. Goehrs, Saint Cloud

The luncheon program was featured, also, by a style show presented by Freimuth Department Store of Duluth.

A Round-up Breakfast, at which all visitors were guests again of the St. Louis County Auxiliary, concluded the program Wednesday at the Duluth Women's Club. All doctors' wives who attended gained valuable information to take back to their local auxiliaries and all were grateful to the committee and hosts who helped to make it a successful meeting.

Mrs. David J. Halpern
Recording Secretary

Communication

Arthur H. Wells, M. D.
Editor, Minnesota Medicine
915 East 1st Street
Duluth, Minnesota

Dear Dr. Wells:

I thought your Journal would be interested to learn that the Hamm Memorial Psychiatric Clinic was officially opened on July 1 of this year. As you may recall, the Clinic was established upon the recommendation of a citizen's committee appointed by the St. Paul Area of Public Health Council. The Clinic is financed by the Hamm Foundation.

Our initial professional staff has been appointed and consists of a director, two psychiatric social workers and a clinical psychologist, as follows:

Mrs. Phillippa H. Eggleston, B.A., M.S., is chief psychatric social worker and Rosabelle Snahr, B.A., M.S., serves as psychiatric social worker. Murray S. Stopol, M.A., Ph.D., is clinical psychologist for the clinic.

Thanking you for your past co-operation, I am

Sincerely yours,
Clarence J. Rowe, M. D.

Saint Paul, Minn.
August 25, 1954

Of General Interest

Six Minneapolis heart specialists, members of the Minnesota Heart Association board of directors, took part in the Second World Congress of Cardiology and the 27th scientific sessions of the American Heart Association last month in Washington. They were **Ancel Keys, Ph.D.**, and **Drs. John J. Boehrer, Maurice B. Visscher, Lewis Thomas, Richard L. Varco** and **Henry L. Taylor**, all of Minneapolis.

* * *

On hand for the 100-mile stock car races during the State Fair was **Dr. C. E. Sutton**, ninety-three, of Minneapolis, the man who won the first auto race staged on the Minnesota Fair track. Dr. Sutton, who served as official starter at this year's event, won the 1908 race in a Locomobile streamer. In 1910 he was starter at the first races to be held in conjunction with the Fair. Dr. Sutton has practiced medicine for sixty-five years.

* * *

Dr. M. D. Starekow of Thief River Falls has been appointed examining surgeon for the Winnipeg division of the Soo Line, it has been announced. Dr. Starekow succeeds Dr. Edward Bratrud. **Dr. Dan Greene** was named Thief River Falls surgeon for the Soo Line, succeeding the late Dr. O. F. Mellby and Dr. Theodor Bratrud, now of Minneapolis. Both appointments were made by **Dr. Harvey Nelson** of Minneapolis, chief surgeon for the railroad.

* * *

Off on a two-month business and pleasure tour of Europe, Africa and the Near East is **Dr. Marie K. Bepko** of Cloquet. Dr. Bepko will attend the Seventh Congress of the Medical Women's Internation Association at Gardone, Lake Garda, Italy, and participate in an international tour arranged from members including stops in Portugal, Algieria, Spain and Italy. In Rome, Dr. Bepko will attend the Assembly of the World Medical Association. Before returning to Cloquet, she will visit Lebanon, Syria, Israel and Jordan.

* * *

Dr. and Mrs. Arthur M. Olsen of Rochester are touring the Scandinavian countries on the first leg of a two-month European trip. The Olsens will also visit France, Switzerland, Italy, Spain and Portugal. A member of the Mayo Clinic staff, Dr. Olsen will present papers at the Third International Congress of Diseases of the Chest in Barcelona, Spain, and at the Third International Congress of the Broncho-Esophagological Society in Lisbon, Portugal. The couple will return to Rochester the latter part of October.

* * *

Doctors and wives of the Range Medical Society and Auxiliary were entertained for their annual summer meeting at the home of **Dr. W. C. Heiam** of Cook..

Dr. Gerald S. Buchanan of the Deer River Hospital Clinic Staff has received notice to report for duty with the Army Medical corps at Fort Sam Houston, Texas. Dr. Buchanan has been given the rank of Captain.

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Residing in Rochester are **Dr. and Mrs. Victor Hugo Gardner**, who were married recently in Fairmont. The bride is the former Dolores Randall, daughter of Dr. and Mrs. Selby Wetmore, St. John's, New Brunswick, Canada. Dr. Gardner, son of Dr. and Mrs. V. H. Gardner of Fairmont, is a fellow in orthopedic surgery at the Mayo Clinic.

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Speaker at a dinner meeting of the Sister Elizabeth Kenny Foundation fund appeal volunteers was **Dr. Paul M. Ellwood,** director of in-patient service at Kenny Institute, Minneapolis. The meeting was part of an all-day rally of Wisconsin, Iowa and Minnesota county fund appeal chairmen held last month at the Nicollet hotel.

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Guest speaker September 21 at a meeting of the Blackhawk County Medical Society, Waterloo, Iowa, was **Dr. Hamlin Mattson** of Minneapolis. Dr. Mattson spoke on the topic "Surgery of the Peptic Ulcer."

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Dr. William R. Tucker, chief of the tuberculosis service at Veterans Hospital for seven years, left the Minneapolis post to become chief of the pulmonary disease service of the Veterans Hospital at Durham, North Carolina. Dr. Tucker assumed his new duties last month. He is also a member of the Duke University School of Medicine faculty at Durham. Dr. Tucker is succeeded in Minneapolis by **Dr. William W. Stead.**

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Visiting an old friend in Minneapolis, **Dr. John F. Briggs,** last month was one of England's leading authorities on heart disease, **Dr. Geoffrey Bourne.** Dr. Bourne, who is director of cardiology at St. Bartholomew's Hospital and Medical School of London, is a specialist in hypertension. During his stay here he spoke at a staff meeting at St. Joseph's Hospital and visited Variety Club Heart Hospital and the Ancker Research Center.

* * *

Guest speaker for a five-county Christmas Seal dinner last month at Morris was **Dr. Kathleen Jordan** of Riverside Sanatorium, Granite Falls. Dr. Jordan is field physician for the state Christmas Seal organization. Counties participating in the workshop and dinner meeting were Pope, Big Stone, Traverse, Stevens and Swift.

Dr. Charles W. Mayo of Rochester was honored last month when he was one of four men given honorary fellowships by the American College of Hospital Administrators. The group met in Chicago.

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Benefits polio victims have received from Sister Elizabeth Kenny treatments were reviewed by **Dr. Miland E. Knapp**, Minneapolis, at the September meeting of the American Congress of Physical Medicine. Dr. Knapp, chief of physical medicine at the Kenny Institute, praised the Kenny method of treatment in a paper prepared for delivery at the meeting in Washington, D. C.

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Dr. Robert Priest has been named new president of the Northwestern Hospital medical staff. **Dr. Richard Horns** will be vice president with **Dr. Richard Reiley** as secretary-treasurer. Past president of the group is **Dr. R. E. Hultkrans.**

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Second prize winner in the Foundation of the American Society of Plastic and Reconstructive Surgery scholarship contest was **Dr. A. M. Struthers** of Rochester. Dr. Charles Horton of Duke University, Durham, North Carolina took first place. The winning reports were presented at the society's October meeting in Hollywood, Florida.

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In recognition of his profession contribution to the community, **Dr. E. J. Huenekens**, Minneapolis pediatrician, was awarded the St. Barnabas Bowl in a ceremony at the Cathedral of St. Mark. The silver bowl is an annual award by the board of trustees of St. Barnabas Hospital to a doctor selected by the Hennepin County Medical Society. Previous winners of the bowl were Dr. S. Marx White in 1952 and Dr. Henry L. Ulrich, 1953. Dr. Huenekens also received the 1954 distinguished service certificate of the Community Chest and Council.

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Dr. James A. Johnson of Minneapolis was re-elected president of the Hennepin county district, Minnesota division, American Cancer Society, at a recent meeting of the group. Dr. Johnson has served in this position since 1944.

* * *

Dr. Lawrence M. Randall, chairman of the Mayo Clinic sections on obstetrics and gynecology, has been named president-elect of the American Association of Obstetricians and Gynecologists. Dr. Randall, a Mayo Foundation professor, joined the Clinic in 1924. His election took place at the association's annual meeting at Hot Springs, Virginia.

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A total of $405,456 has been donated to the 1954 American Cancer Society fund drive by Minnesotans, **Dr. David P. Anderson**, Austin, state president, has announced. Goal for the drive was $354,000.

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On a six-week trip to Europe are **Dr. and Mrs. T. B. Magath** of Rochester. In Madrid, Spain, Dr. Magath, chairman of the sections of clinical pathology and biochemistry at the Mayo Clinic, will deliver a paper at the International Congress of Hydatid Disease. The Magaths will tour Spain and visit France, Portugal and England before returning home.

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Guest speaker at the first fall meeting of the Scott County (Iowa) Medical association at Davenport, Iowa was **Dr. Tague C. Chisholm**, Minneapolis. "Emergency Surgery in Infants" was his speech topic.

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Three Pipestone physicians, **Dr. R. W. Keyes, Dr. R. J. Kotval** and **Dr. W. G. Benjamin**, took part in the first of a series of medical seminars held last month in Slayton. Other seminars followed the initial meeting, which was sponsored by the University of Minnesota and the Minnesota State Medical Association.

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The following physicians have recently completed Fellowships or terminated their periods of assignment at Mayo Clinic, Rochester: **R. M. Akey, J. J. Antel, J. A. Benson, Jr., J. T. Brandenburg, H. D. Breidahl, L. T. Brown, T. A. Burcham, Jr., R. J. Burleson, Edward Cutler, R. A. Dobson, P. L. Eichman, Luis Fernandez-Herlihy, C. C. Henderson, William Hunt, A. L. Karavitis, Andrew Helen, R. G. Khayat, K. S. Kim, R. H. Livingstone, Mary J. MacDonald, K. L. Morris, S. S. Morrison, L. S. O'Holleran, J. E. Osborne, M. B. Oxford, E. W. Parry, M. A. Peters, R. A. Rabens, M. G. Ringer, Jr., H. M. Roberts, A. G. Rogers, J. T. Shepherd, L. R. Smith, H. J. C. Swan, J. C. Tarantino, T. M. Terpinas, Richard Thors, F. A. Ubel, Jr., Geoffrey Watkinson, J. A. Witt, W. W. Wood** and **Eileen E. Zeitler.**

Going on military leaves of absence are **Drs. J. D. Bonnet, N. L. Cadman, A. R. W. Climie, A, B. Gould, Jr., R. E. McCaslin, Jr., R. A. Peake, G. J. Petropoulos, H. J. Schultz, H. J. Semler and A. W. Silver.**

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Dr. L. J. Swanson, St. Paul, has returned from a two-year tour with the U.S. Air Force, and will open his civilian practice at 950 South Robert Street.

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Dr. W. P. Eder, Minneapolis, has announced the opening of new offices at 1629 Medical Arts Building, Minneapolis. He will specialize in the practice of surgery. Dr. Eder has been surgical resident at General Hospital the past two years.

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Dr. C. J. Henry and **Dr. M. W. Johnson** of Milaca will open offices in St. Cloud, it has been announced. The physicians will locate in the Foley Hardware Building.

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New operative techniques in obstetrics and gynecology were reviewed when **Dr. A. O. Swenson**, Duluth, president-elect of the Minnesota State Medical Society, spoke to members of the Southwestern

Medical Society at Worthington. The meeting was one of three to be held in Worthington, with the annual meeting set for Pipestone.

* * *

Dr. John F. Pohl, Minneapolis orthopedic surgeon at the Micheal Dowling School of Crippled Children, spoke at a regular meeting of United Cerebral Palsy of Duluth. His topic was "Orthopedic Management of Cerebral Palsy."

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Dr. John J. Beer, chairman of the Medical Advisory Committee for the St. Paul Rehabilitation Center, which is appointed by the Ramsey County Medical Society, announces the appointment of the following supervisory staff at the Rehabilitation Center: Donna Pauley, R.P.T.; Mrs. Mary Alice Frawley, O.T.R.; and Eleanor Swanson, M.A., Speech Therapy.

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Dr. J. S. Blumenthal, clinical associate professor of medical and chief, Allergy Clinic of the University of Minnesota, addressed the Glen Lake staff recently on the subject "Skin Tests in Allergy—Their Values and Limitations." He also spoke on "Allergy and the Need of Research in this Field" at the Oak Park Home auxiliary, Minneapolis.

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Dr. Waltman Walters, Rochester surgeon, has been honored as recipient of the 1954 Honor Award given by the Mississippi Valley Medical Society. Dr. Walters is professor of surgery, Mayo Foundation, University of Minnesota, and chief editor, AMA Archives of Surgery and the Lewis-Walters practice of surgery. The Honor Award is given from time to time to non-members of the Society "who have made distinguished contributions to clinical medicine," the Society announced.

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Dr. J. Arthur Myers, chief of the University of Minnesota Chest Clinic, was speaker at the annual McLeod County public health dinner held in Winsted. His topic was "Hunting the Tubercle Bacillus." Dr. Myers also presented Tuberculosis accreditation awards to county commissioners and to school superintendents in McLeod county.

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Dr. Viktor O. Wilson of Rochester, Olmsted county public health officer, and **Dr. Ralph Paffenbarger,** epidermiologist with the poliomyelitis laboratory of Johns Hopkins University, Baltimore, are working together on a new study of the 1952 polio outbreak in the county. The study is designed to determine if either prior removal of tonsils or presence of pregnancy increases risk of contracting paralytic polio.

* * *

Dr. Ezra V. Bridge, superintendent of Mineral Springs sanatorium, was the main speaker at a district Christmas Seal workshop recently in Austin. Counties represented were Faribault, Freeborn and Mower.

Senior medical officer at Red Lake Indian hospital for the past six years, **Dr. Herman Kleinman,** is registered for a year's study at the University of Minnesota. He will receive a public health degree, returning to Red Lake in June.

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Representatives of five counties in southeastern Minnesota attended the district Christmas Seal Workshop in Rochester. Speakers on a question- and-answer panel at the noon luncheon meeting were **Dr. Ezra V. Bridges,** superintendent of Mineral Springs sanatorium; **Dr. David Carr** and **Dr. Bruce Douglass** of the Mayo Clinic, and **Dr. Viktor O. Wilson,** Rochester-Olmsted county public health officer. **Dr. Tom Mulrooney,** state TB field representative, was moderator and **Dr. H. Z. Giffin** of Rochester, Olmsted county Tuberculosis and Health Association president, presided.

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Dr. Richard M. Hewitt, Rochester medical writer and editor, was recipient of the 1954 Distinguished Service Award given by the American Medical Writers' Association. Dr. Hewitt is the senior consultant Section on Publications, Mayo Clinic; assistant professor of Medical Literature, Mayo Foundation, University of Minnesota; former assistant editor, JAMA. Dr. Hewitt has served as chairman of the Education committee of the American Medical Writers' Association since it was organized in 1951 and is largely responsible for the establishment of the new four-year courses in medical journalism and writing at the University of Illinois and the University of Missouri.

Dr. Hewitt was also awarded a Fellowship in the American Medical Writers' Association by the President of the Association at the banquet held on the eleventh annual meeting of the Association in Chicago. These fellowships are given "in recognition of high qualifications, personal and professional, and of established standing as a medical writer, journalist or publisher."

NEW LOCATIONS

Dr. Earl V. Wetzel, who has recently completed a three years' residency in obstetrics and gynecology at Ancker Hospital, has become associated with Dr. Jane E. Hodgson, St. Paul. Dr. Wetzel was previously in general practice in St. Cloud for five years.

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Just returned from overseas service with the Navy is **Dr. William H. Card,** who has moved into the office of the late Dr. William J. Noonan in the Minneapolis Medical Arts building. Dr. Card, who served with the Navy for two years, will practice urology.

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Dr. John Verby, a member of the Rochester baseball team in 1947, has returned to that town to join Drs. H. A. Wente and James Doyle in their medical practice. A graduate of Carleton College and the University of Minnesota School of Medicine, Dr. Verby served both with the Navy in World War II and with the Army Medical corps in Korea.

Now practicing in Bricelyn is **Dr. Elbert Gamble,** who has completed a year's residency in psychiatry at the Hastings State Hospital. Dr. Gamble, a native of Albert Lea, was graduated from the University of Minnesota Medical School in 1952, and served his internship at Ancker Hospital.

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Dr. T. G. Rollins, formerly of Hoopa, California, is now associated with Dr. Sidney Finkelstein at the Chippewa Hospital, Cass Lake. Dr. Rollins will be senior assistant surgeon of the United States Public Health Service.

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Opening new offices in St. Cloud is **Dr. Everett J. Schmitz,** who will limit his practice to general and thoracic surgery.

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Dr. George Friedell will leave his practice at Ivanhoe to become a member of the staff of Mount Sinai Hospital, Minneapolis. He will open offices in St. Louis Park.

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Dr. Richard P. Groschupf has joined the staff of the Bemidji Clinic as a general practitioner. He is the son of Dr. and Mrs. T. P. Groschupf of Bemidji, and a medical graduate of Creighton University in Omaha. He interned at St. Mary's hospital, Duluth.

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Dr. G. W. Schossow, a North Dakotan, has moved into Erskine's new clinic and has begun practicing. Dr. Schossow is a graduate of the University of Nebraska, and did his internship at St. Luke's Hospital, Fargo.

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Dr. Charles J. Hedlund, formerly of Northfield, has taken over the practice in Owatonna of **Dr. R. J. Wilkowske.**

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Dr. Newton D. Smith, a member of the Mayo Clinic staff since 1929, and an associate professor of proctology in the Mayo Foundation, will leave the clinic to practice proctology at Fort Worth, Texas.

* * *

Dr. John Pone, a graduate of the Universities of Latvia and Munich, Germany, has joined **Dr. K. A. March** in his practice at Cambridge. Dr. Pone practiced in Bavaria from 1929 to 1948, and was licensed in Minnesota in 1952.

MINNESOTA BLUE CROSS-BLUE SHIELD

From an analysis of Blue Shield data for the first six months of each of the year 1953 and 1954, it is found that the cost of claims, the number of services paid, the average number of claims per month, together with the average cost per claim, have increased for this period in 1954 as compared with that of 1953.

During the first six months of 1953, claims cost amounted to $2,188,888, whereas for the first six months of 1954, payment of claims amounted to $2,465,876. From these figures it is apparent that $276,988 more was paid for claims for the first six months of 1954 than during the same period of 1953.

For the first six months of 1953, 69,650 claims for Blue Shield benefits were received. During the same period in 1954, 77,471 claims for benefits were processed. It can be seen that the increase of approximately 1,300 claims per month, if projected over the entire year, indicates that as many as 15,000 more claims may be received in 1954 than in 1953.

An additional factor of interest is that the average cost per Blue Shield claim has also increased. During the first six months in 1953 the average cost per service was $29.11. For the first six months in 1954 an increase to $30.19 per service was realized. The increase is most marked in cases of obstetrical and medical services.

During the first six months of 1954, 7,688 participant subscribers were enrolled in Blue Shield bringing the net enrollment as of June 30, 1954, to 635,130 participant subscribers.

During the first seven months of 1954, 93,268 participant subscribers were enrolled in Blue Cross. Including cancellations the net enrollment as of July 31, 1954 was 1,006,537 participant subscribers.

A total of $10,319,273 was provided for hospitalization of Blue Cross participant subscribers, an increase of $1,650,415 over the same period last year.

BOOK REVIEWS

Books listed here become the property of the Ramsey, Hennepin and St. Louis County Medical Libraries when reviewed. Members, however, are urged to write reviews of any or every recent book which may be of interest to physicians.

CLINICAL APPLICATIONS OF SUGGESTION AND HYPNOSIS. William T. Heron, M.A., Ph.D., Professor of Psychology, University of Minnesota, Minneapolis, Minnesota. 2d ed. 137 pages. Price $3.75. Springfield, Ill.: Charles C Thomas, 1953.

The second edition of this small monograph gives a succinct and practical presentation of its subject. From the study of this book alone, most practitioners will not feel competent to use hypnosis, but they will appreciate much better what it has to offer. In addition, the part that suggestion plays in the practice of medicine is made more clear.

D.M.C.

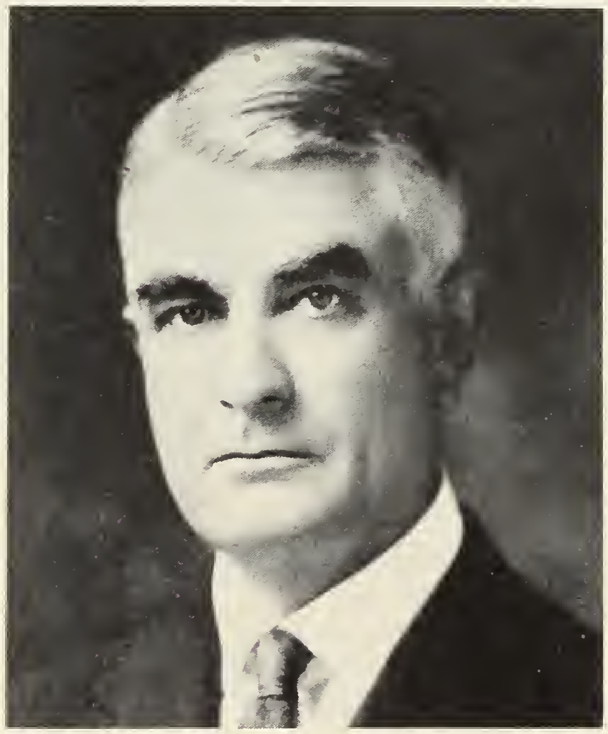
DIRECTORY OF PROFESSIONAL MOTION PICTURE FILMS AND AUTHORS. Compilation of information on nearly 3,000 films, 1,000 authors. 336 pages. Lawrence, Kansas: Professional Publications, 1954. Price $7.50.

This Directory of Professional Motion Picture Films and Authors is announced as the only comprehensive, classified, modern reference book on professional motion picture films and their authors. It enables educators to locate, evaluate, select and procure suitable films with a minimum of time and effort.

Handsomely bound, the Directory gives descriptive information concerning both films and authors, with special sections on appropriate films for training hospital personnel and educating lay groups.

Copies are available upon approval from the publishers, Professional Publications, 2010 Kentucky Street, Lawrence, Kansas.

The Mayo Memorial



William James Mayo

Charles Horace Mayo

The Mayo Brothers

HELEN CLAPESATTLE
Minneapolis, Minnesota

To the noble work and nobler vision of William James and Charles Horace Mayo is due the paradox of Rochester, Minnesota. They made of this "little town on the edge of nowhere" one of the world's greatest medical centers to which men come from the ends of the earth for treatment and instruction.

William was born in Le Sueur, Minnesota, in 1861; Charles in Rochester four years later. As sons of a busy horse-and-buggy doctor, one who believed that "children should be handy," the boys learned medicine almost with their a b c's. Among their boyhood chores, they drove their father on his country rounds, helped him with the lengthening queue of office patients, did his bidding at postmortems and operations. "We were reared in medicine as a farmer boy is reared in farming," said Doctor Will.

After graduation from medical school—William from the University of Michigan in 1883, Charles from the Chicago Medical College of Northwestern University in 1888—the brothers took over their father's practice. A disastrous tornado in Rochester and the consequent building of St. Mary's Hospital by the Sisters of St.

(Continued on Page 805)

Miss Clapesattle is Director of the University of Minnesota Press and the author of "The Doctors Mayo" first published in 1941. A condensed version of the book came off the press October 21, 1954.

The Mayo Memorial Dedicated

HAROLD S. DIEHL, M.D.
Minneapolis, Minnesota

A number of years ago as Mrs. Diehl and I stood on the rim of the Grand Canyon of the Colorado marvelling at the beauty and the grandeur of this wonder of the world, we noticed a man nearby walk away from the rim and then return and gaze again into the canyon; again walk away and again return. After repeating this performance several times I heard him say to himself, "I still cannot believe it." That is the way I feel about the completion of the Mayo Memorial. Even though we have been occupying and using it for sometime, I still feel like touching the walls to see whether they are real or whether they are a continuation of the dreams which we have had about this building for the past fifteen years.

Actually, the building is not only in use, but has been dedicated. The theme of the dedication was "Medical Education and Research: Freedom and Progress in the Mid-Twentieth Century." The program was a series of thoughtful, philosophical and forward-looking discussions of the opportunities and responsibilities of medical education and medical research in the years ahead. The speakers were distinguished guests representing the various fields of activity which are housed in the Mayo Memorial. Certainly nothing could be a more appropriate memorial to the vision, the faith and the achievements of the Doctors Mayo than such a program in dedication of a building which will be devoted to carrying on in perpetuity toward the objectives which they set for themselves and to which they devoted their lives.

Although I have written from time to time in MINNESOTA MEDICINE about the Mayo Memorial, a bit of recapitulation, now that the Memorial is a reality might be in order. The idea of a memorial to the Doctors Mayo was initiated by Governor Harold Stassen in the summer of 1939, a few months after the deaths of Doctor Will and Doctor Charlie. He appointed a Mayo Memorial Commission with the statement that it was fitting that the State of Minnesota should

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Dr. Diehl is Dean of the School of Medicine, University of Minnesota.

give appropriate recognition to its two most distinguished citizens. This commission spent several years considering the question as to the type of memorial which should be provided. The report of this commission was presented to the 1943 Legislature with the recommendation that the memorial should be a building on the University of Minnesota Campus to carry forward teaching and research in medicine. The Legislature, in joint session and by unanimous action, accepted this report and adopted the following resolution:

> Creating a Committee of Founders of the Mayo Memorial, Providing for the Appointment of Members Thereof, and Prescribing the Purposes Thereof.
>
> WHEREAS, the Doctors Mayo, William J. and Charles H., were two great humanitarians who brought to mankind many outstanding advances in medical science, and
>
> WHEREAS, we were proud to have them as citizens of our state while their works and their influence knew no bounds, and
>
> WHEREAS, they brought enduring fame to the State of Minnesota and substantial assistance and inspiration to the University of Minnesota, and
>
> WHEREAS, plans are now being formulated for a suitable memorial to the Doctors Mayo, and
>
> WHEREAS, it is but proper that the State of Minnesota should officially recognize and participate in such memorial,
>
> NOW THEREFORE, BE IT RESOLVED, by the Senate of the State of Minnesota, the House of Representatives concurring therein, that a committee of founders of the Mayo Memorial to carry out the plans for such memorial be hereby established; that three representatives be appointed by the Committee on Committees of the Senate, and that the Governor of the State of Minnesota be hereby requested to appoint such additional members as may be suitable, all for the purposes set forth in this resolution.

Pursuant to this action of the Legislature, Governor Stassen appointed Dr. Donald J. Cowling as Chairman of the Committee of Founders to make this memorial proposal a reality. The Committee of Founders was then appointed by Governor Edward J. Thye, who succeeded to the governorship when Governor Stassen entered military service. The Committee of Founders consisted of the following distinguished citizens of Minnesota: Governor's Appointments; Dr. Donald J. Cowling, Chairman, Dr. George Earl, Secretary, James F. Bell, Earle Brown, Mrs. George Chase Christian, Frank T. Heffelfinger, Jay C. Hormel (deceased), George W. Lawson, Ward Lucas, Most Rev. John G. Murray, I. A. O'Shaughnessy, Dr. Edward L. Tuohy. From the Legislature; Sen. Walter Burdick, Sen. Raymond J. Julkowski, Sen. Charles N. Orr (deceased), Sen. William B. Richardson (deceased),

FOYER, MAYO MEMORIAL AUDITORIUM

Sen. A. O. Sletvold, Rep. Claude H. Allen, Rep. Ben D. Hughes (deceased), Rep. Leo D. Madden, Rep. Mabeth Hurd Paige.

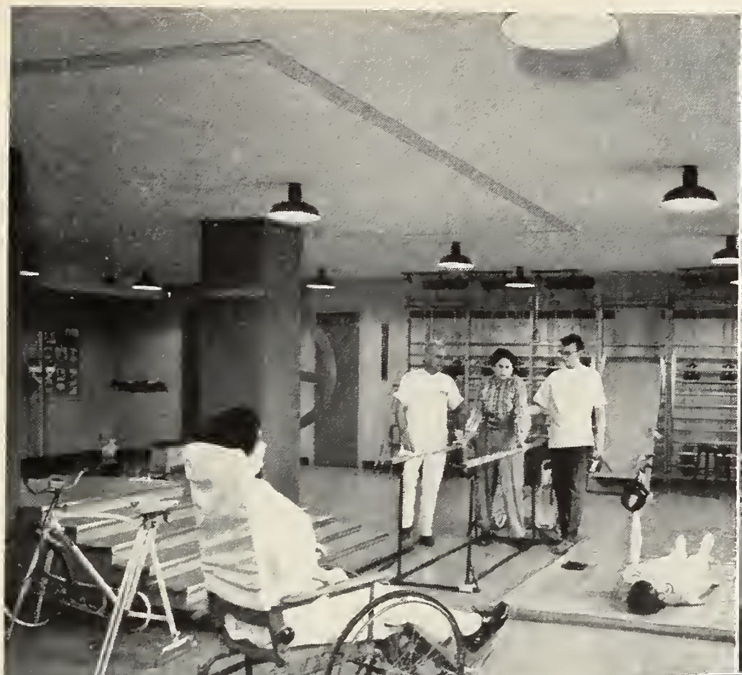
This committee, after due and careful consideration, decided that the most appropriate way to provide a memorial would be in part by voluntary contributions and in part by appropriations from public funds. The initial goal of the memorial commission was $2,000,000. However, as plans for a building to provide the needed facilities for medical research and medical education crystallized and as building prices increased several fold as a result of war and postwar inflation, it became obvious as time went on that much larger funds would be necessary to provide the facilities needed. This resulted in two appeals to the public for private gifts, several requests for legislative appropriations and for grants from federal funds. The difficulties, discouragements and disappointments seemed endless, but by the end of 1949 approximately $12,500,000 were available for the construction of a 22-story building as a Mayo Memorial. These funds came from the following sources: appropriations by the Minnesota State Legislature, $7,000,000; federal grants, $3,000,000, of which $2,000,000 came from Minnesota's allotment under the Hill-Burton Act for hospital construction and approximately $1,000,000 from the National Cancer Institute and the National Heart Institute construction funds; contributions from private gifts and other sources, $2,500,000.

At this time, everything seemed in hand and contracts were to be let not later than January 1, 1950. Delays in the completion of plans, however, resulted in the postponement of bids until the latter months of 1950 at which time, as a result of the outbreak of war in Korea, building prices had risen another 25 to 30 per cent. The result of this was that the building had to be reduced from twenty-two stories to fourteen stories. Four of these stories were moved to a different location where construction was less expensive. The other four stories, including two floors for the Medical Library and two floors for the Department of Pathology, were lost. These remain as such urgent and critical needs that a very real note of sadness dims the pleasure and satisfaction of a completion of this magnificent project.

The Mayo Memorial Building which we finally obtained is a splendid and invaluable addition to the facilities of the Medical School and University Hospitals. In general, this building contains the central service units of the University Hospital such as the operating rooms, the laboratories, the blood bank, the x-ray department, the administrative offices, the pharmacy, the record rooms, the central supply rooms, services and shops, expansion of the outpatient department, the visitors' entrance and main lobby of the hospital, the offices and research laboratories of the clinical departments. In addition, the Mayo Memorial contains underground animal quarters and associated research laboratories, the Rehabilitation Center, the School of Public Health, the Department of Bacteriology with its research and teaching laboratories, the administrative offices of the Medical School, the offices and conference rooms of the Department of Continuation Medical Education, the Mayo Memorial Auditorium with a seating capacity of 550 and two associated class rooms of 180 capacity each, and an underground garage with a capacity of 240 cars.

The elimination from this building of the Medical Library, the Bell Museum, and the offices, research and teaching laboratories of the Department of Pathology remains a very real disappointment and handicap. We hope that in the very near future the Legislature will provide funds to add to our University of Minnesota Medical Center facilities for these two important units of the Medical School as well as for the remodeling of Millard Hall and Jackson Hall which after forty years of use are urgently in need of reconditioning.

At the moment, however, we are rejoicing over the completion of this new building which not only serves as a magnificent and worthy memorial to Doctor Will and Doctor Charlie Mayo, but what is even more important, it provides the facilities for education, for research, and for service in medicine and allied health fields which will enable the University to carry forward for all time the work, the ideals, and the objectives to which these two great Minnesota physicians devoted their lives. This will be the most significant and vital memorial to all.



Gymnasium, Rehabilitation Center, 7th Floor

Main Lobby, Mayo Memorial

Department of Medicine Nursing Station, 3rd Floor

Research Laboratory, Department of Bacteriology and Immunology, 10th Floor

Recovery Room, Department of Surgery, 4th Floor

Learning

ALAN GREGG, M.D.

New York, New York

The memories we have of great men pass through stages, as time removes us further and further from their living presence. The first of these stages begins soon after their death. It starts with stories and anecdotes about them. Then come biographies, and at last what is called the "definitive life." The great man's actions and influence dominate such records and reminiscenses—as the transitive verb gives substance to a sentence because a verb is a word of action. Though he has died, his acts have still living witnesses and so continue to impress us. As long as there are still men living who remember them, the Mayo brothers' dynamic effect upon many an aspect of Great Medicine will act as a living stimulus and an unquestioned force.

When all his contemporaries too have gone, the reputation of a great man undergoes a sort of metamorphosis. This I would think of as the second stage. A kind of selective process sets to work upon the thousands of recorded acts of the great man, choosing some as especially characteristic, distinctive, and remarkable—in fact so much so that his very name comes to serve as a descriptive term, a standard for comparison, and a measure of excellence. We thus speak of Jeffersonian democracy, of Shavian wit, and of Christian virtues. So by an admiring but almost reckless selection and condensation, we turn a man's whole life into an adjective. We have chosen to remember him for one superlative characteristic of his personality. I rather think that statues, monuments, and memorial buildings come into being as the last of his contemporaries anticipate this second stage. In any event, the stage of selective commemoration deserves sharp scrutiny, for during this period we decide—often arbitrarily—for what achievement a great man is mainly to be known.

In the third and final stage, a great man becomes by general consent a noun, standing for a single aspect of his character, indeed becoming the personification, the type and print of some human quality. "He became," we say, "the Moses of his people." With such convenient clichés, Hippocrates became the synonym for the doctor, Robin Hood the incarnation of the beloved outlaw, Beau Brummel the model of sartorial excellence, or Daniel Boone the protagonist of pioneering. It is in this stage that patron saints and lesser deities arise; here is the birth of the myth, the unqualified, oversimplied, but unchallenged myth. Here, too, we must be on guard, for such magnifications and distortions as occur in myth-making correspond more closely to our desire for the myth than to the accuracy of its derivation. As loans often describe the need of the borrower more accurately than the character of the lender, so myths arise to meet the need for myths.

In this splendid building, you have well solved the problem of appropriate commemoration. In recognition of the greatness of the Mayo brothers, you have chosen to plan, construct, and equip a building to serve the material needs of medical research. Indeed, one might say not merely in *recognition* of the Mayos, but in *resonance* with one of the great qualities of those famous brothers, you have created circumstances that facilitate finding new knowledge. Your purpose was to catch something of their spirit in a kind of communion. In walking through this new building, I kept thinking of the most primitive and yet impressive form of communication—the African forest dweller's drum whose resonance so perfectly matches that of another drum sounding far away that it catches and continues sounds and messages from afar that otherwise remain unheard. This building, rightly called a memorial, could thus in a physicist's imagination be likened to a resonator that picks up the vibrant vitality of two great men and continues their resounding messages.

Happy are the Mayos to have had in you successors quick to understand their meaning in

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Dr. Gregg is Vice President of the Rockefeller Foundation.

Presented at the Mayo Memorial Dedication Dinner, Minneapolis, Minnesota, October 21, 1954.

American medicine, and determined to protect, continue, and extend their excellence. I would offer felicitations for this remarkable response to the remarkable example of two great men. President Morrill, Dean Diehl, Dr. Cowling, Mr. Amberg, and Governors Stassen, Thye, Youngdahl and Anderson, and all of your collaborators, I congratulate you.

The only solicitude that accompanies my congratulations concerns the certain fact that this wonderful building clearly snatches from its users one of the most convenient excuses for not reaching equally notable results in research work. With such a building, no one can attribute his failure in research to the lack of facilities, or to the difficulties of inadequate space or insufficient equipment. You have burned your bridges behind you and foresworn recourse to that subtlest of anodynes, self-pity. One of the commonest of excuses—namely that "facilities are lacking for adequate research work"—no longer exists to console you.

Indeed, if there were any chance that this building might be taken to be an appropriate memorial to the Mayo brothers merely because it facilitates and encourages fine work in medicine, I should raise my hand for permission to ask a question. And if granted permission I would say, "But I thought the Mayo brothers stand in American medical history as the incarnation of achieving without encouragement and working against odds. Can you commemorate the spirit by merely providing the flesh?" Their professional careers have become a legendary example of the aphorism "Where there's a will there's a way." Can a building devoted to the way epitomize or appropriately memorialize the will that lay behind the Mayos' accomplishment? Can you invoke the will merely by providing the way?

By now you will have seen why I drew your attention to what happens after great men have left us. In truth, their meaning to succeeding generations is in our hands. Their lasting significance will depend on what, out of all their deeds and characteristics, we choose to select for underscoring and eventually exclusive emphasis. I am concerned with our responsibility in the matter, for it seems to me that the Mayos mean something as important as it is elusive, as powerful as it is ignored, as significant as anything in our ideas of education and human achievement.

If there be a single word or phrase denoting or even connoting the quality of the Mayo brothers that I would like to convey to you and see associated inseparably with their memory, I could well make it the title, because it is the subject of this talk. Perhaps it is the word "learning"—not erudition, but learning as a habit and an adventure. The quality I seek to describe lies half exposed in such phrases as "unqualified freedom," "natural independence," "ingenuous creativeness," "unaffected and unhesitating originality." It emerges at times in such exclamations as "Why not?"; "I am not satisfied by what others have found"; "Have you tried it?"; "Will it work?"; "Let's find out."

Perhaps I can convey the quality I have in mind by outlining an hypothesis. From infancy onward, a human being continuously receives sensory impressions and those longer sequences we call experiences. Unconsciously, and even as a baby, he receives, associates, sorts, evaluates, interprets, and stores them. What he wants has everything to do with the values and interpretations he puts upon events or chains of events. Needless to say, the whole process is an intensely and absolutely individual affair.

I do not know any current definitions of curiosity that are better than superficial or circular, but most of us would agree that infants and children seem driven, albeit in different degrees, by what we call curiosity, to explore the world about them. The learning process goes on with or without education, and it goes on with an intensity that at times amazes any witness. Of two aspects of the learning process I am sure: one that it takes time, and the other that it results (in varient degrees) in that deep confidence in one's own judgment that makes a path for courage.

Long before his schooling begins, the child has taken decisive steps in learning how to take his world and how to make it. In fact, he is creating his own pattern for this purpose. His greatest need is time to find what is dependable, time to swing on the gate, time to organize his experience and to decide what to do about it. This learning process is continuous. It is loaded with the finest distinctions, the subtlest differences, the most startling surprises. Despite life's complexities and frustrations, the child, forever thus learning, finds what he can depend upon and when to doubt and how to unlearn. My impression is that before he gets to any kind of school, the child has already worked out much of his own way, his characteristic pattern, of taking the world—the kinds of evidence he trusts, where he can safely put his confidence, and what spells danger or failure or uncertainty.

But, sometimes before his pattern is firmly established, we put him to school, thus enormously complicating his life. Invoking a standard within the reach of all, we violate the tempo of his own individual learning process. We impose on him the average pace. This is too slow for him in subjects he has a natural gift for and too fast in the subjects where his aptitudes fall short. The formidable pressures of conformity succeed in penalizing his curiosity, threatening his self-confidence, and frustrating that will power that Ulysses Grant expressed in saying to his powerful critics that he would fight it out his way "if it took all summer." We standardize him. Exhilarating as that process may be as long as you have substandard persons in mind, standardizing superior capabilities carries with it a penalty unimagined, and perhaps unimaginable, by good average minds. The horror of formal education, like the horror of a dictatorship, lies in its very efficiency, and in what it kills or reduces to conformity, impotence and cowardice. Every human being, like every artist, should have time and complete freedom to find his own style.

Each of us had (and some few of us still have) his own independently acquired way of taking the world of experience, his own self-selected correctives, his own self-administered discipline. When we retain these highly individual characteristics, successfully surviving the standardized education imposed upon us, we possess what others are pleased to call originality, tenacity of purpose, forcefulness, courage, and a memorable personality.

I would suggest that where the usual educational processes at least refine our tactical ability, the slower but far more widely-ranging learning process provides us with strategic wisdom. Strategy concerns the choice of ends or objectives; tactics concern the means to attain the ends chosen by strategy.

I do not mean to belittle education, but I am certain that our greatest men, our greatest examples of human potentiality, either had little of the formal education of their time, or had none of it, or survived it because their personal pattern of the way to take the world was strong enough to persist unspoiled. Certainly, the correlation between greatness and having had a remarkable mother is higher than between greatness and a superior education. I say this without sentimentality: the mother is simply the person most probably providing the circumstances of the earlier learning period.

Beyond and above all else, the Mayo brothers stand as examples of continuing to learn, of sustained, tenacious, courageous, undeviating self-education. Their innovations amply deserve to be called original—but if originality connotes to you anything of the whimsical or irresponsible, then you must be reminded that the learning process as they followed it, by its very nature subjects experience to a more relentless evaluation than any outsider's examination or criticism. To me, the Mayos stand in American medicine as startling and unforgettable examples of something far deeper and far more rich and significant than education. Only the learning process—the primitive but also the final pattern for growth, self-impelled but self-correcting, forever gaining yet forever discarding, independent yet essentially humble and selfless—can explain the importance of the memory of the Mayo brothers to us all.

And lastly, if the spirit of research be, as I hold it is, the quintessence of the learning process, driven by innate curiosity, armed with the experimental method, controlled by self-imposed and undeviating honesty, and finding its reward in courageous equanimity, then a building devoted to medical research will not only stand, but will *act* as a fitting memorial to the Mayos.

DRUGS IN PEPTIC ULCER

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TREATMENT of peptic ulcer with drugs should be considered as only one of several means used to control the symptoms and modify the course of the disease. Perhaps we shall agree that, helpful as they may be, drugs are superseded in importance by such immeasurable and subtle factors as rest, peace of mind and soul, and freedom from undue fear, hurry, worry, and nervous, emotional and physical tension. The supreme importance of these influences is pointed out by the patient whose ulcer symptoms dramatically disappear when he leaves his office for a fishing trip, or enters a hospital for a nonrelated surgical procedure. The enforced rest, even in the absence of specific anti-ulcer therapy, is typically followed by remission of ulcer symptoms, often for several months, unless the ulcer is complicated by chronic perforation or obstruction.

It is apparent to all of us that the value of drugs in the therapy of peptic ulcer is difficult to measure. There are several reasons for this fact. One of them is the tendency toward spontaneous remission even without any specific treatment. Another is the question of attributing to each factor favorable toward the control of the ulcer the proportion of value due it. Still another reason is the unreliability of roentgenography in determining whether an ulcer is healed. The importance of these facts was emphasized when my associates and I did a controlled study of the effects of an orally administered preparation of enterogastrone on the control of ulcer symptoms. When the results were analyzed it was pleasing to see that 66 per cent of the patients so treated had either no ulcer symptoms or were definitely improved during the year of treatment. This apparent benefit seemed insignificant when we learned that 59 per cent of the patients who had received a placebo of inert material were similarly relieved.[2]

These preliminary remarks are made, not to decry the use of drugs, but to employ them with an open, inquiring mind so that, on the one hand, we shall not deny to any patient a drug that is beneficial, and on the other hand, not burden his already strained pocketbook with expensive drugs that may have no real benefit.

I should like to divide the drugs into four main categories and discuss them from that viewpoint.

Alkalies

The first group consists of the alkalies—the absorbable and the nonabsorbable varieties. I believe that, except in certain special indications, it is well to avoid the use of absorbable antacids. This is especially true in the presence of pyloric obstruction, alkalotic states and renal disease. It cannot be denied that certain patients seem to experience better relief with absorbable alkalies, and one may occasionally elect to use them in conjunction with the nonabsorbable variety for a limited time in properly chosen cases. Patients, as well as doctors, vary in their preference for the different brands of nonabsorbable alkalies, most of which contain aluminum hydroxide, either alone or combined with compounds of magnesium. It is desirable to use alkalies frequently, say every hour between meals during the day, not only while the patient is having symptoms but for perhaps four to six weeks after ulcer symptoms disappear. While the symptoms may disappear after a day of strict treatment, it is well known that four to six weeks, sometimes longer, may be required for the ulcer crater to disappear.

The use of alkalies seems to be one of the most logical measures in the control of peptic ulcer. I wonder, therefore, if we are giving our patients the utmost in prophylactic treatment if we do not insist that they take antacids regularly throughout the year, even during remission. I prefer to see these patients take a nonabsorbable alkali about one and one-half hours before and after meals and at bedtime during periods of remission. Too often I have seen patients who were

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taking their alkalies immediately after meals or immediately before meals, thus losing most of the beneficial effects.

Antispasmodics

The second group of drugs consists of the antispasmodics. Most of these drugs in proper dosage possess an incidental but less potent effect in reducing secretion of hydrochloric acid. In this group are the belladonnalike drugs and the anticholinergic drugs. The former drugs act chiefly on the parasympathetic nerve endings or effector site. Drugs of the latter group suppress the parasympathetic ganglia as well as the effector site by interference with the cholinergic transmission of nerve impulses.[8] The anticholinergic drugs suppress both motor and secretory function, the latter less consistently and less potently than the former.[5] In larger dosage these drugs may have a sympathetic blocking action and a curarelike effect.[8] The apparent advantage of the anticholinergic over the belladonnalike drugs is that in effective dosage they cause less disturbing side effects such as dryness of the mouth and blurred vision. That the former reduce both volume of gastric secretion and concentration of gastric hydrochloric acid seems fairly well established.[1,4,10]

Initial reports on the use of anticholinergic drugs were so enthusiastic that the possibility of their replacing conventional methods of medical therapy was considered.[3] Subsequent experience has demonstrated that although such drugs are helpful adjuncts, their use should in no wise obviate the need for other forms of treatment.[6,11] It is sometimes stated that these agents produce an effect of "medical vagotomy." However, unlike vagotomy, they have little effect on the cephalic phase of gastric secretion.[7-9] Their antisecretory effect may last from 30 minutes to several hours. There is no evidence for a cumulative action or tolerance with prolonged use. Their effect is to slow down gastric evacuation and to cause persistalsis to cease for a variable time. Propulsive and segmental activity of the small bowel is usually abolished.[8]

Some of the anticholinergic drugs currently in use are methantheline (banthine), diphenmethanil methyl sulfate (prantal), oxyphenonium bromide (antrenyl), and propantheline (pro-banthine) bromide. A number of similar compounds are under investigation in various centers.

Among the contraindications for the use of anticholinergic drugs are glaucoma, cardiospasm, organic pyloric obstruction, free perforation of an ulcer and obstructive disease of the prostate; also they should not be used just prior to or after gastric surgical procedures and just before roentgenographic study of the gastrointestinal tract.[8]

Sedatives

The third group of drugs is made up of the sedatives. They may be used either for their sedative or for their soporific effect. The latter effect may be especially helpful for the restless, nervous person prone to insomnia who is having nocturnal pain due to the ulcer. Perhaps their greatest usefulness is among patients hospitalized for ulcer therapy, who, of course, represent the more troublesome ulcers. The choice of barbiturate or other sedative to use and the dosage to be given vary somewhat with the preference of the physician and the patient. As we all know, sedatives and soporifics may be depressing mentally to some patients, which may negate some of their good effects. It seems best to give as small doses as possible for effect.

Miscellaneous Drugs

We might designate as miscellaneous the fourth group of drugs used for patients with peptic ulcer. While in some ways they are not thought of as drugs, the multivitamine supplements might be included here. These are indicated for all patients during periods of strict dietary therapy and may be desirable for certain other patients on ambulatory treatment. As a rule, the opiates are not indicated in the therapy of peptic ulcer but may rarely be required temporarily for pain caused by acute or subacute perforating ulcers unresponsive to other measures. This type of ulcer usually requires surgical intervention. Aspirin and related pain relievers are illogical agents for the pain of duodenal ulcer but may be required for pain due to associated conditions. Because of its irritating effect, aspirin should be taken with milk or with sodium bicarbonate and liberal amounts of water.

As stated at the outset, while various drugs are

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HOME, SAFE HOME

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ONE out of every fifty-two deaths in Minnesota last year was caused by a home accident. Deaths from accidents taking place in the home or yard occurred at a rate of one every fifteen hours—eleven every week of the year. While home accidents claimed the lives of more than 300 people sixty-five years of age or older, these mishaps also took the lives of 107 children under five years of age. In the age group five to fourteen years, home accidents were responsible for thirty-five deaths; in the age group fifteen to twenty-four years, eleven deaths; in the age group twenty-five to forty-four years, twenty-nine deaths; and in the age group forty-five to sixty-four years, fifty-eight deaths (Table I).

TABLE I. HOME ACCIDENT DEATHS BY CAUSE AND SPECIFIC AGE GROUPS
Minnesota, 1953

Type of Accident	All Ages	0-4 Yrs.	5-14 Yrs.	15-24 Yrs.	25-44 Yrs.	45-64 Yrs.	65 & Over
Home Total	568	107	35	11	29	58	328
Poisonings (gas excepted)	16	6	—	—	4	2	4
Poisonings by gases and vapors	22	—	4	1	8	5	4
Fire, explosion of combustible material	87	20	8	—	12	12	35
Hot substance, corrosive liquid, steam	9	8	—	—	—	—	1
Mechanical suffocation	35	30	—	1	—	1	3
Firearms	28	3	10	7	1	4	3
Falls on same level	153	—	—	—	—	8	145
Falls to different level or unspecified	156	9	1	1	1	21	123
Other specified home accidents	62	31	12	1	3	5	10

TABLE II. ACCIDENTAL DEATH SUMMARY
Minnesota, 1953 and 1952

Type	1953	1952	Change + or −
Occupational	156	157	0
Home	568	591	− 4%
Motor vehicle	668	575	+19%
Public	357	317	+13%
Late effects	25	18	+ 4%
Total	1774	1658	+ 7%

Leading cause of home accidents was falls, which were responsible for 309 fatalities. Fires, explosions and hot substances caused ninety-six deaths; poisonings thirty-eight deaths; mechanical suffocation thirty-five deaths; and firearms, twenty-eight deaths. Twenty-two of the poisonings resulted from poisonous gases or vapors, while sixteen were caused by accidentally swallowing poisonous substances. (Fig. 1).

Accidents of all kinds claimed 1774 lives in the state in 1953. Six out of every 100 deaths were due to accidents. Motor vehicle accidents ranked as the number one cause with 668 fatalities. Home accidents were in second place with 568 deaths, responsible for one-third of all accidental deaths. Public accidents such as drownings were next with 357 deaths, followed by occupational deaths with 156. The 1953 total of 1774 represented a 7 per cent increase over the 1952 total of 1658 (Table II). This increase was largely due to an upturn in the number of motor vehicle fatalities, which rose from 575 to 668. There was also a 13 per cent increase in public accidents. Home accident fatalities, however, decreased from 591 to 568. Of the home accident fatalities, 288 occurred in urban homes and 280 in rural homes. Rural non-farm homes were the scene of 203, while seventy-seven were rural farm home accidents.

More home accidents occurred in the month of January, when there were seventy such fatalities, than in any other month. Approximately 55 per cent of the fatal accidents oc-

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curred during the colder months of the year, October through March, reflecting the greater amount of time spent in the home. During the summer months of June, July, and August, there were 30 per cent fewer deaths than occurred during January, February, and March. In August there were fewer than half as many fatal home accidents as occurred in January (Fig. 2).

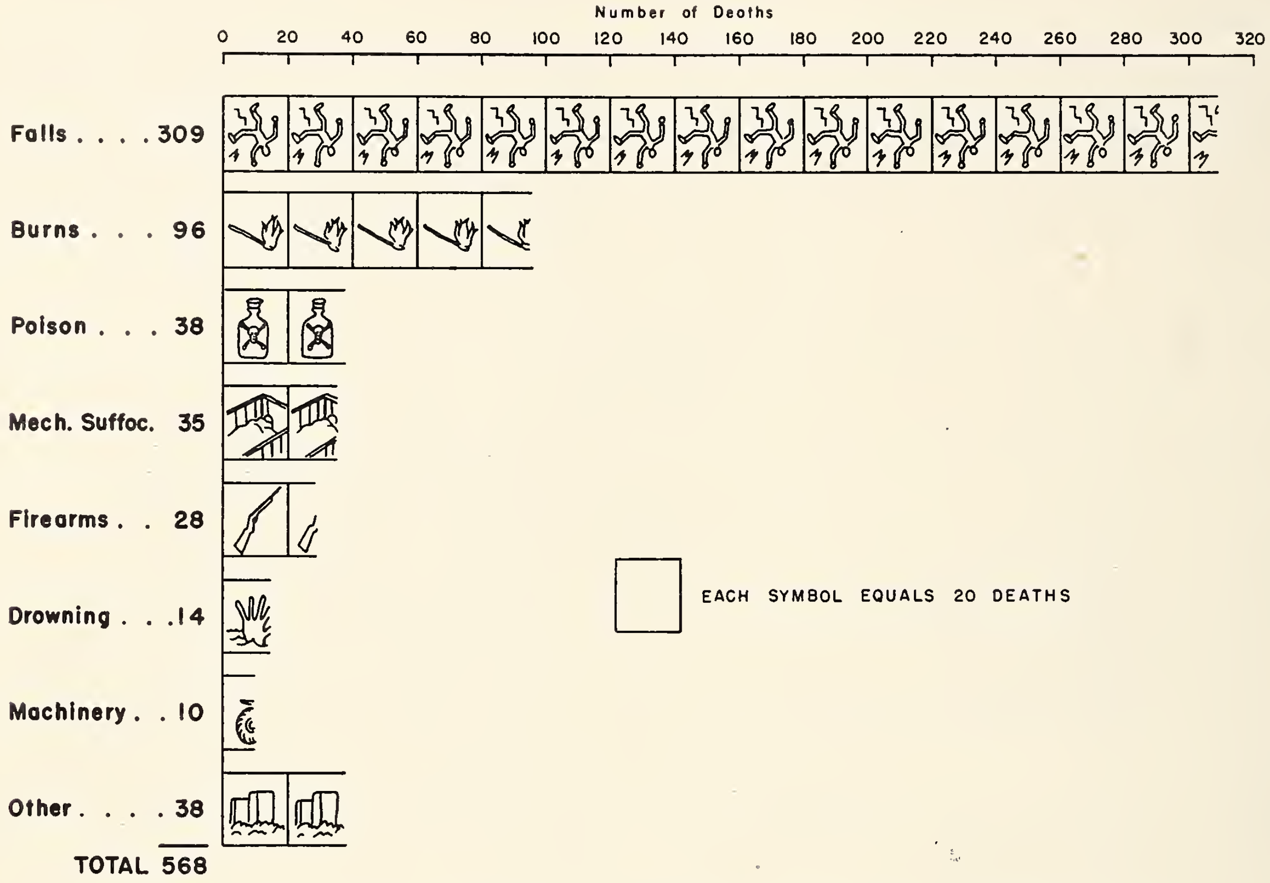

Fig. 1. Major causes of fatal home accidents by occurrence in Minnesota, 1953.

Falls

Falls took the lives of almost 300 persons forty-five years of age and older, 90 per cent of these being sixty-five years or over. Only twelve fatal falls were recorded for persons under forty-four years of age, and nine of these deaths occurred among children under four years of age. It is evident that falls are a particularly serious problem for those in the sixth decade and beyond. While falls are frequent in toddlers, they cause few deaths in that group. Fatal falls resulted from accidents in all rooms of the house and on the stairs. In some cases the victim tripped over a carpet or fell from a bed or a chair.

Falling from a chair resulted in the death of a seventy-four-year-old Todd county woman who had been confined to a chair for many years. "The accident could have been prevented if someone had been near," a relative wrote. The victim apparently lost her balance and slipped from the chair. This mishap points up the need for constant supervision of the infirm.

In attempting to saw a limb from a tree overhanging the garage roof, a sixty-eight-year-old Minneapolis minister placed a stepladder on the flat roof of the garage. As he was sawing the limb, he lost his balance. The ladder tipped from the roof, carrying him crashing to the ground. Death resulted from internal injuries. In writing about the accident, the victim's wife stated that if someone had been holding the ladder, the accident probably would not have occurred.

A scatter rug at the head of the basement stairs contributed to the death of a sixty-three-year-old Minneapolis man who stumbled as the rug slipped from beneath his feet. He plummeted head first down the stairway. The victim was dead on the arrival of the doctor, minutes later.

To help prevent falls, adequate lighting should be provided for stairs, with switches at both the head and the foot of the stairway. Stair carpeting and scatter rugs should be tacked down or non-skid rubber matting should be provided. Furniture should be arranged so that an open passage is provided from room to room. In the bedroom, bedside lights and light switches at doors help to prevent falls. Hand grips and bath mats for shower stalls and bathtubs help protect against falls in the bathroom.

Many falls occur when work is being done in the house or about the yard. Gardening tools and other equipment should be stored away when not in use, since implements left lying about the yard frequently cause falls. Makeshift ladders of boxes or chairs often cause falls. When a straight ladder is used it must be placed at the proper angle with the spreaders fully extended. To help prevent falls during the winter months, outside steps and walks should be kept free from snow and ice.

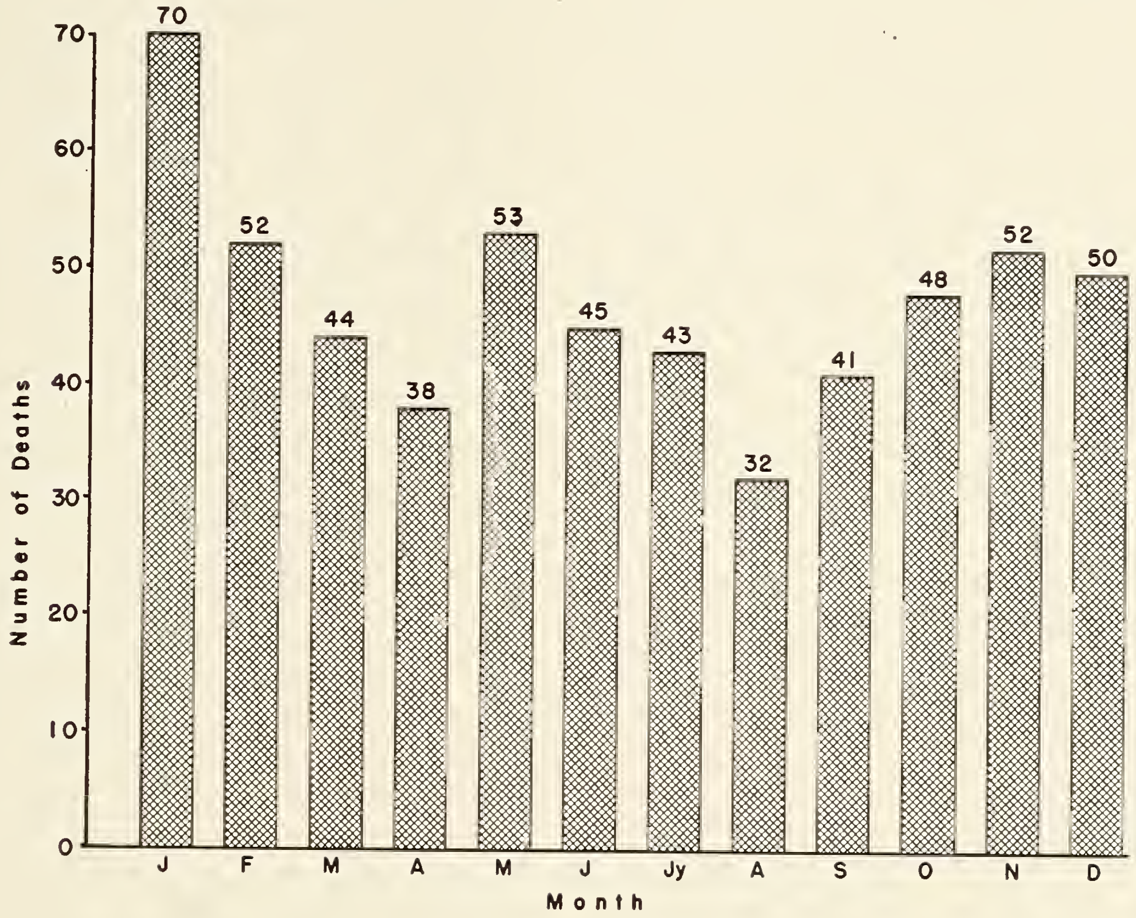

Fig. 2. Fatal home accidents by month of occurrence in Minnesota, 1953.

Fires, Explosions and Burns

Fires, explosions, and burns caused almost 100 deaths in the state last year. Twenty-eight of these deaths occurred among children under five years old, while thirty-six took place among persons aged sixty-five and over. Approximately one-fourth of these deaths were among persons twenty-five to sixty-four years old.

The danger of an elderly person burning grass was illustrated by an accident that occurred in Isanti county in the early spring. An eighty-six-year-old man started a fire in a pile of grass near his garden. The fire, whipped by a strong wind, got away from him. When he attempted to stop it, his trousers caught fire. Shock and second degree burns caused his death in less than twenty-four hours.

A twenty-eight-year-old dental technician was fatally burned in the garage of his Minneapolis home when he used an acetylene torch to remove the top of an empty metal drum. Flames from the torch ignited fumes from the drum. The resulting explosion caused fatal burns.

Smoking in bed or in the bedroom caused several fatal accidents in the state. In a south Minneapolis accident, a thirty-two-year-old woman was burned to death in the early morning hours when she was putting up her hair while smoking and her bathrobe flashed up in flames. The arson squad investigator termed it "a careless-smoker fire."

While sitting up in bed to light a cigarette, a thirty-year-old Minneapolis woman was fatally burned when a matchbox she was holding was ignited as she struck a match. The box fell into her lap, and the bedclothes were set aflame.

Faulty equipment played a part in several home accident deaths. The failure of the pilot light on a gas stove resulted in an explosion and fire in a Roseau county home which took the life of a seven-year-old boy. Gas leaked through the house from the stove, which had been turned on accidentally. The explosion was evidently caused by a spark or an open flame in another part of the house.

Hot lard from a french fryer spattering over an eight-month-old infant girl in Watonwan county caused the child's death when she climbed up by means of the table leg and pulled down the fryer and its boiling contents. The accident would not have occurred if the fryer had been placed out of the child's reach, the father of the victim stated.

The danger of entering a burning house was illustrated by a fatal accident in Fillmore county. A sixty-year-old man returned from a trip to town to find his farm home burning. He dashed into the house to call the fire department, and was unable to get out again. Suffocated by heavy smoke, he was burned beyond recognition. The fire apparently started either from the furnace or faulty wiring.

To prevent fires, chimneys, flue passages, and heating equipment should be inspected annually. All equipment such as heating, lighting, and gas apparatus should be installed by experts, since faulty installation often results in fires.

Hot ashes should be deposited in all-metal containers. Removing from the house all rags smeared with oil, grease, or paint prevents fires resulting from spontaneous ignition. Bonfires should never be left untended and burning should be done in a close-mesh wire basket.

To help prevent burns and scalds, babies and toddlers should not be allowed in the kitchen, particularly when meals are being prepared. Containers of boiling water or grease should be placed so that they cannot be reached by children. All pot and pan handles should be turned inward with the handles placed so that they do not extend over lighted burners.

Scalding water in the shower or bathtub is frequently responsible for serious burns. Shower or bath water should be tested with the hand or foot to make certain it is not too hot. Matches should be stored in metal containers out of the reach of children. It is a sound safety rule never to smoke in bed.

When there is a gas leak, all open flames as well as the main gas supply must be turned off. It is extremely dangerous to use a lighted match or candle to locate the leak. Even the spark from snapping on an electric light may cause an explosion. The leakage should be reported to the gas company or to a qualified plumber immediately.

Serious burns and electrocution can result if electrical appliances are touched when the body is wet. Electrical equipment that requires lifting or moving should never be used in the bathroom. A broken kitchen clock, a worn extension cord, and a heavy kitchen stove all played roles in a series of events that led to the electrocution of a nine-month-old baby boy in Stearns county. An older child in the home had broken the kitchen clock. The mother moved the stove to sweep up the glass from the clock. Because she was unable to push the stove back into place alone, the baby was able to crawl between the stove and the refrigerator and reach the extension cord which had been used to connect the clock. Wet, and with his body against metal, the baby bit through the extension cord to the live wire and was instantly electrocuted.

How could the accident have been prevented? By pulling the extension cord from the wall immediately, the mother of the victim wrote. She added that the cord was old and was made of soft rubber which made it possible for the baby to bite through it. Worn cords and broken electrical fittings should be promptly replaced.

Poisoning

Poisonings by gases and vapors caused twenty-two deaths in the state in 1953. Seventeen of these deaths occurred among persons aged twenty-five and older.

A whistling noise in the engine of his automobile led to the death of its owner, a forty-one-year-old mining company employe in St. Louis county. The man drove his car into the garage to find the cause of the noise. He was found dead of carbon monoxide poisoning several hours later. The hood of the car was up, the engine was running, and the garage doors were closed. "If the garage doors had been left open, the accident would not have occurred," the wife of the victim stated.

A thirty-nine-year-old businessman in Hennepin county was asphyxiated by carbon monoxide from smoke when the wood paneling in his home became ignited by a charcoal burner attached to the fireplace. Asleep in a second floor bedroom when the fire broke out on the first floor, the victim was trapped by smoke and flames.

Carbon monoxide gas from an unvented gas heater used to heat a small unventilated cabin caused the death of a twenty-six-year-old salesman in Becker county. In commenting on the accident a Becker county official recommended that a state law be passed to make it compulsory to "outside vent" all open gas-burning heating units.

The engine of a car should never be run in the garage unless doors or windows are open. As soon as the engine starts, the car should be moved into open air. If the engine must be run in the garage, all doors and windows should be wide open. A car with its motor running in a closed garage can create enough lethal carbon monoxide in approximately 4 minutes to cause death. Carbon monoxide can also be built up in a stalled or parked car while the motor is running.

When lighting a gas stove or heater, the safest method is always to strike the match first, then turn the gas on. When the gas is turned on first, it will continue to escape unnoticed if it is not lighted immediately. Water boiling on a gas stove should be watched so that it does not boil over and put out the flame. All rooms heated by gas heaters should be well-ventilated.

Accidentally swallowing poison resulted in sixteen deaths in the state last year. Six of these deaths were among children under four years of age and the same number occurred among persons over forty-five.

A sixty-six-year-old Saint Paul airlines employe went to the medicine cabinet one evening to take castor oil. To disguise the taste he mixed the castor oil with orange and lemon juice. About twenty minutes after he had swallowed the mixture, he went into a deep snoring sleep, which proved to be a coma. Examination of the bottle containing the liquid revealed that he had taken mosquito repellent by mistake. Five days later the victim died in a St. Paul hospital.

The victim's daughter said that the bottle of castor oil and mosquito repellent were of exactly the same size and shape. She added that the mosquito repellent was not labelled poison, nor were the contents or an antidote stated. She suggested that in order to prevent such accidents, poisons should be clearly labelled as poisonous, the ingredients should be clearly printed, and the antidote should be included on the label.

In Renville county a one-year-old child drank a bottle of cleaning fluid when the mother, who was using the fluid to remove spots, turned her attention to other duties for a few moments. The child died of benzine poisoning 48 hours later.

To protect against taking poison accidentally, medicine should never be taken in the dark. Before medicine is taken the label should be read twice. Poisons should be clearly marked and kept in a locked cabinet. Tape placed over the covers of bottles containing poison serves as a reminder of the contents. A list of antidotes should be posted near the medicine cabinet for use in case of emergency. If poisoning does take place, the proper antidote should be given and the doctor called at once.

Firearms

Accidents involving firearms caused twenty-eight deaths in the state last year. Thirteen of these deaths occurred among children under fifteen years old, eight were among persons fifteen to forty-four, and the remaining seven fatalities among persons over forty-five.

The danger of having firearms nearby when drinking is being done was illustrated by an accident in St. Louis county. A young man drinking in his home with his friends picked up a loaded gun and jokingly said, "Let's play Russian roulette." He placed the gun against his head, pulled the trigger, and was instantly killed.

In a Beltrami county home, a two-year-old boy and one of his friends were playing in the utility room. The mother of the two-year-old was doing her housework in another part of the house. "It was so quiet I didn't hear them or know they were in there until I heard the shot," the mother wrote in describing what occurred. Her child died ten minutes after he was struck in the head by the blast from a shot gun. "I don't think anyone knew there were shells in the gun . . . My husband and older son have been so careful with guns," the mother added.

Playing with a playmate at home, a ten-year-old in Carlton county was struck in the chest by a shell. The playmate was showing the youth how to operate the gun. During the demonstration, the gun went off. Death occurred almost instantly. "Teaching by the parents on the proper handling of firearms would have prevented the accident," a county official said.

A two-day vacation at a lake cottage in Otter Tail county ended in tragedy. Two boys had been spending their time at the cottage, fishing, swimming, and sharpening up their shooting eyes by firing at targets. They had just come into the cottage after target practice. One of the boys placed the gun on the table. It accidentally discharged and the bullet struck his friend in the heart, killing him almost instantly. Faulty equipment played a part in this accident. The boys had been using a .22 caliber rifle which had been converted into a pistol. It was found to have a defective firing pin.

Among the main causes of firearm accidents are failure to unload guns, cleaning them improperly, dropping them or knocking them off a support, exhibiting them, or leaving them where children can find them. A gun should never be pointed at a person under any circumstances. Guns and ammunition should be kept in separate places and under lock and key.

Mechanical Suffocation

Mechanical suffocation in bed or cradle was listed as the cause of death of thirty children under the age of one in the state last year. Four deaths from suffocation occurred among persons more than forty-five years old and one was twenty-four years old. The following case histories are typical.

In a Goodhue county home a two-month-old child was found dead in his baby buggy. The child was lying on his stomach with his face in the pillow.

A sixteen-day-old infant who was put to bed at 11:00 p.m. in a Hennepin county home was found dead at seven the next morning. Cause of death was asphyxia. The child was apparently in excellent health when put to bed.

In a Dakota county home a fourteen-day-old baby was placed in his crib for his afternoon nap. His two-year-old brother came into the room to get toys from a toy box beside the crib and accidentally pushed the blanket which was on the toy box into the crib and over the baby's face. The infant was found dead a short time later.

Many so-called cases of suffocation by bed clothes are actually the result of an acute respiratory infection, frequently virus in origin. While some of these deaths may be due to suffocation, many are due to unrecognized disease. Physicians and coroners should seek the actual cause of death by an adequate autopsy. Under such circumstances parents, too, should insist on an autopsy. Only by this means will it be possible to avoid that unfortunate "guilt complex" that parents may develop in the belief that carelessness was responsible for the death of their baby.

Farm Accidents

There were 280 rural home fatalities. Of all rural fatalities, 73 per cent (203) were nonfarm and 27 per cent (seventy-seven) were farm accidents. Falls caused seventeen farm deaths; firearms accounted for fourteen; fire and explosion caused thirteen, farm machinery caused ten (Table III).

TABLE III. FARM HOME ACCIDENT FATALITIES
Minnesota, 1953

Type	No.	Per Cent
Falls	17	22.1
Firearms	14	18.2
Fire and explosion	13	16.8
Machinery	10	13.0
Drowning	7	9.1
Poisoning (solid, liquid, gas)	4	5.2
Mechanical suffocation	3	3.9
Other	9	11.7
Total	77	100.0

There were 106 drownings in 1953, an increase of 18 per cent over the ninety drownings in 1952. While most drownings were public accidents on lakes and rivers (ninety-two), an additional fourteen were home drownings. Of these, six occurred in or around non-farm homes and included two infants under one year of age who drowned in their baths when their mothers left the room for a few minutes. This emphasizes the danger of leaving young children alone in the bathtub. Eight were rural farm home drownings, all children under the age of fifteen years. Four were one year of age. Of these, three drowned in open stock watering tanks or troughs and one drowned in an unfenced lily pool in the farm yard. A nine-year-old boy fell off a raft on a pond in the pasture, an eleven-year-old boy drowned while wading in a field flooded by the Minnesota River, and two brothers, aged respectively thirteen and fourteen, drowned in a gravel pit. None could swim.

All non-occupational farm machinery fatalities occurred in children. Eight of the ten machinery accidents were in children between two and five years of age, one was aged ten and one 11. Six of the fatalities involved tractors, indicating the danger of permitting children to drive or ride on tractors.

Summary and Conclusions

Home accidents were responsible for one-third of all accidental deaths in Minnesota in 1953. This is just 5 per cent less than the number caused by motor vehicle accidents and is a slight decrease from 1952. Falls were responsible for more than one-half of all home fatalities, with more than 85 per cent of such falls occurring among people sixty-five years and older and only 3 per cent in children under fifteen years. Seventeen per cent of home accident deaths were caused by burns, more than one-third of the victims being under age fifteen and a similar number sixty-five years and older. Poisoning was in third place, causing thirty-eight deaths. It was followed by mechanical suffocation, firearms, drowning, and farm machinery. Home fatalities occurred mostly in older people, only 25 per cent of the deaths occurring in children under fifteen.

A study of home accident fatalities indicates that most accidents are due to carelessness, thoughtlessness, ignorance, impatience, haste, and failure to use common sense. Elimination of all hazards in and around the home and a well-developed safety consciousness are essential in reducing the toll of preventable home accidents.

INCREASE IN NURSES

Nearly 400,000 nurses—an increase of 16,000 since 1950—are now working in the United States, according to new figures announced by Surgeon General Leonard A. Scheele of the Public Health Service, U. S. Department of Health, Education, and Welfare. Estimates based on the latest available figures from the forty-eight states and District of Columbia show a total of 389,600 professional nurses in active practice in the Nation. However, the demand for nursing services is so great, Dr. Scheele said, that the present recruitment goal for the Nation as a whole is 55,000 student nurses a year.

The number of hospital nurses, the largest single group, has increased by 15 per cent in the last four years to a total of 231,000. Private duty nurses, the next largest group, who are also at the bedside, number 74.000. The 35,200 nurses working in doctors' offices; 25,300 public health nurses, 14,000 industrial nurses, and the 8,200 nurse educators in schools of nursing make up the remainder of the total, along with 1,900 nurses in a variety of other fields.

The ratio of all nursing personnel, including student nurses in general hospitals, has risen from 69 per 100 patients to 74 per 100 patients since 1950—making it the highest in history.

"The steady rise in the nurse supply of the nation is significant to the total health picture," Dr. Scheele pointed out, "because nurses play such a vital part in all phases of our national health. For that very reason the nursing profession today offers a wide variety of excellent opportunities in the field of public health, in hospitals, schools, industry, doctors' offices, and, of course, in our schools of nursing."

INJURIES TO THE DIAPHRAGM

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THE diagnosis and treatment of diaphragmatic hernia has received more consideration in recent years because more frequent recognition has changed its status from that of a rare condition to one that is not infrequently encountered. This subject is of interest to the clinician and the radiologist, because the diagnosis is of first importance, the symptoms are often complex, and the differential diagnosis often difficult. The subject is of primary concern to the surgeon since operative intervention with restoration of the normal anatomic relations is the only method that promises relief of symptoms to the patient.[15]

With the increasing number of automobile accidents, traumatic hernia and rupture of the diaphragm are becoming more frequent, but are often overlooked. Many cases of acute obstruction are recorded that are found to be due to diaphragmatic hernia or rupture of the diaphragm from an injury which occurred years before the acute episode of obstruction.[4,6]

Rupture of the diapragm should be considered in the differential diagnosis of every severe compression of the chest or abdomen regardless of whether or not the chest wall is intact.[16,18] This is particularly true if a deformity of the left diaphragm is noted on the chest x-ray or if there is an unexplained density in the left base, partially obscuring the diaphragm without other evidence of pulmonary infection.[17]. After any crushing injury of the diaphragm, it is essential to be sure of the integrity of the diaphragm before discharge of the patient.[20]

Embryology

The formation of the diaphragm must be considered to understand the usual locations of these hernias and ruptures. The anterior, lateral, and central parts (forming the greater portion of the adult diaphragm) are formed from the transverse septum and the fused ventral mesentery. The remaining, posterolateral portion is formed by the fusion of the dorsal mesentery and the mesoderm derived from the receding Wolffian body and the pleuroperitoneal membrane derived from the pulmonary ridge. The mesodermal cells from the Wolffian body form the right and left crura of the diaphragm. The pleuroperitoneal membrane grows ventrally and, by fusion with the transverse septum, closes the remaining opening which may be designated as the "pleuroperitoneal hiatus," between the peritoneal celom and the pleural celom. This completes the formation of the posterolateral portion of the diaphragm.[12] Hernias are prone to occur through these points of fusion or locations of normal openings. Harrington believes that classifications based on etiology, pathological anatomy, presence or absence of sac, et cetera, are of little value. He suggests classification in two main groups—traumatic and non-traumatic.[11]

Etiology

1. *Indirect Injury*[18,23]

(a) This may be due to a tearing or bursting force causing a greatly increased intra-abdominal pressure. Jack-knifing of the body is an example.

(b) A sufficient lateral pressure on the chest and abdomen will distort the normal ovoid shape of the diaphragm and may produce a laceration.

2. *Direct Injury*

(a) Knife and gunshot wounds.

3. *Inflammatory Necrosis*

(a) Sub-diaphragmatic abscess or necrosis from drainage tubes in empyema.

Location

Traumatic rupture of the diaphragm is the second most common type of diaphragmatic defect in adults, the most common being the esophageal hiatus hernia.

In a later compilation, Harrington[14] found seventy-one traumatic hernias in a total of 559 cases. Sixty-nine of these were on the left side and only two on the right side. Hedblom[15] has stated that 95 per cent of traumatic diaphragmatic hernias occur on the left side. Strode[21] found only five cases of right side herniation in a comprehensive review of the literature.

The most common sites of traumatic rupture are the dome or the posterior half of the left

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TABLE I. ETIOLOGY AND LOCATION OF HERNIA IN 430 CASES[15]

Site of Opening	Cases	Cause
Esophageal hiatus.	308	Congenital defect.[17] (History of trauma)
Short esophagus type.	35	Congenital defect.[12]
Hiatus, pleuroperitoneal. (Foramen of Bochdalek)	9	Congenital defect.
Absence of posterior fourth of left diaphragm.	12	Congenital defect.
Foramen of Morgagni. (Subcosto-sternal)	8	Right side..... 6 Bilateral 1
Left diaphragm.	57	Trauma. Indirect ......44 Direct .... 7 Inflammatory.. 6
Right diaphragm.	1	Trauma. (Direct).
	430	

diaphragm.[15] It may be localized in the periphery of the diaphragm or may extend completely across the diaphragm into the esophageal hiatus.[22]

Signs and Symptoms

The increase in the incidence of rupture of the diaphragm may be apparent rather than real, and it has been said, that there is a direct proportion between the incidence of the condition and the frequency with which it is included in the differential diagnosis of any case of severe injury.[6] Many cases are not diagnosed until years after the initial injury and then only at operation or autopsy. Carter[4] collected thirty-four traumatic cases of strangulated hernia of which fourteen were correctly diagnosed. The interval between the injury and the attack varied from one day to sixteen years. The interval was less than three years in 85 per cent of these cases.

1. Immediate[8,18]—any or all may be present: shock, pain in upper abdomen, radiating to the shoulder, dyspnea, cyanosis, diminished chest expansion, impairment of resonance, adventitious chest sounds, cardiac displacement, asymmetry of hypochondria (due to displacement of abdominal viscera into the thorax), and changes in the percussion and auscultation findings, suggestive of pneumothorax.

2. Interval phase: Symptoms may be vague and suggest gall bladder disease, coronary disease, peptic ulcer, or incomplete intestinal obstruction. May have pleurisy with or without effusion.

TABLE II. LOCATION IN 91 CASES OF CONGENITAL HERNIA[10]

Foramen of Bochdalek	
Left	69
Right	13
Esophageal hiatus	5
Retrosternal area	
(Foramen of Morgagni)	4
	91

3. Phase of obstruction or strangulation: Symptoms depend on the organ or organs displaced and the degree of strangulation or obstruction. Incarceration may produce gaseous distension, eructation, obstipation, dyspnea, et cetera. The symptoms are not proportional to the degree of injury, but rather to the altered physiology of bowel obstruction, vascular engorgement, pulmonary ventilation or cardiac distress. Borborygmus over the thorax is pathognomonic.

X-Ray Diagnosis

The diagnosis was very difficult prior to the advent of x-rays. Bowditch (cited by Carter)[5] collected eighty-eight cases in the literature to that time and presented a personal case. His own, and one other case, were the only cases diagnosed prior to autopsy.

X-rays of the chest should be the starting point in the investigation. Lateral and postero-anterior chest x-rays may make the diagnosis, although it may be necessary to do a gastrointestinal series and/or colon x-rays. The x-rays may show what is erroneously interpreted as a pneumothorax.

X-Ray Findings

1. Simulation by the herniated viscus of high diaphragm. (This finding with a history of trauma should be considered as a possible diaphragmatic hernia until proven otherwise.)
2. Cardiac shift to the right—almost always present and noteworthy. (Not apt to be present with high diaphragm except with large amounts of fluid or viscera in the thorax.)
3. Air bubble above normal level.
4. Evidence of fluid in the thorax.

Treatment

Traumatic diaphragmatic hernias demand immediate attention if there is any evidence of injury to the abdominal or thoracic organs. Unless the repair is done soon after the injury, any abdominal organs forced into the thorax may become adherent to the raw edges of the torn diaphragm. Also, if the organs remain in the thorax for a time, they lose their right of domain in the abdomen. They become dilated due to mechanical interference with normal function and the abdominal cavity shrinks in their absence. The subsequent increase in intra-abdominal pressure after repair favors a recurrence of the hernia.[1]

The majority of surgeons have a preference for the thoracic route of repair in the uncomplicated adult case. A notable exception is Harrington.[15] He prefers the abdominal route in the left sided repair and reserves the thoracotomy route for the right side. In a report on sixty-seven cases of traumatic hernia, he confirms this (Table III).

In many early cases, evidence of an injury to abdominal organs may require immediate surgery. In these cases an abdominal approach may be best. If no injury is present, or if not too severe, the diaphragm may be repaired at the same time. Otherwise the diaphragm may be sutured through a standard thoracotomy incision at a later date.

If there is no real prospect of injury to the abdominal organs, most writers seem to prefer a standard thoracotomy approach, quite often through the eighth interspace or the bed of the eighth rib.

Treatment of primary shock is, of course, mandatory prior to surgery. The patient is given whole blood, plasma, or other parenteral fluids as required to correct his fluid balance. High oxygen concentration is supplied by the means at hand. It is advisable to have gastric suction in place and functioning. Sedatives are used sparingly, if at all. Antibiotics are given prophylactically at the discretion of the surgeon. Penicillin and Streptomycin are probably as satisfactory as other combinations.

Anesthesia is best given with the aid of a positive pressure machine and an inflatable cuff endotracheal tube. The type of agent is not as important as the familiarity of the anesthetist with the agent in use. It is important that the anesthetic allows a high oxygen concentration.

TABLE III. PLATE—SIXTY-SEVEN CASES[13]

1. Inflammatory necrosis	7
2. Injury	
a. Direct	6
(Gunshot wounds—5)	
(stab wounds—1)	
b. Indirect	54
(Auto accidents—42)	
(Crushing injuries, falls, etc.—12)	
	67
Location.	
Left side	65
Right side	2
Sex.	
Male	55
Female	12
Repair.	
Abdominal	64
Thorax	2
Combined	1
Abdominal Viscera involved:	
Stomach	66
Colon	59
Small Bowel	38
Spleen	30
Liver	20
Kidney	2
Operative deaths. 5 (7.4%)	
Pre-operative rupture of stomach	1
Intestinal obstruction	1
Pneumonia	3
Recovered without recurrence: 62 (92.6%)	

Technique

Most surgeons do a single or double layer closure of the defect with interrupted, nonabsorbable suture. When indicated, many surgeons reinforce the repair with strips of fascia lata. A temporary paralysis of the diaphragm is almost universal procedure. This is best done at the level of the diaphragm to cut down the incidence of permanent paralysis that occurs in a fair percentage of cases when done at a higher level. In a few cases where the condition of the patient precludes immediate surgery, it may be advisable to do a phrenic crush in the supraclavicular area to aid in relaxing the muscle to prevent spasm and produce relaxation.

There are various methods of getting additional tissue for closure of the defect where there has been extensive loss of substance of the diaphragm:

1. The diameter of the thorax may be narrowed by resecting a few inches of the 8th, 9th and 10th ribs at the angles.

2. Strips of fascia lata may be interwoven and then stabilized with interrupted sutures of cotton or silk.[15]

3. A large defect may be closed by turning down a flap of parietal pleura.

4. The diaphragm may be attached to the chest wall at a higher level.

5. Some cases with foramen of Bochdalek hernia present a problem in repair because of lack of peripheral diaphragm for attachment of the central portion of the diaphragm. It is possible to place a series of mattress sutures of silk or cotton through the edge of the defect and then bring them out through the chest wall and tie them over rubber tubing. If the edge of the diaphragm and the parietal pleura are scarified, the two edges will usually heal satisfactorily.[8]

The thorax is closed by usual methods unless there is extensive damage to the chest wall, in which case the closure is fashioned by whatever means available. It is advisable to have closed suction drainage. This is easily done by means of two or more catheters connected to a modified Carlson suction with a Stedman pump as a source of power. The negative pressure is regulated to 14-18 cm. of water by means of a water seal.

Postoperative Management

1. Patient is placed in oxygen tent with oxygen administered at a rate of 16 liters per minute.

2. Gastric suction is continued until the patient is expelling flatus (usually by the third day.)

3. Parenteral fluids are supplied as required.

4. X-rays of the chest are taken at the completion of surgery and then as required.

5. Negative pressure is maintained on the chest catheters until the lung is completely expanded and the catheter drainage is less than 50 cc. in twenty-four hours.

6. The patient is compelled to co-operate in coughing to keep the tracheo-bronchial tree clear of secretions. Aspirations and bronchoscopy are resorted to as necessary.

7. Antibiotics are given for the first five days and then as necessary.

Complications

These may be as varied as the extent of the chest injury and the injuries to the other organs. A few may be worthy of note:

1. *Pneumothorax*—usually controlled by a properly functioning set of catheters connected to an adequate suction apparatus.

2. *Ileus*—controlled by a properly functioning gastric suction apparatus.

3. *Hemorrhage*—properly done surgery with attention to good hemostasis will usually eliminate this difficulty. If it occurs it must be dealt with immediately and under proper conditions, i.e., in the operating room.

4. *Pleural effusion and empyema*—ordinarily the chest drainage and antibiotics will prevent these complications. If they occur, they will require the same treatment as if the diaphragm had not been the etiological factor.

5. *Pneumonitis and atelectasis*—proper tracheo-bronchial toilet and antibiotics will usually prevent these complications. Bronchoscopy may be required in a patient who will not or cannot cough up the secretions as they occur.

6. *Incisional hernia*—repair under proper conditions.

7. *Evisceration*—this is a surgical emergency and is thus treated.

8. *Recurrence*—the procedure is repeated under the most favorable conditions possible.

Case Reports

Case 1.—A forty-nine-year-old white man was sawing wood at about 3:00 p.m. on November 26, 1952. He was using a circular saw attached to the rear axle of his truck. The saw flew off the axle and cut the patient on the anterior surface of his chest and abdomen. The local physician closed the large sucking wound of the chest, using a very adequate pressure dressing, and gave the patient a whole blood transfusion while arranging for transportation over the 75-mile distance to a local hospital.

The patient received continuous oxygen therapy by means of a mask during his transfer.

The patient was admitted to the hospital in comparatively good condition at 5:00 p.m. the same day. Blood pressure was 140/90 with a pulse of 90. There was a laceration on the anterior surface of the body extending from a point 3 inches below the xiphoid process of the sternum diagonally upward to a point in the anterior axillary line of the axilla. Multiple puncture wounds extended out over the shoulder and upper humerus. This represented the course of the saw as it passed over the patient. There were four divided ribs.

The lung, heart and omentum were clearly visible through the laceration. There were multiple lacerations of the right hand and thumb.

Repair was begun at 5:30 p.m. with Baird's Solution (penthothal and curare) plus oxygen through an inflatable cuff endotracheal tube and a positive pressure gas machine. The wound was thoroughly debrided.

A 3-inch laceration in the diaphragm at the anterior chest wall attachment was repaired with interrupted 0 chromic catgut after an inspection of the upper abdomen showed no evidence of injury to the upper abdominal organs. The stomach was filled with gas. Fragmented portions of the fractured ribs were removed, preserving the periosteum. Lacerations of the upper left rectus and chest wall muscles were repaired with catgut. The skin was closed with silk. The remaining lacerations were repaired by the orthopedic surgeon while the chest wound was being closed. A number 22 Robinson catheter was placed in the chest through the eighth interspace and connected to a suction machine with 14 cm. of water suction. The patient received 2 units of plasma and 1000 cc. of whole blood during the procedure which was completed at 7:00 p.m. A post-operative chest film showed complete expansion of the lung. The catheter was removed on the third post-operative day. The patient received 300,000 units of penicillin, each 3 hours for five days and then 300,000 units of Duracillin for 3 days. The sutures were removed on the eighth postoperative day and he was discharged from the hospital on December 5, his ninth postoperative day.

This patient was last seen on March 25, 1953 when he had no symptoms referable to his injury. There was complete expansion of the lung with normal movement of the diaphragm. The patient has since moved to Illinois and on June 1, 1954 was working and asymptomatic.

Case 2.—Patient, aged fifteen, was injured in a car that overturned early in the evening of June 12, 1953. The patient was taken to a hospital in a nearby town and then transferred to a local hospital (a distance of 40 miles) when it was evident that he had a chest injury. He arrived at 11:50 p.m., blood pressure was 150/0 with a pulse of 150. Respirations were approximately 50 and the patient was cyanotic.

Physical examinations revealed a previously sutured laceration of the forehead. Breath sounds were absent on the left side. X-rays revealed a high diaphragm on the left side and a fluid level in an air containing sac beneath it. Abdominal examination was essentially normal. There was a wide separation and fracture through the left sacroiliac joint and ilium together with a fracture of both superior and inferior rami of the left pubic bone.

He received 500 cc. of whole blood, plus continuous oxygen by mask. A partial atelectasis of the left lung had become nearly complete by 11:00 a.m. June 13. A thoracotomy tube failed to drain any of the fluid from the chest and a nasal suction tube was inserted. This aspirated a large amount of fluid and a diagnosis of ruptured diaphragm with herniation of the stomach was made. Blood pressure was 150/70 at this time. Respiratory rate was 40.

Thoracotomy was begun at 12:40 p.m. on June 13, twelve hours after admission. The anesthetic agent was Baird's pentothal-curare solution with positive pressure 0_2 through an inflatable cuff endotracheal tube and a positive pressure gas machine.

The chest was opened through the left seventh interspace with the patient in the lateral position. The entire stomach and most of the omentum were in the left chest. There was an 8-inch tear in the diaphragm, extending from the mid-clavicular line anteriorly, to the left crus of the diaphragm and extending into the triangular ligament of the liver. The phrenic nerve was intact but had been stripped free of the diaphragm, the torn edges being separated by a distance of some 3 inches. This stretch had caused a paralysis of the diaphragm. The heart and lung were intact as were the spleen and stomach.

The diaphragm was repaired with interrupted 2-0 cotton. The lung was then expanded and patient's blood pressure and pulse returned to normal almost immediately. The incision was closed with chromic catgut for the muscle and silk for the skin. Two number 20 Robinson catheters were inserted into the chest through the ninth interspace and connected to a negative suction apparatus with a negative pressure of 14 cm. of water. The patient received 500 cc. of whole blood during the procedure which was completed at 2:00 p.m. Post-operative x-ray revealed good expansion of the lung with a normal position of the diaphragm.

The patient received 300,000 units of Duracillin, every 12 hours for five days. Nasal suction was continued for four days with parenteral feedings. He was returned to his home town hospital on June 19, his sixth post-operative day. At this time his lung was completely expanded and he was eating a general diet.

He was last seen on June 1, 1954 at which time he was asymptomatic. His diaphragm showed normal motion and the heart and lungs were normal.

Summary

A brief review has been made of the subject of trauma to the diaphragm. Two cases are presented—one due to direct trauma and one due to indirect trauma.

References

1. Baird, J.: Personal communication.
2. Berman, F. J.: Traumatic rupture of the diaphragm, J. Indiana M. A., 46:197 (March) 1953.
3. Bowditch, H. I.: Diaphragmatic hernia. Buffalo M. J., 9:1-39, 65-94, 1853-1854. (Cited by Carter[4]).
4. Carter, B., & Giuseffi, J: and Felson, B.: Am. J. Roentgenol. 65:56-72, 1950.
5. Carter, B. N.: & Giuseffi, J., Strangulated diaphragmatic hernia, Am. Surg., 120:210 (Aug.) 1948.
6. Denman, P. R.: Traumatic rupture of the diaphragm with medico-legal aspects. Texas State J. Med., 35:825 (April) 1940.
7. Dorsey, J. M.: Surgical repair in various types of

(Continued on Page 825)

BCG VACCINATION: TECHNIQUE AND EFFECT ON THE TUBERCULIN TEST

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AS THE INCIDENCE of tuberculosis decreases in the United States, a decreasing number of persons who reach young adulthood are cutaneously sensitive to tuberculin. In a number of Minnesota counties, less than 10 per cent of young persons react to tuberculin. This low incidence, indicating widespread lack of exposure to tuberculosis, is of some concern to those who must assign student nurses to work where they will come into intimate contact with patients whose secretions contain living tubercle bacilli. The problem also arises in connection with laboratory personnel engaged in diagnostic or research work in this disease, as well as with physicians who care for such patients.

At the instigation of Dr. C. H. Hodgson and his colleagues at the Mayo Clinic, and to provide a large number of positive reactors to tuberculin among student nurses, a program of vaccination with the bacille Calmette Guérin (BCG) was carried on at the Methodist-Kahler School of Nursing, Rochester, Minnesota.* This program was not started because of any serious incidence of tuberculosis among student nurses in this school; in fact, the occurrence of this disease has been minimal at this school during the past two decades. This report presents a description of the technique of vaccination used and its results with respect to immediate changes in sensitivity to tuberculin.

Technique of BCG Vaccination

BCG vaccination consists of administration of an attenuated strain of tubercle bacilli originally derived from the udder of a tuberculous cow. It is done in an attempt to produce some degree of immunity against subsequent exposure to tuberculosis. The vaccine is usually a liquid, milky suspension of living organisms; it is made up in various strengths depending on the intended method of administration.

The vaccination may be performed in one of several ways. The original method consisted of oral administration. This is a simple technique but it does not allow accurate determination of how much vaccine is absorbed. It also is not satisfactory for older children and adults, who do not absorb material from the alimentary tract as readily as do infants. The vaccine may be given subcutaneously but this method may cause abscesses and it is rarely used at present.

The technique of injecting 0.1 ml. of vaccine intradermally has been used since 1927. This method is accurate and fast; it is used for mass immunization in some parts of the world. However, draining ulcers occasionally appear at the site of injection.

A multiple-puncture method was described by Rosenthal[3] in 1939. In a later variant of this technique, a drop of vaccine was spread over an area of skin measuring about 1 by 3 cm. and then thirty tangential punctures were made with an ordinary needle through this vaccine into the skin. Modifications of this technique are extensively used at present, and gadgets have been devised to speed up the procedure. One of these, designed by Rosenthal, is a rectangular plate that contains thirty-six points; the plate is applied to the skin by means of a magnet. The multiple-puncture technique employing this plate was the method used in the present study. The materials and vaccine were supplied through the co-operation of Dr. Sol R. Rosenthal, director of the Institution for Tuberculosis Research, University of Illinois College of Medicine.

The student nurses selected for vaccination had had normal thoracic roentgenograms and had been completely insensitive to intracutaneous administration of both "single-strength" (0.0001-mg.) and second-strength (0.005-mg.) doses of purified protein derivative (PPD). The technique of vaccination was as follows: The skin of the upper and outer portion of the arm was washed with acetone. The surface of the magnet was washed with alcohol and a sterile plate, containing the thirty-six points already referred to, was placed so that one end projected beyond the surface of the magnet (Fig. 1). Two drops of a fresh suspension of vaccine was placed on the clean skin from a 20-gauge needle attached to a

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From the Section of Publications, Mayo Clinic, Rochester, Minnesota.

*This work was done by the author while a member of the Section of Clinical Pathology.

syringe containing the vaccine. The projecting end of the plate was then dipped into the drops of vaccine and used to spread it uniformly over the area of puncture (Fig. 2a). The plate was then centered on the surface of the magnet and enough pressure was exerted on the magnet by the palm of the hand to drive the points into the skin (Fig. 2b). At the same time the skin of the arm was stretched by the vaccinator's other hand placed under the site of the vaccination. A slight rocking motion of the magnet and plate facilitated deep penetration of the points. The magnet was then slid off the plate, leaving the points of the latter embedded in the skin. The plate was then lifted off the skin with the fingers and one end was used to tap the liquid vaccine into the holes produced in the skin by the points. The penetration of the points was deep enough to cause slight bleeding when the plate was lifted from the skin (Fig. 3a). The area was left open to the air for a few minutes and instructions were given not to wash the site of vaccination for twenty-four hours.

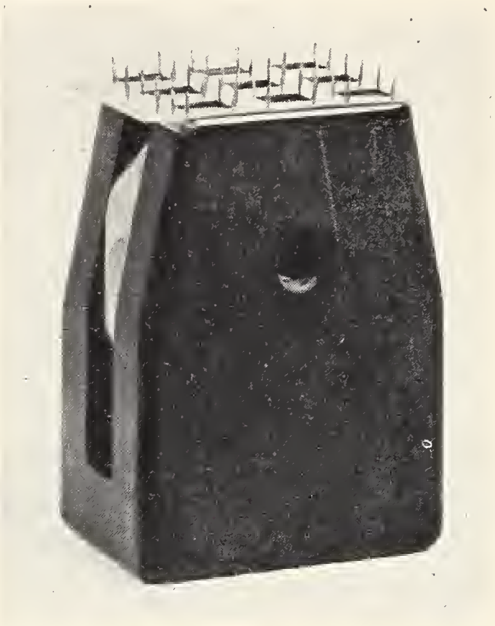

Fig. 1. Magnet and multi-pointed plate used in the Rosenthal multiple-puncture method of BCG vaccination.

An adequate local reaction usually was evident within a week by the appearance of papules situated at the individual puncture wounds (Fig. 3b). Slight lymphadenopathy occasionally was present in the axilla adjacent to the inoculated arm. The papules gradually diminished in size and, after six months or so, little or no local evidence of vaccination was present.

Effect on the Tuberculin Test

After preliminary observations, it became routine in this survey to administer 0.0002 mg. of PPD as a tuberculin test six weeks after vaccination in order to determine the rate and extent of conversion of the subjects to a tuberculin-positive state. This dose of PPD provided a maximal number of positive results without producing unduly severe reactions. All persons who experienced a doubtful or negative reaction to this dose, which is 10 times the standard first-strength dose of PPD, were given the standard second-strength dose of 0.005 mg.

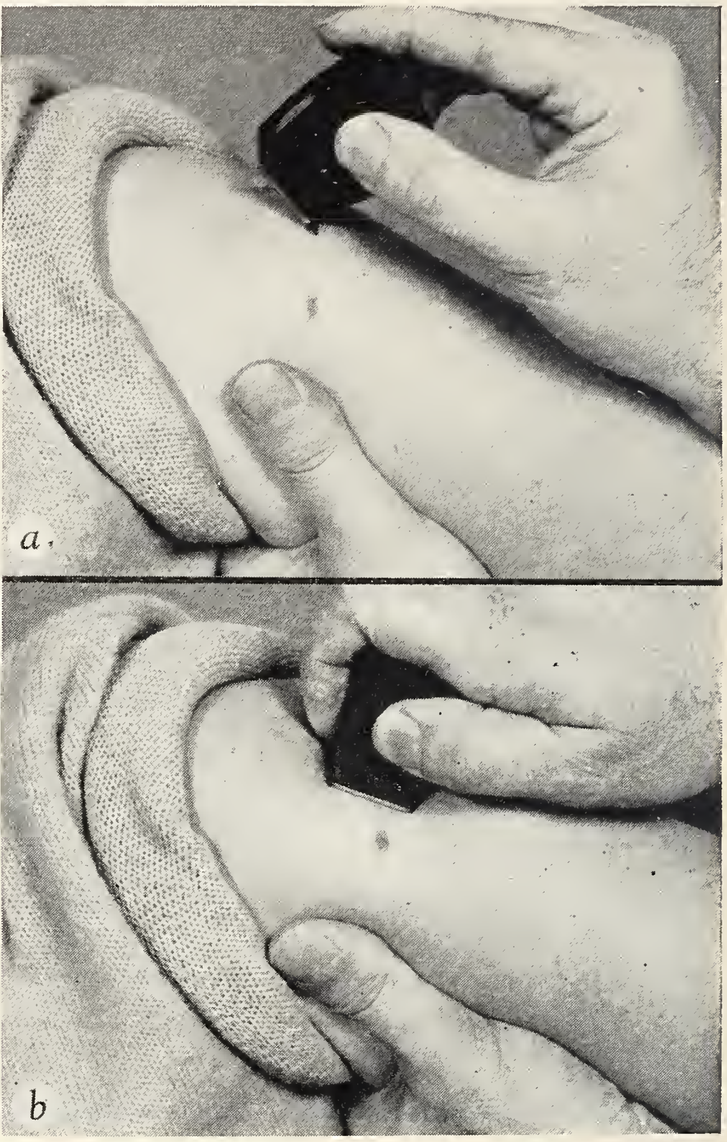

Fig. 2. (a) Use of long end of plate to spread vaccine. (b) Technique of cutaneous puncture.

More than 90 per cent of the student nurses reacted positively to tuberculin six weeks after vaccination. A typical experience is represented in Figure 4. The initial test (0.0002 mg. of PPD) induced definitely positive reactions in seventy-three (44 per cent) of the 165 persons. The stronger dose (0.005 mg. of PPD) was then given to the remaining ninety-two persons, who had reacted either doubtfully or negatively to the initial test; eighty-one of these experienced definitely positive reactions, whereas the results were doubtful in ten and negative in one. Thus a total of 154 (93 per cent) of these 165 student nurses became sensitive to tuberculin after BCG vaccination. The tuberculin reactions were classified according to the criteria of the National Tuberculosis Association, which do not require repetition. No 4-plus reactions were encountered in spite of the relatively large dose of PPD used for the initial postvaccination test.

Comment

No agreement exists with regard to the proper dose of tuberculin to be used in selecting persons for vaccination. If small doses of tuberculin are used, an increased number of persons who are not cutaneously sensitive will be made available for immunization, but there also will be an increased incidence of undue reactions to the vaccine. If a person who has undergone a primary tuberculous infection, and has been rendered sensitive to tuberculoprotein, is vaccinated, the Koch phenomenon usually takes place. This consists of a generalized accelerated reaction that, while not particularly dangerous, is uncomfortable. The accepted procedure for mass programs is to give 0.0001 mg. of PPD or its equivalent in old tuberculin (OT) as a single screening test. Every person who displays a negative reaction to this dose is then vaccinated.

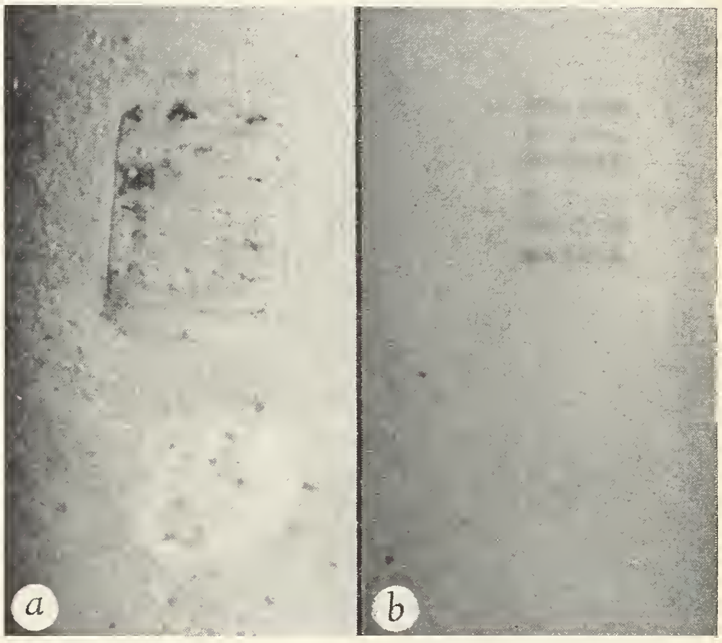

Fig. 3. (a) Site of vaccination after removal of plate. Note slight amount of bleeding. The plate was inserted twice in this instance for pictorial purposes. (b) Site of vaccination showing development of individual papules at each point of entry of vaccine.

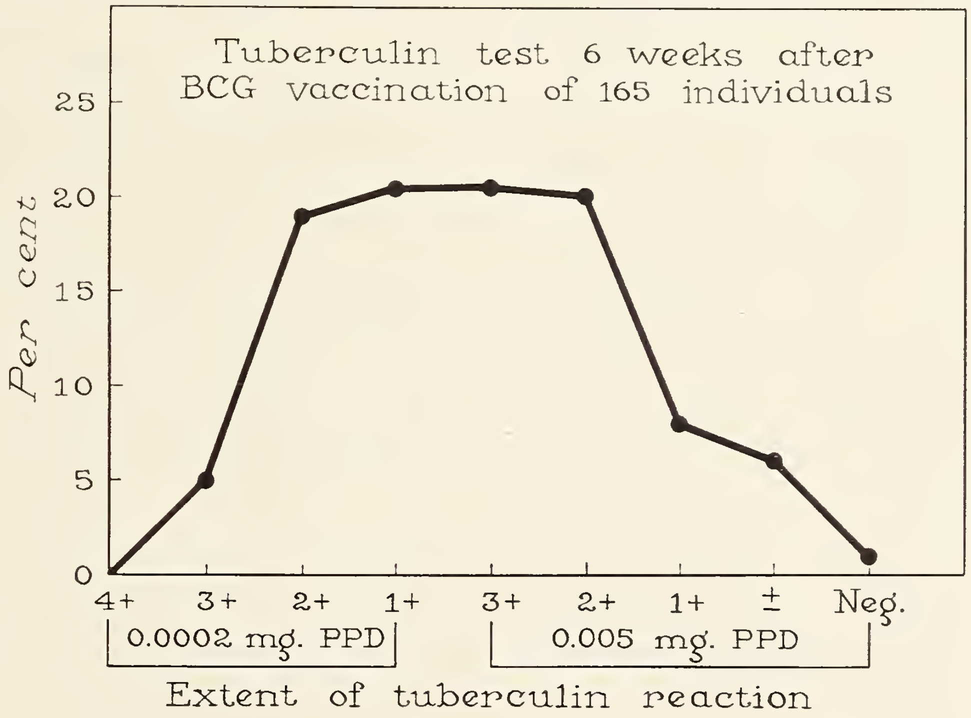

Fig. 4. Quantitative analysis of extent of tuberculin reactions in 165 consecutive persons six weeks after BCG vaccination.

Cutaneous sensitivity to tuberculin after BCG vaccination is the only immediate, tangible and objective evidence than any change in immunologic status has occurred. Opinion differs regarding the type of tuberculin to be used in postvaccination tests. Many workers insist on OT only, in the belief that use of this preparation will demonstrate the maximal number of conversions to a tuberculin-positive state. In preliminary work in the present program, simultaneous postvaccination tests were done on a number of persons with comparable doses of PPD and OT. The reactions were practically identical.

Rates of production of sensitivity to tuberculin after BCG vaccination depend on the technique of vaccination and the dosage of organisms. Some reports indicate that fewer than 50 per cent of persons vaccinated become sensitized. As already noted, the rate in this survey was more than 90 per cent. Some observers have obtained such consistently high rates that they no longer perform routine immediate postvaccination tuberculin tests. In the hands of experienced workers using either the intracutaneous or the multiple-puncture method with fresh, potent vaccine, production of sensitivity should approach 100 per cent.

Persistence of sensitivity to tuberculin induced by BCG vaccination also varies greatly. It depends, again, on techniques of vaccination, dosage of organisms and the severity of the initial postvaccination reaction. Persons who exhibit the strongest cutaneous reactions usually remain sensitive longest. Sensitivity produced by intracutaneous vaccination ordinarily remains longer than that seen after multiple-puncture vaccination. In some surveys many persons have displayed negative reactions within a year. According to other reports, many are still sensitive after five years. The present study does not purport to include long-range follow-up studies. However, an appreciable number of the student nurses became insensitive to tuberculin less than six months after vaccination. At the end of one year, approximately 30 per cent of those who had experienced postvaccination induction of sensitivity to tuberculin had reverted to an insensitive state. This also has been the recent experience of Sams[4] in Japan.

This loss of induced sensitivity to tuberculoprotein is an important phase of any BCG program. No one knows just what the status of immunity is in a person who had an adequate local reaction to the vaccine, who experienced excellent sensitivity to tuberculin, and who then reverted to insensitivity within a year or so. Some clinical and experimental evidence suggests that allergy or sensitivity, as demonstrated by a positive cutaneous reaction to tuberculin, may not be completely parallel to immunity. Thus, some observers consider that some immunity to tuberculosis may persist in spite of loss of cutaneous sensitivity. This problem will be settled only by a long-term, controlled study. While awaiting data derived from such a study, other workers think that revaccination should be performed to maintain constant cutaneous sensitivity to tuberculin.

Finally, comment should be made regarding the most controversial aspects of BCG vaccination, namely its true efficacy and the extent to which a program of BCG vaccination should be applied. At one extreme is the view expressed by O'Brien,[2] in 1950: "Vaccination [BCG] should begin at birth in every country in the world. When individuals again become tuberculin negative they must be revaccinated." In concluding his 1950 report, O'Brien said: "Let us have the honesty to accept the futility of our present methods alone, and the courage to embark immediately on a program of extensive BCG vaccination, without which, tuberculosis will present the same problems a thousand years from now as it does today."

The conservative view recently was summarized by Myers,[1] who justifiably re-emphasized the fact that practically no valid statistical conclusions can be drawn from the world experience, in spite of the many millions of vaccinations that have been given. This absence of statistical validity stems from many factors, such as inadequate control groups, lack of comprehensive follow-up studies, the impossibility of environmental control of the persons who are vaccinated and variations in the vaccine itself. Although many isolated experiences have tended to demonstrate the efficacy of BCG vaccination, such experiences carry little scientific weight. Opponents of BCG vaccination also emphasize that artificial induction of sensitivity to tuberculin in large masses of people will negate the great efficacy of this test as an epidemiologic weapon in the eradication of tuberculosis.

Somewhere between the extreme and the conservative groups there should be room for those who consider justifiable the administration of BCG vaccine to tuberculin-negative persons, such as nurses, physicians and laboratory workers, who are intimately and continuously exposed to Mycobacterium tuberculosis.

Summary and Conclusions

A program of BCG vaccination was instituted in an effort to produce some immunity against tuberculosis in tuberculin-negative student nurses. The Rosenthal multiple-puncture method was used.

More than 90 per cent of persons vaccinated gave positive cutaneous reactions to tuberculin within six weeks after vaccination, but this induced sensitivity disappeared within one year in approximately 30 per cent of these students.

Only long-term, controlled studies will demonstrate whether any immunity to tuberculosis exists when the induced cutaneous sensitivity to tuberculin has disappeared.

Little valid statistical data can be adduced to prove the efficacy of BCG vaccination. However, many workers consider that its use is indicated for selected persons who are unduly exposed to tuberculosis.

References

1. Myers, J. A.: Present Status of Immunity in Tuberculosis. Minnesota Med. 37:529-534 (July) 1954.
2. O'Brien, E. J.: Honesty and Courage Can Eradicate Tuberculosis. J. Thoracic Surg. 20:1-12 (July) 1950.
3. Rosenthal, S. R.: The Multiple Puncture Method of BCG Vaccination. Am. Rev. Tuberc. 39:128-134 (Jan.) 1939.
4. Sams, C. F.: Experiences in Immunization Against Tuberculosis With BCG Vaccine in Japan. Am. J. Pub. Health. 44:903-908 (July) 1954.

THE MAYO BROTHERS

(Continued from Page 779)

Francis accelerated their specialization in the new surgery that was building so mightily on the work of Joseph Lister.

From the first, the two young doctors from the West made it their business to go wherever the latest and best in surgical methods was to be learned. Refining and adapting these methods with startling mastery in their own practice, they were soon drawing patients by the thousands from far and near. And doctors, too, for the exceptional skill of the Mayo brothers quickly made their operating rooms in St. Mary's Hospital one of the nation's outstanding schools of surgery.

Needing more and more help, William and Charles Mayo put into effect their father's precept: "No man is big enough to be independent of others." They chose the ablest men they could find to be their associates. Adding internists to surgeons and scientists to both, they gradually built up the Mayo Clinic, the world's first and still foremost example of co-operative group medicine in private practice. To complement the work of the Clinic, the Doctors Mayo in 1915 established and endowed the Mayo Foundation for Medical Education and Research, which, affiliated with the University of Minnesota, rapidly became an internationally renowned center for the training of medical specialists, scientists, and educators.

Acclaimed throughout the world for their humanity and their brotherhood, no less than for their contribution to medicine, Doctor Will and Doctor Charlie, as they were affectionately known to thousands, died within a few weeks of each other in the late spring of 1939. To honor the memory of these greatest of her sons, Minnesota has fittingly chosen to build a useful memorial that will render service to all mankind, for the achievements of William James and Charles Horace Mayo do not belong to Minnesota alone. They make a shining page in the record of all medicine and in the history of all America.

FRACTURES OF THE SPINAL COLUMN AND PELVIS

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FRACTURES of the spinal column and pelvis occur commonly in industrial and traffic accidents. Their presence may not be obvious, especially if there are associated serious injuries to the extremities, head, thorax or abdomen. In such a situation, especially if the patient is unconscious, it is almost mandatory to make roentgenograms of the spinal column and pelvis; this serves the twofold purpose of completing the accurate assessment of the patient's injuries and of completing the record in preparation for the almost inevitable insurance report or legal testimony.

Spinal Fractures

Injuries to the spinal column affected by osteoporosis, metastatic malignancy and other disease processes will not be considered here. Fractures of the normal spinal column are easily divided anatomically into three groups:

1. Fractures of transverse and spinous processes. These serve for the attachment of muscles, and fractures in these locations are usually due to sudden violent muscular contraction, or to direct blows. They are not serious and are commonly overtreated. Rest, administration of anodynes and other symptomatic treatment usually suffice.

2. Fractures of the vertebral bodies, which transmit weight.

3. Fractures of the laminas, articular processes and pedicles, which form the neural arch behind.

Fractures of the vertebral bodies constitute the most frequent spinal injury; at times there may be accompanying fracture of some of the posterior elements.[1] The injury is produced by forced flexion of the spinal column caused by such means as a fall from a height, a weight falling on the patient's back or shoulders, or the violence of an automobile or aircraft accident. Any portion of the spinal column may be involved, but the injury usually occurs at the junction of a movable with an immovable portion. Hence, the cervicothoracic and thoracolumbar junctions are the most frequently involved sites. Often two adjacent vertebrae are crushed, and at times one sees two injured segments, separated by one or more normal vertebrae.

Depending on the direction the flexion force takes, the injury may be wedging of one or more vertebrae, comminution of a vertebral body, or the more serious fracture-dislocation. As the force is applied, the pedicles act as a fulcrum and the fracture occurs in the cancellous bone just below the superior vertebral plate. At times the plate itself may be broken, forcing some of the disk material and plate into the substance of the body. There is always damage, to some extent, to the strong anterior and posterior longitudinal ligaments with a varying amount of hemorrhage.

Clinically, the patient usually complains of some degree of fairly well-localized back pain, with or without radiating pain. The intensity of the symptoms varies with the severity of the injury, and the interval since the injury. With the more severe injuries there may be a visible kyphos, but the most reliable sign is tenderness over the spinous process of the involved segment associated with some degree of spasm of the erector muscles. Retroperitoneal hemorrhage may give rise to signs resembling an acute surgical condition of the abdomen. If the spinal cord is intact but under pressure owing to hemorrhage, the deep reflexes are exaggerated and the muscles hypertonic. If the cord has been more seriously injured, there may be flaccid paralysis with absence of the deep reflexes.

Treatment starts when such an injury is suspected. The segments of the trunk above and below the site of injury are powerful levers and can be made to inflict more damage if the patient is improperly handled. Good roentgenograms should be made, both in the anteroposterior and lateral planes. After satisfactory films have been made and the site and extent of the injury determined, the patient should be placed flat on a

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From the Section of Orthopaedic Surgery, Mayo Clinic and Mayo Foundation. The Mayo Foundation is a part of the Graduate School of the University of Minnesota.

Part of the Symposium on Fractures of the Spine and Lower Extremities presented at the meeting of the Minnesota State Medical Association, Duluth, June 8, 1954.

hard bed, or on a gently curved Bradford frame and symptomatic treatment instituted. In these patients, paralytic ileus is apt to occur and it is advisable to treat them in bed for a few days before attempting any additional treatment.

These fractures always heal. In the case of minimal or mild compression without fracture of posterior elements, it is sufficient to fit the patient with a high back corset or Taylor brace and allow him to be ambulatory after the acute symptoms have subsided. With moderate or severe compression deformity, and in all cases in which there is fracture of posterior elements, more secure immobilization is desirable. Correction of the deformity may be difficult or impossible. Recumbency on a curved frame may accomplish some reduction. After acute symptoms have subsided and the danger of paralytic ileus has passed, forced hyperextension by suspension or on a fracture table may be tried. General anesthesia may be required. Correction is checked by means of lateral roentgenograms and is then maintained by the application of a snug plaster extending from the suprasternal notch to the symphysis pubis.

Regardless of the severity of the fracture, it is essential to start spinal hyperextension exercises as soon as possible, and to continue such exercises until the fracture has healed. Three to four months of immobilization should be sufficient. By that time roentgenograms should show sound bony healing, and if the erector spinae muscles have been kept strong by exercises, one should be able to discontinue external support. However, because of irregularity of the involved joint surfaces and the altered mechanics of the spinal column, some localized pain may persist. This is usually aggravated by exertion and relieved by rest. Spinal fusion is the procedure of choice in this situation and can be expected to give uniformly good results.

Pelvic Fractures

The most common injuries to the pelvis are those resulting from a crushing force, although muscular violence may produce avulsion fractures, and at times the sacrum and coccyx are injured by a fall to the sitting position.

The least serious injuries are isolated fractures of the pelvic ring; the fracture may be through, or adjacent to, the symphysis pubis, through any part of the innominate bone, or through the sacroiliac joint. Since there is only one fracture, gross displacement of the fractured parts cannot occur, and as a rule, only symptomatic treatment is required. Rest in bed until the acute symptoms subside, followed by guarded activities for 10 to 12 weeks, usually results in full recovery. Chronic pain may follow sacroiliac subluxation, and if symptoms warrant, a sacroiliac fusion may be indicated.

Lateral compression of sufficient force may result in a combined, or double, injury of the pubic portion of the pelvic ring. Both pubic rami may be fractured bilaterally, or fracture of both pubic rami on one side may be combined with separation of the pubic symphysis. Displacement is usually not great, and if these patients are nursed on their backs to avoid further lateral compression, a good result may be expected.

Compression of the pelvis in an anteroposterior plane results in a combined injury wherein one fracture is through the pubic segment of the pelvic ring and the other is somewhere through the iliac portion. One half of the pelvic girdle may be widely displaced so that there is obvious deformity and inequality in the length of the legs. Here some attempt to reduce the deformity should be made. As pointed out by Watson-Jones,[2] the disclocated pelvis is like a partly opened clam shell; when these patients are nursed on their backs gravity and the weight of the limb tend to increase the deformity. Reduction may be obtained at times simply by turning the patient on his uninjured side, pressing the iliac crests together. If there is upward displacement, it may be necessary to exert traction in the long axis of the leg on the injured side. Reduction should be maintained by applying a double plaster spica; it may be necessary to incorporate continuous skeletal traction. About three months will be required for sound bony healing to occur.

In double fractures of the pelvic ring, there may be serious injury to the urinary tract or genitalia, and this is always a more urgent problem than the fracture itself. The same force that produces the pubic portion of the injury may result in crushing or avulsion injuries to the external genitalia. Loose bone fragments or

(Continued on Page 837)

SYPHILIS OF THE STOMACH

Report of a Case

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IN 1914, I reported upon two cases[8] of hourglass stomach in patients with positive Wassermanns, and their improvement under anti-luetic treatment. My attention had been drawn to a similar report by Holitsch,[6] his patient having shown similar improvement based upon the combination of x-ray and Wassermann findings. In the year of my report, 1914, the literature available in the Surgeon General's Library, Washington, D.C., although offering much on syphilis in general, revealed relatively few reports dealing with syphilis of the stomach. In the intervening four decades since my report, seven other unreported cases have been cross-indexed in the charts and records of the Duluth Clinic. These cases covered patients showing intrinsic roentgen defects (chiefly at the pyloric end of the stomach) which, without exploration but with positive serology and under anti-luetic treatment, reportedly lost their distresses coincidental to the disappearance of the roentgen evidences of gastric mucosal distortion. This additional case is reported at this time because, despite the drop in the incidence of syphilis, it is still the great mimic that Osler designated it over a half century ago. Furthermore, since so great an effort is presently made to uncover earlier malignant lesions of the stomach—which well may occur in individuals with positive serology—it is well worth while to sharpen up our diagnostic sensitivity to the great value of therapy as a diagnostic agent.

Case Report

In July, 1953, M.S., a fifty-year-old man, came to Duluth from Canada, where his doctor had made a diagnosis of carcinoma at the pylorus. This working man had been ill for about two months, the trouble beginning with soreness across the upper abdomen with occasional bouts of diarrhea and vomiting. This latter occurred without special pain and seemed to be a part of over-distention of the stomach with simple regurgitation. The fact that his pain was increased immediately after eating testified to the probability of pyloric obstruction, which had been clearly shown on the previous gastro-intestinal fluoroscopies. He had been given the usual treatment on the basis of possible ulcer—frequent feedings and alkalies—but without any especial relief. Doctor H. G. Moehring, roentgenologist for the Duluth Clinic, agreed fully in the referring doctor's diagnosis and reported as follows: "Esophagus and stomach are negative; the gastric antrum shows a constant deformity and loss of flexibility and several filling defects surround a small palpable mass—all quite characteristic of carcinoma of the antrum of the stomach. The body and fundus appear uninvolved." He was admitted to St. Mary's Hospital, Duluth, Minnesota, for surgical care and observation. The following day, the report came back from the Clinic Laboratory that he had a four plus positive Kahn blood test.

The surgical consultant agreed that he would favor a period of preparation for surgery, with injections of penicillin, but felt that the x-ray evidence of malignancy called for immediate surgery. At exploration, it was reported "a rather soft, diffuse lesion of the pyloric end of the stomach. The stomach wall was not rigid, but there were eight or ten small nodes found in the gastric mesentery." When the resected portion of the stomach was carefully reviewed, no evidence of malignancy was found; neither was there a definite area of penetrating ulceration. On account of the very great interest centering around the prevalence of cancer of the stomach and the pressing need for early diagnosis and prompt removal of borderline lesions, numerous histologic sections were studied by our pathologist, Dr. William V. Kroll, who consulted with several other distinguished pathologists. The sum total arrived at was "a granulomatous lesion with moderate infiltration of round cells which is compatible with a syphilitic lesion." The patient made a satisfactory recovery and every assurance has been taken that his follow-up and treatment will be persistently and adequately checked.

The Literature and the Development of Reliable Diagnostic Criteria

Syphilis is the same disease it always has been down through the centuries of its recognition. Some diseases seem to fall easily into the field of the various specialties, but syphilis can copy so many entities that men in the various specialties, as well as in the special diagnostic fields, such as roentgenology and gastroscopy, have been able to add most notably to contributions stressing their techniques. Eustermann,[34] reporting on his large experience at the Mayo Clinic, in 1917 analyzed twenty-three cases and in 1931 summarized the total up to that date of eighty-nine cases. Fancher,[5] in 1935 added another case to the rapidly

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From the Department of Internal Medicine, The Duluth Clinic, Duluth, Minnesota.

Reported before the Minnesota Society of Internal Medicine, Saint Paul, Minnesota, October, 1953.

developing literature on the subject of gastric syphilis. Carey and Ylvisaker,[2] in 1938 reported further on the gastroscopic features indicating the field for this diagnostic procedure. Williams and Kimmelsteil[9] reported in 1940 and endeavored to outline "the syphilitic origin of this lesion may be adequately proved by gross and microscopic studies of removed tissue and the recovery of spirochaeta pallida." Unfortunately, the histologic evidence may be both fleeting and non-committal. The gross appearance of gummatous lesions is more apt to be distinctive than the microscopic sections prove to be. Palmer and Schindler,[7] in 1943, the former with his extensive and praiseworthy reporting on the relationship of peptic ulcer to gastric cancer, the latter with his fundamental work with the gastroscope, pointed out and discussed the "clinico-roentgenologic, gastroscopic and pathologic features of the gastric lesion considered to be ulcerated gummatous syphilis." You will note in this careful wording the avoidance of any dogmatic or definitive designation of syphilis as the clear-cut and incontrovertible diagnostic agent. This only re-emphasizes the need of using every aspect of any clinical diagnostic profile in arriving at methods of therapy for any disease. Berger[1] in 1950 restated what many reporters and groups had previously maintained, in terms of the elusive diagnosis of gastric syphilis, and outlined under the following headings "The criteria demanded for the diagnosis of gastric lues." He summarized: "a demonstrable radiographic defect, gastric symptoms, inability to alleviate symptoms or change radiographic appearance by conventional therapy, the symptomatic relief and/or disappearance of the roentgen defect after anti-luetic treatment," and finally concluded that penicillin most easily fulfilled the last criterion.

Comments and Inferences

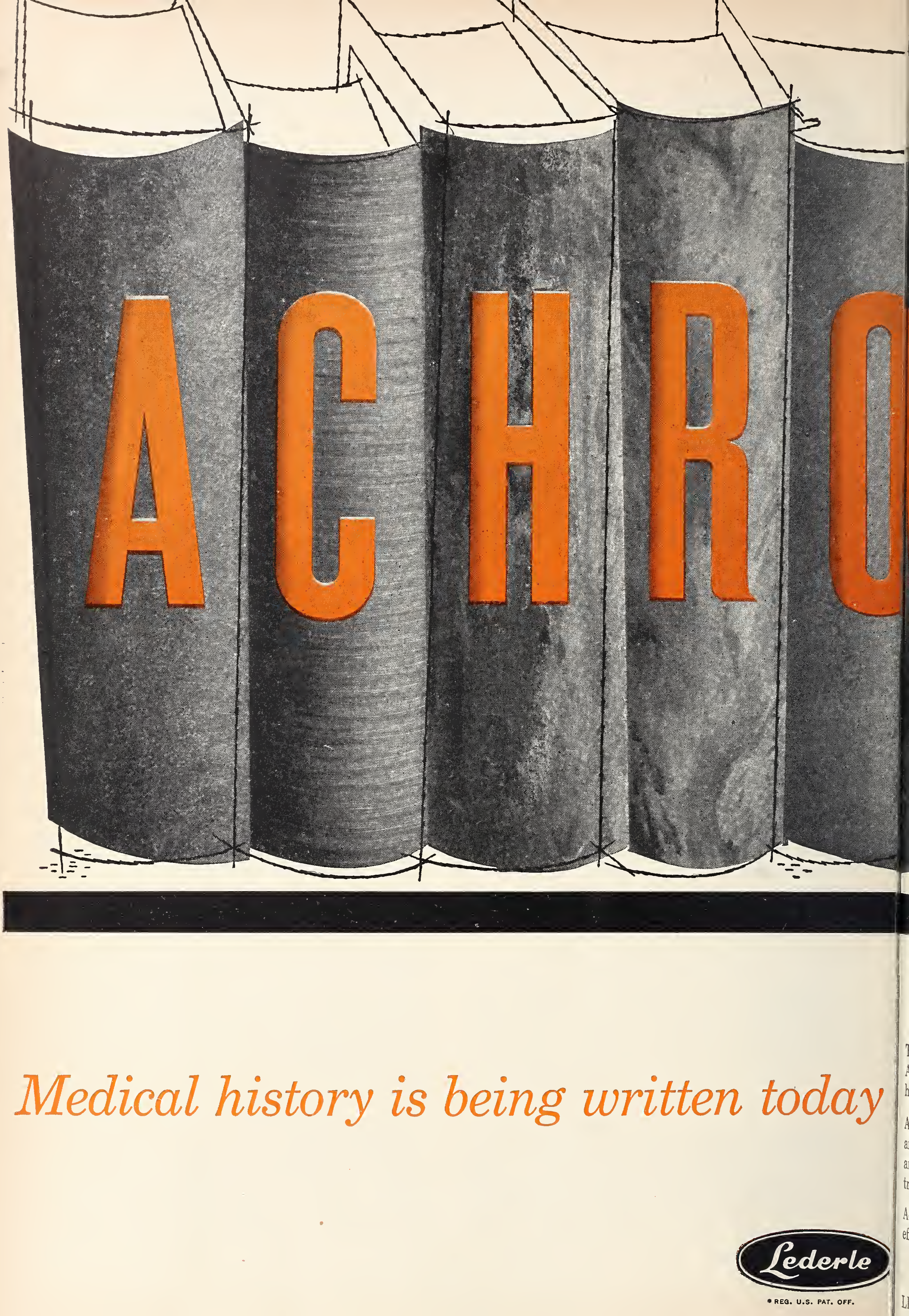
Recent graduates in medicine will never have the opportunity that some of us who have been in the field as students and practitioners for a half century have had to see syphilis at first hand. I am presently informed that there are few cases available for student teaching at our medical schools. In the span of one medical generation (thirty-five years), iodide of potash has shed most of the reputation it had, as the heavy metals, the arsphenamides and penicillin, so admirably fill the needs for therapeutic purposes and investigation. However, I well remember that the use of iodide or some form of mercury helped the older clinicians to resolve the diagnosis in not a few baffling chronic disorders.

From the standpoint of pinning down the diagnosis of a gummatous syphilitic lesion, neither the x-ray nor the microscope are so discriminating as to eliminate completely all diagnostic doubts. This circumstance was amply illustrated in the intensive studies of many sections removed at operation in the instance of my report. In like manner, the roentgen evidence was incomplete, since recheck after a period of penicillin therapy was not made.

It seems fair to urge the younger surgeons and roentgenologists to be alert, despite the rapid disappearance of syphilis, to the possibility that a suggested lesion involving the gastric mucosa might be given therapeutic trial. Obviously, this is especially true if there is serological evidence of syphilis, and it must be freely admitted that patients with old syphilitic disease may also fall victim to gastric cancer. It follows that the Wassermann screening of all patients, and especially those with chronic gastrointestinal disorders, is still, despite the very small "take," a worthwhile procedure.

References

1. Berger, Herbert: A new diagnostic criterion for gastric syphilis. Gastroenterology, 14:147, 1950.
2. Carey, J. B., and Ylvisaker, R. S.: Gastroscopic observations of syphilis of the stomach. Ann. Int. Med., 12:544, 1938.
3. Eusterman, Geo. B.: Gastric syphilis: observations based on ninety-three cases. J.A.M.A., 96:173-179, 1931.
4. Eusterman, Geo. B.: Syphilis of the stomach, a clinical and roentgen study with a report of twenty-three cases. Am. J. M. Sc., 153:21-39, 1917.
5. Fancher, Paul S.: Syphilis of stomach. Ann. Int. Med., 35:240, 1935.
6. Holitsch, R.: Roentgen findings in a case of syphilitic hour-glass stomach. Tr. German Roentg. Soc. Congress IX, p. 57, 1913.
7. Palmer, Walter Lincoln, Schindler, Rudolph, Templeton. Frederick E., and Humphreys, Eleanor M.: Syphilis of stomach; a case report. Ann. Int. Med., 18:393-406, 1943.
8. Tuohy, E. L.: Luetic contractures of stomach. A report of two cases of hour-glass stomach with positive Wassermann reactions and improvement under antispecific treatment. Interstate M. J. (St. Louis), 21:1036-45, 1914.
9. Williams, Carrington & Kimmelsteil, Paul: Syphilis of the stomach. J.A.M.A., 115:578-582 (Aug. 24) 1940.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STROMAL ENDOMETRIOSIS

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Northfield, Minnesota

THERE have been not more than approximately fifty cases of stromal endometriosis reported in literature. In 1908, Doran and Lochyer first described two cases under the title, "Two Cases of Uterine Fibroids Showing Peritheliomatous Changes; Long Immunity from Recurrence After Operation." These are probably the first two cases fully described which, according to Park, from the illustrations and descriptions belong in this classification. However, until the reports of Henderson (1942) and Goodall (1940 and 1942), this condition was neither fully described nor given any semblance of classification. Park's review, in 1949, is the most comprehensive to date. The only other cases reported since 1949 were: a report of a case by Kettal, et al in 1949, which was not included in Park's study; one by Lash and Lash in 1951; and one by Wheelock and Strand in 1953.

This is a disease primarily from the ages of thirty-five to fifty, but one case is reported in the age group of twenty-one to twenty-five and one in the sixty-one to sixty-four group.

There is usually a long period of infertility. The symptoms and signs based on Park's review[5] of forty-three cases are: menorrhagia fourteen, metrorrhagia four, vaginal bleeding (non-descriptive) eleven, post-menopausal bleeding three, dysmenorrhea four, abdominal or pelvic pain eight, abdominal swelling six, loss of weight five, and various combinations of these. There is no constant symptom complex. Six patients were menstruating normally. Seven patients were anemic. The main and constant finding on clinical examination was an enlargement of the uterus, usually symmetrical.

The usual finding at operation was a symmetrically enlarged uterus. In addition some cases revealed as follows:[5] (1) Visible or palpable spread beyond the uterine boundaries in twelve cases, (a) One case with multiple tissue nodules on the pelvic peritoneum, (b) Eleven cases with no separate nodules; (2) One case with a localized nodule on the peritoneal surface of the uterus. The conditions included, (a) Ectopic endometriosis was present in seven cases—six in ovary and one beneath the uterine peritoneum, (b) Adenomyosis was reported in four cases.

The chief pathological feature is of neoplastic tissue, more commonly in one wall, but it may be generalized. Grossly this tissue is a dirty white or yellowish color (Goodall emphasized these yellowish areas of crumbly material). They vary in consistency. It may be well localized, but more typically, in countless rubbery bands throughout the myometrium as though filling the channels of a sponge. On the cut surface it has a "rough towel" appearance. In some areas the neoplastic tissue blends intimately with the myometrium. These cords may extend into the broad ligaments and in nineteen cases formed polypoid growths extending into the uterine cavity.

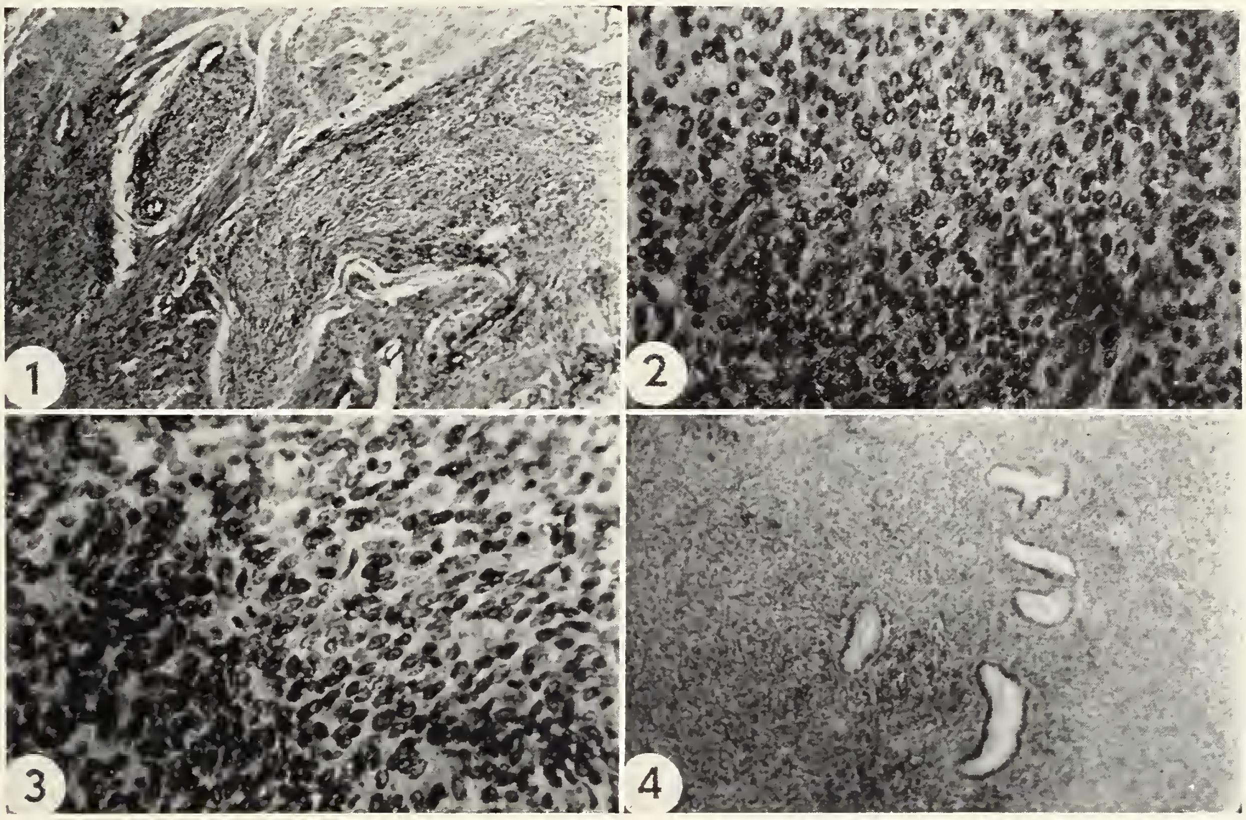
Microscopically the tissue is composed of a solid mass of round, or spindle-shaped cells. The nuclei are prominent. The actual cell margins are blended or overlapped with adjacent cells. Mitosis is rare. The cells resemble the stromal cells of a midcyclic endometrium. There is some differentiation to a branching trabeculated pattern and, in some, a tendency to early tubal or glandular formation. In the majority of cases the new growth merged with the endometrium, but in some cases there was no such continuity.

The course and prognosis varies according to the stage of the disease at the time of diagnosis, as there is no uniform pattern. Of Park's study of forty-three cases, eight died as a direct result of the disease; three died from unrelated causes; twenty-two were alive at the time their case histories were published; and thirteen had no follow-up. The survival rate was up to seventeen years from the time of the original operation. The onset of menopause, natural or artificial, had no influence on the outcome. Cell polymorphism and mitotic activity may indicate a more rapid progress, but a uniform and inactive appearance is no guarantee that recurrence will not occur. In this series there were seven cases with metastasis. In two of Goodall's it was after uremia had developed. The main risk is local recurrence and spread.

The treatment is very unsatisfactory. In some of the early cases there was some response to x-ray therapy. As menopause has no effect on the condition, sterilization seems to be of no value.

Regardless of the differences of opinions of pathologists, the knowledge that by far the majority of these cases will have a long life expectancy is of extreme importance from the patient's standpoint and to our handling of their cases.

The pathogenesis is not established. The tumor may arise in the uterine wall with no direct connection to the endometrium. Gruenwold and Park believe that adenomyosis, stromatous endometriosis, carcinosarcoma of the uterus and other occasional tumors of the uterus should be grouped together:[5] "These are all apparent tissue maldevelopments arising from unrecognized and unrecognizable precoursor cells within the uterine wall; that they have a certain appearance depending on the type and degree of differentiation; that microscopically one or the other of these may show none, or some, or many of the features especially found to be associated with a liability to metastasis; and that from past experience it will be known which of the pathological findings are likely to be associated with what clinical behavior." Gruenwold theorizes that all cells have the innate ability or propensity to differentiate or change in response to certain or definite stimuli. He does not agree with the theory of embryonic rests.



Figs. 1-4. Photomicrographs from sections of tissue removed in 1938 by Dr. E. T. Bell. (1) Low magnification reveals characteristic infiltrating strands of stromal tissue. (2 and 3) Stromal tissue under higher magnification shows the large vaculated nuclei, the rare mitotic figures and the overlapping spindle-shaped cells. (4) Section revealing some remnants of endometrial glands in the stromal new growth which reveals the true character of this as a stromal endometriosis.

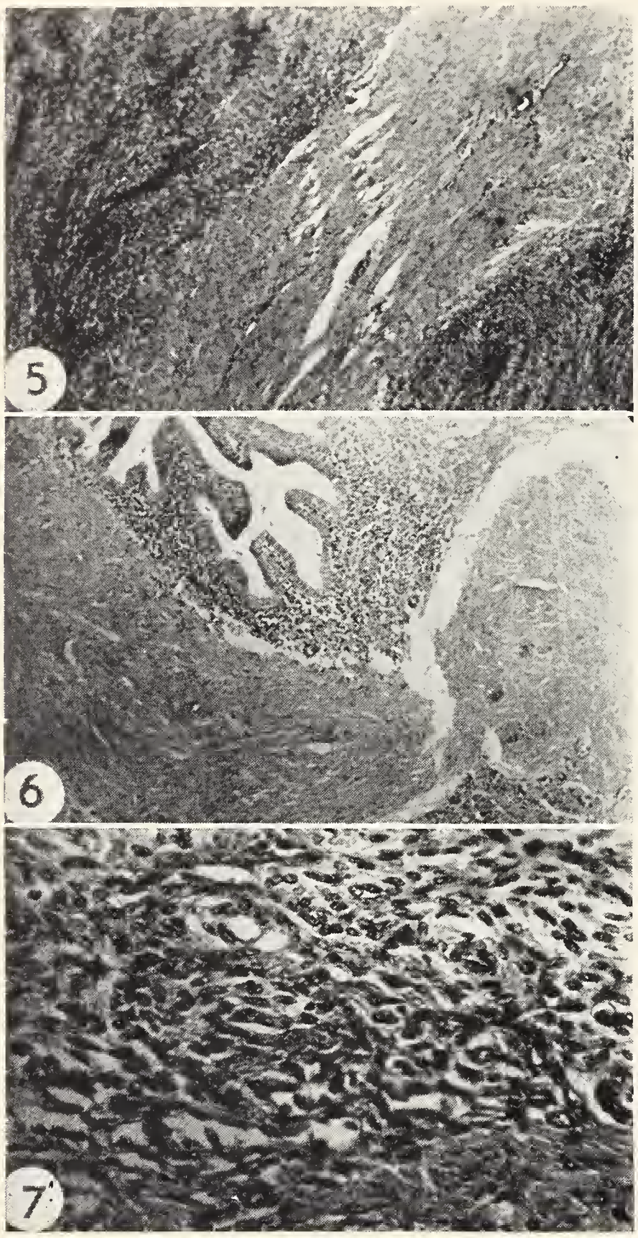
In this light, stromatous endometriosis need not be defined as either malignant or non-malignant. Park suggests a better term as "stromatoid new growth of the uterine wall" or "stromatoid mural sarcoma." The diagnosis of this is by microscopic section.

Case History

Mrs. B., aged sixty-three, in April, 1952, complained of minimal but rather persistent vaginal bleeding which had lasted for a period of two weeks.

Her past history outside of the generative tract was essentially negative except for removal of cataracts—one in 1943, and one in 1946. Severe hypertension was found at the time of surgery in 1943, and still is a problem, but is asymptomatic. Her history relevant to the generative tract was normal until 1938. She married at the age of thirty. She had only one normal pregnancy at the age of thirty-five. There were no more children and no contraceptive methods were used.

In 1937, at the age of forty-eight she began to bleed profusely with clots for a period of six months. She became quite anemic and run-down and was hospitalized for two weeks prior to a pelvic exploratory operation by Dr. B. J. Gallagher of Waseca in 1938. Dr. Gallagher described his findings—a number of white cauliflower-like growths over the pelvic viscera which were removed. The uterus was normal in size and appearance and was left intact. The tissue was examined by Dr. E. T. Bell and he returned the diagnosis of sarcoma. Mrs. B.'s postoperative course was uneventful. She had only one episode of vaginal bleeding six months later, which was extremely profuse, lasting only two to three days.



Figs. 5-7. Photomicrographs from the tissue removed in 1952. (5 and 6) Low magnification, with 5 revealing again the stromal infiltration; 6 the remnants of endometrial glands. (7) High magnification showing the characteristic cells and nuclei as in 2 and 3.

She had never had any pelvic pain or discomfort and there is none to date. Her examination was normal except for dilated pupils, blood pressure of 286/138, and the pelvic findings. The cervix was normal; there were hard fibrotic cauliflower-like growths high in the vaginal vault varying in size from a few millimeters to 2 or 3 cm. (These were noted in Kettal's case.[3]) The uterus was about four times normal size and regular in outline. The pelvic organs were completely frozen. The laboratory tests were normal.

In March, 1952, a biopsy of the vaginal growths and curettement of the uterus was performed. Nothing was obtained on curettement except a little necrotic and purulent appearing material. The pathological report of the vaginal lesions was simply of scar tissue and nothing of note from the curettements.

She was sent to Dr. Leonard Lang of Minneapolis for consultation. Surgery was recommended with a diagnosis of probable sarcoma of the ovary. Surgery was performed by Dr. Lang in May, 1952. The uterus was symmetrically enlarged. The pelvic viscera laterally was a fused mass and the elements were indefinable.

An attempt was made to remove the uterus. This was impossible as it was very firmly fixed to the sacrum, the bladder and pelvic walls laterally. There were none of the growths found by Dr. Gallagher present. About one-third of the fundus was removed. Her postoperative course was uneventful. There has been no bleeding or pain since this surgery.

The tissue was examined by Dr. John I. Coe, pathologist of the Minneapolis General Hospital, who made a microscopic diagnosis of stromal endometriosis. The sections of tissue removed in 1938, were reviewed with the same findings.

There has been only one other case found in the pathology laboratory at the University of Minnesota and this apparently has not been reported in the literature.

Acknowledgment

The photomicrographs in the paper were prepared by Dr. John I. Coe, to whom I wish to express my thanks.

References

1. Goodall, J. R.: A Study of Endometriosis. First Edition. Philadelphia: Lippincott, 1943.
2. Henderson, D. Nelson: Endolymphatic stromal myosis. Am. J. Obst. & Gynec., 52:1000, 1946.
3. Kettal, William C., Lee, James G., and Randall, John H. Stromal endometriosis. Journal-Lancet, 69:261, 1949.
4. Lash, A. F. and Lash, J. R. Stromal myosis. Am. J. Obst. & Gynec., 62:1163-1166 (Nov.) 1951.
5. Park, W. Wallace. Stromatus endometriosis. J. Obst. & Gynec. Brit. Emp., 56:759-776, 1949.
6. Wheelock, M. C. and Strand, C. M.: Am. J. Obst. & Gynec., 2:284, 1953.

CAT-SCRATCH DISEASE ASSOCIATED WITH ENCEPHALITIS AND HERPES ZOSTER

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CAT-SCRATCH disease has only recently been recognized as a separate entity. Even though Foshay made some unpublished observations on this condition in 1932, it was not until 1950 when Debré[3] in France reported the disease under the term "maladie des griffes de chat." Since then the number of reported cases has increased rapidly and a variety of clinical pictures has been recorded.[1,2,4,8,9]

Here is the summary of the course of a typical case. There is usually a history of a cat scratch or bite which causes a slowly healing primary lesion. The regional lymph nodes become swollen, tender and may suppurate within a period of four weeks. There is no lymphangitis and cultures of the pus obtained from suppurative lymph nodes are consistently sterile. Systemic symptoms usually coincide with the appearance of regional lymphadenopathy and consist of fever, chills, malaise, nausea and generalized aching. In about 10 per cent of the cases a maculo-papular rash has been observed; this is not necessarily limited to the part of the body afflicted with the primary lesion. The disease is self limited and the symptoms gradually subside over a period of one to five weeks. The disappearance of the lymphadenopathy is rather slow and may require several months. The more unusual manifestations of the disease include conjunctivitis (Parinaud's oculoglandular syndrome) encephalitis, erythema nodosum, granulomatous bone lesions, pulmonary infiltrates with hilar adenopathy, pharyngeal and mesenteric adenitis.[2,9] There are no specific differentiating hematological laboratory tests. The leukocyte count and the sedimentation rate may be normal or slightly elevated, and the differential count is usually normal except for neutrophilia which occurs occasionally. The diagnosis is based upon an intradermal test with an antigen prepared from the pus of suppurative nodes. A positive Mantoux type of reaction occurs after forty-eight to seventy-two hours.

To the author's knowledge, the present case of cat-scratch disease is the first reported in the State of Minnesota. The opportunity for careful observation of the patient from the onset of symptoms, the rather extensive laboratory studies, specifically of serological nature, and the unusual association with herpes zoster justify its publication.

Case Report

The patient, a twenty-six-year-old white man, was in good health until May 29, 1954, when he felt a node behind his left ear. On the subsequent day, the node became tender and the patient experienced nausea and malaise. He was admitted to the University of Minnesota Hospitals on May 31, where he was found to have a chain of large and tender lymph nodes along the posterior cervical muscles and the superior border of the trapezius on the left. With the exception of one small non-tender node in the right axilla, which was present at the time of the onset of symptoms, no other lymph nodes could be felt. The tip of the spleen was palpable on deep inspiration. The unusual and unilateral location of the enlarged nodes suggested a scalp lesion which was found in the form of a tender elevated papule with a central incrustation in the left parietal region. The patient recalled that this was the result of a cat scratch approximately three weeks earlier.

Cat-scratch disease was suspected and later confirmed by a positive skin test. Generalized muscle aching started on June 1, associated with a remittent type of fever. There was also a maculo-papular rash over the "belt zone" between the rib margin and the iliac crest on the right. The patient had noticed some paresthesias in this region for approximately five days. One June 2 the appearance of blisters led to the diagnosis of herpes zoster. On June 3 the patient developed a severe headache necessitating the administration of narcotics. The only positive neurological finding at this time was nuchal rigidity. A spinal puncture revealed an elevated pressure of 22 cm. of water, 37 cells per cu. mm. with a mononuclear: neutrophil ratio of 10:4. Spinal fluid proteins were 30 mgm. per cent, glucose 47 mgm. per cent and chlorides 127 mgm. per cent. Blood and spinal fluid cultures were sterile. Inoculation of spinal fluid intracerebrally into young mice and into tissue cultures for virus studies were negative. Tetracycline (Achromycin) was started on June 3 in a daily oral dose of 1500 mgm. The temperature fell to normal, the symptoms subsided rapidly, and the patient was discharged on June 8. The lymph nodes disappeared very slowly and were still palpable on the fortieth day after hospital discharge. The clinical course and the hematological findings are illustrated in Figure 1.

Except for a leukopenia early in the course of the disease and a temporary lymphocytosis during recovery,

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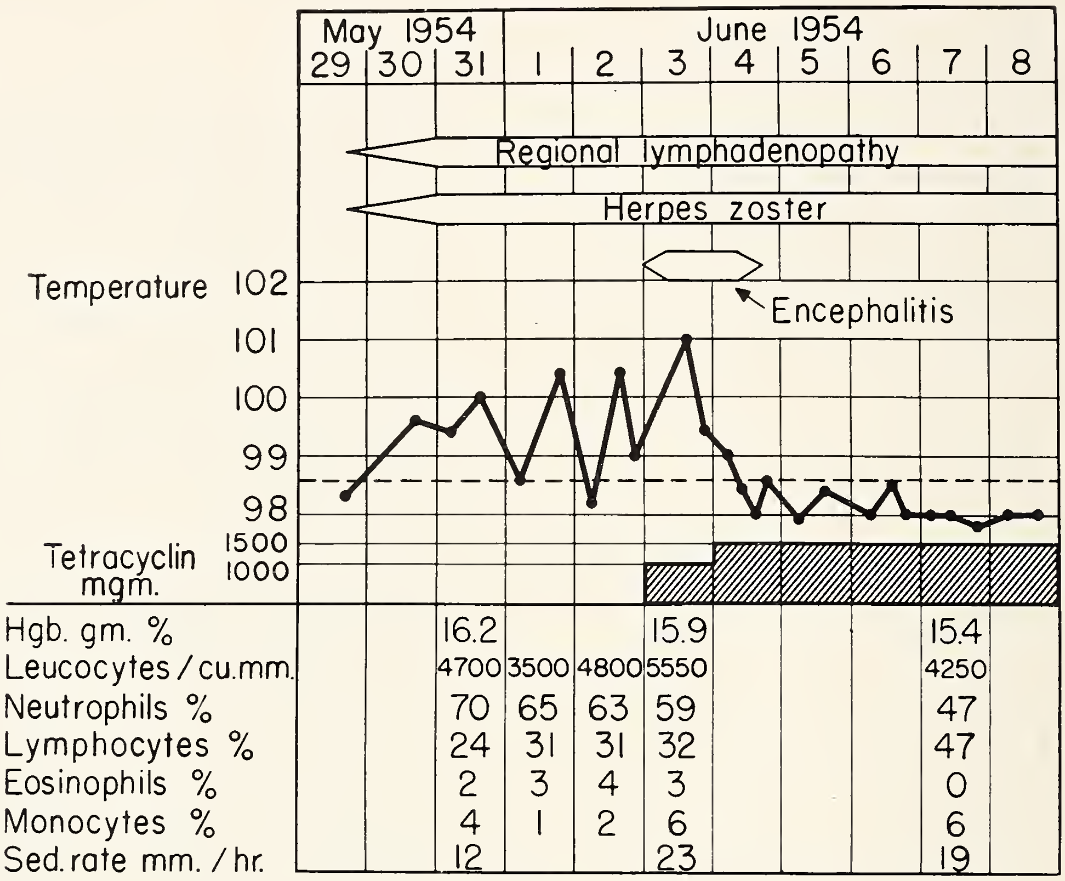
all tests were consistently normal. Urinalysis was negative. Repeated heterophile antibody titer determinations varied between 0 and 1:14. No cold agglutinins could be demonstrated. There was no agglutination titer for brucella, pasteurella and proteus OX-2. The agglutination titer for proteus OX-19 was 1:40. Complement fixation tests for psittacosis and lymphogranuloma venereum were negative. Blood specimens collected on the first, twelfth, twenty-second and forty-fifth day after onset of symptoms were checked for antibodies against western equine encephalitis, St. Louis encephalitis, mumps and lymphocytic choriomeningitis. There was no rise in antibody titer. Liver function tests: total bilirubin, 0.2 mgm. per cent, cephalin cholesterol flocculation negative, total lipids 17 units, thymol turbidity 2 units, total serum proteins 7.7 gm. percent, albumin 4.5 gm. per cent, globulin 3.2 gm. per cent. Follow-up observation over a period of two and one-half months showed that the patient had recovered completely.



	May 29	30	31	June 1	2	3	4	5	6	7	8
Hgb. gm. %			16.2			15.9				15.4	
Leucocytes / cu.mm.			4700	3500	4800	5550				4250	
Neutrophils %			70	65	63	59				47	
Lymphocytes %			24	31	31	32				47	
Eosinophils %			2	3	4	3				0	
Monocytes %			4	1	2	6				6	
Sed. rate mm. / hr.			12			23				19	

Fig. 1.

Past History.—One year prior to the present illness the patient underwent a thoracotomy for removal of several mediastinal nodes which caused pressure symptoms on the esophagus. Histological studies of the nodes revealed a granulomatous structure with central necrosis. The absence of acid-fast bacilli in the nodes and the persistently negative Mantoux test led to the presumptive diagnosis of the recently described mediastinal granuloma of unknown etiology.[5]

Discussion.—It is probable that cat-scratch disease is more frequent than presently recognized. The relatively recent description of the disease and the fact that many physicians are unaware of its existence and clinical picture account for the alleged rarity. Practically any condition with lymph node involvement should be included in the differential diagnosis: lymphoma, leukemia, infectious mononucleosis, pyogenous lymphadenopathy, rat bite fever, sporotrichosis, brucellosis, tularemia, tuberculosis, syphilis, lymphogranuloma venereum, et cetera. The importance of the skin test should be stressed because it may save the patient a lymph node biopsy which is usually nondiagnostic in this condition. The histological picture of an affected regional node is that of chronic inflammation with or without central necrosis. Epithelioid cells are usually found, and no bacteria can be detected on either sections or cultures.[10] According to Daniels,[2] the skin test has a high degree of specificity; among several hundred normal individuals only three had a positive skin test, probably as a result of past infection. In the case reported here two other members of the household had been scratched by the same cat but had never had clinical symptoms. Both were skin tested and found to be negative.

The etiology of cat-scratch disease is not definitely known. Current thinking favors a virus as the etiological factor. This is largely based on the work of Mollaret[6,7] who reported successful transmission of the infection to human beings and monkeys. He also demonstrated the presence of inclusion bodies with specific staining properties in the cells of the primary lesion and involved lymph nodes. Cats which have been a proven source of the disease are usually asymptomatic and have a negative skin test. It is impossible to infect cats with material obtained from suppurative lymph nodes. These observations support the idea that the cat is only a vector of the pathogenic agent.

A review of the symptomatology has been given in the introduction of this paper. The incidence of encephalitis is very low.[8] Its occurrence in this case may be related to the site of inoculation, the scalp. The association of cat-scratch disease with herpes zoster has not been reported thus far. Even though this may represent a coincidence, one should not disregard the fact that herpes zoster is a frequent complication of diseases involving lymph nodes; hence a direct relation between cat-scratch fever and herpes zoster is quite possible. The value of antibiotic therapy in such a self limited disease is rather difficult to assess. Daniels feels that oxytetracycline (Terramycin), chlortetracycline (Aureomycin), and chloramphenicol (Chloromycetin) may be beneficial. In the present case tetracycline (Achromycin) was started at a stage when the patient was most critically ill. The prompt recovery and the completely afebrile course after institution of therapy suggest that tetracycline was of value.

In reviewing the history of this case, one may wonder if the enlarged mediastinal nodes removed in 1953 were in some way related to the present acute illness. This is highly unlikely, because the two diseases had a completely different course: the first one was a chronic condition, the second one was acute. The patient had completely recovered from his first disease when he was scratched by a cat, and the course of the acute febrile illness was identical with the previously described cases of cat-scratch disease.

Summary

1. A case of cat-scratch disease associated with encephalitis and herpes zoster is presented.

2. Etiology, typical and atypical clinical features, diagnostic tests, prognosis, and therapy of this recently discovered disease entity are reviewed.

Acknowledgment

The author is indebted to Dr. W. W. Spink for the diagnostic antigen obtained from Dr. Mollaret in Paris.

References

1. Cassady, J. V., and Culbertson, C. S.: Cat-scratch disease and Parinaud's oculoglandular syndrome, A.M.A. Arch. Ophth., 50:68, 1953.
2. Daniels, W. B., and MacMurray, F. G.: Cat-scratch disease; a report of one hundred sixty cases. J.A.M.A., 154:1241, 1954.
3. Debré, R.; Lamy, M.; Jammet, M. L.; Costil, L.; Mozziconacci, P.: La maladie des griffes de chat. Bull. et mém. Soc. méd. hôp. Paris, 66:76, 1950.
4. Greer, W. E. R., and Keefer, C. S.: Cat-scratch fever: A disease entity. New England J. Med., 244:545, 1951.
5. Kunkel, W. M. Jr., Clagett, O. T., and McDonald, J. R.: Mediastinal granuloma. J. Thorac. Surg., 27:565, 1954.
6. Mollaret, P.; Reilly, J.; Bastin, R., and Tournier, P.: La découverte du virus de la lymphoréticulose bénigne d'inoculation: I. Caractérisation sérologique et immunologique. Presse méd., 59:681, 1951.
7. Mollaret, P.; Reilly, J.; Bastin, R., and Tournier, P.: La découverte du virus de la lymphoréticulose bénigne d'inoculation: II. Inoculation expérimentale au singe et colorations. Presse méd., 59:701, 1951.
8. Thompson, T. E.: Cat-scratch encephalitis. Ann. Int. Med., 39:1946, 1953.
9. Usteri, C.; Wegmann, T., and Hedinger, C.: Über atypische Formen der sogenannten Katzenkratzkrankheit, einer benignen Virus-Lymphadenitis. Schweiz. med. Wchnschr., 82:1287, 1952.
10. Winship, T.: Pathologic changes in so-called cat-scratch fever. Am. J. Clin. Path., 23:1012, 1953.

NAME CHANGE FOR AMA COUNCIL

"Only the name has been changed . . ." At its last meeting, AMA's Board of Trustees approved a change in name only for the Council on National Emergency Medical Service which from now on will be known as the Council on National Defense. As formerly, the Council assists with national and state disaster relief plans and co-ordinates the work of state emergency medical service committees.

It helps physicians prepare themselves for the management and care of casualties which might result from atomic, biological or chemical attacks. Also, the Council collects and disseminates civil defense and disaster relief information for both military and civilian use and assists governmental agencies, medical and hospital groups and local disaster relief groups in planning adequate civil defense programs. The Council works closely with the surgeons general of the armed forces and the Armed Forces Medical Policy Council of the Department of Defense in formulating plans and procedures for improving the medical care for members of the armed forces.—*AMA News Notes,* August, 1954.

Seminar

PITUITARY INSUFFICIENCY

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It has long been recognized that destruction of the anterior pituitary gland produces a fairly typical disease pattern in man. Simmonds first demonstrated this in 1914. In recent years, however, this condition has been exhaustively studied and is now more thoroughly understood. This paper is intended to be a summary of the important practical aspects of pituitary insufficiency.

The terminology in this disease deserves clarification since the literature has perpetuated some misleading designations. Because cachexia is not characteristic of the syndrome, the terms "pituitary cachexia" and "Simmonds' cachexia" should be abandoned. The term "panhypopituitarism" is applicable only if all the functions of the pituitary are disturbed, and this is not always the case. Because of the extensive research of Sheehan,[15,17] on postpartum necrosis of the anterior pituitary, this entity is now often referred to as "Sheehan's syndrome."

Etiology

Any process resulting in destruction of the anterior pituitary gland may cause the syndrome of pituitary insufficiency. Sheehan and Summers[17] in an extensive review of this subject separated the important etiological factors into four groups:

1. *Healed Postpartum Necrosis.*—When severe circulatory collapse occurs after delivery the anterior pituitary gland suffers ischemic necrosis. The severity of the general collapse determines the extent of necrosis and the subsequent scar. This lesion accounted for sixty-two of the ninety-five cases reported by Sheehan and Summers. Sheehan[15] did not consider pituitary insufficiency due to postpartum necrosis to be extremely rare and estimated that in each 10,000 of population there are about two instances of severe disease and seven instances of lesser severity. Destruction of more than half of the anterior lobe is probably required before the lesion is of any functional significance. This is true regardless of the cause of the destruction. In addition it is felt that total destruction of the gland probably never occurs from any cause.

2. *Other Chronic Fibroid Lesions.*—This group comprises a large proportion of the clinically important cases of pituitary insufficiency. Some of these lesions are apparently due to healed tuberculosis of the pituitary and others to healed gummatous necrosis of the pituitary. Oelbaum[10] reviewed the literature concerning syphilis as a cause of hypopituitarism and reported two cases in males with typical clinical and laboratory findings. In many lesions, however, the nature of the original change is not apparent, for all that remains is a scarred, fibrotic or atrophic pituitary. Cerebral arteriosclerosis may play a part in some of these cases. Trauma to the head apparently caused the lesion in two accepted cases.

3. *Hypophysectomy.*—This is not important clinically as a cause of pituitary insufficiency, although two cases have been recorded.

4. *Cysts and Tumors.*—These lesions result in pituitary insufficiency when they have destroyed sufficient functioning pituitary tissue. They may cloud the syndrome, however, by producing various pressure effects in addition to the endocrine deficiency. By far the most frequent lesion in this group is pituitary adenoma. The chromophobe adenoma comprises 70 to 80 per cent of all pituitary adenomas. It is nonsecretory and therefore produces its effects entirely by pressure. Clinically, the sella turcica is ballooned in 86 to 94 per cent of cases and visual field defects are noted in almost all cases. Usually this is manifested by bitemporal hemianopsia. Calcification in these tumors is unusual, occurring in 1 to 2 per cent.[12]

It should be emphasized that destruction of the anterior pituitary must be present for months or even years before the typical clinical picture develops. Acute necrosis of the anterior pituitary is not associated with obvious signs of hypopituitarism. This necrosis heals in a few weeks at most and a scarred pituitary remains. Pituitary insufficiency will then develop, its severity related to the amount of the gland that was destroyed.

Clinical Picture

The clinical pattern of this disease varies mostly with the degree of destruction of the anterior pituitary.[9,11,17,18] Vague symptoms probably reflect minimal damage while the full-blown picture usually means nearly complete destruction.

The symptoms and signs in this disease are the result of lack of hormones from the target glands of the pituitary. These target glands may be involved unequally so that adrenal function may remain adequate while thyroid and gonadal activity is lost. Likewise any other combination of involvement may occur.

The "classic" patient is a woman who has a severe postpartum hemorrhage and nearly dies in shock. Following this there is failure of lactation, which is the first sign. The only other early sign is that the pubic hair, having been shaved for delivery, grows back only slowly and scantily. Chronic ill-health then slowly develops. There is amenorrhea, atrophy of the gonads and genital tract, loss of libido, almost complete loss of axillary and pubic hair. and absence of normal cutaneous pigment, particularly obvious around the nipples. The facies is characteristic, with thinning of the eyebrows and pallor of the cheeks. There is absence of sweating of the skin, as well as loss of normal greasiness of the axillae. The patient has increased sensitivity to cold, is physically weak, lacks ambition or interest, is often mentally slow, and usually talks with slow, soft speech.

Several of these features deserve further comment. Sheehan and Summers[17] made many of these observations in their study of ninety-five proved cases of pituitary insufficiency. It should be noted, however, that these were all examples of severe degrees of pituitary insufficiency.

Sex Function.—Complete and permanent loss of sex function is an invariable consequence, whatever the origin of the pituitary lesion. In women menstruation always ceases. Only seven men were included in this series; all of them were impotent and had no libido.

Hair.—Loss of both pubic and axillary hair appears to be of considerable diagnostic importance whereas loss of axillary hair alone is not significant. Axillary hair is lost in a few months but pubic hair may persist for a year or two. The eyebrows tend to be thinned mostly at their lateral margins, as in myxedema. About half the patients have thinning of scalp hair.

Skin.—Nearly always the skin is dry, smooth and soft, but sometimes it is scaly on the limbs. Striking pallor is often present, having been described as waxy-white or like alabaster. In some a lemon-yellow tinge is noted. Although anemia is usually present, it is not so severe as the degree of pallor would indicate.[9] Much of the pallor is due to a true interference with production of melanin.

Facies.—The characteristic facial appearance is emphasized by most authors.[9,11,17,21] There is lack of animation, giving an expression suggestive of a person asleep. Slight puffiness is commonly present, with the pallor and thinning of the eyebrows completing the picture. This is at least partly suggestive of myxedema, and often these patients are mistakenly considered to have primary myxedema. Progeria is infrequently described, although it was one of the features in Simmonds' early writings.

Cardiovascular System.—The heart is usually small in contrast to that usually seen in myxedema. The electrocardiogram resembles that seen in myxedema, with low voltage and nonspecific T-wave changes. Treatment with thyroid may produce a return to normal. The mean systolic blood pressure in the series reported by Sheehan and Summers[17] was 108 mm. of mercury. However, the pressure was found to be extremely labile.

General Symptoms.—The pronounced sensitivity to cold is emphasized. Mental torpor, loss of initiative and a state of placid inertia overtake these patients. Speech is slow, quiet, and monotonous. Rheumatism of the knee joints is frequent.[9,11,17]

It should be pointed out again that the clinical features already listed are those of full-blown panhypopituitarism. Gradations in severity may occur and a selective defect may be seen, one target gland being affected more than another. The clinical features are certainly not completely uniform.[9,21]

The duration of this disease can be determined quite well from a study of postpartum necrosis, since it is possible to date the onset of this condition exactly. Most of the patients live from five to fifteen years but some survive for as long as 40 years.[17]

These patients appear to have an undue susceptibility to tuberculosis, and Sheehan[15] postulated that this may be related to endocrine insufficiency.

A grave danger facing patients with hypopituitarism is the development of a particular type of coma that is often fatal. This is often preceded by a short period of anorexia or vomiting, followed by a confusional state. The patient then becomes stuperous and lapses into unconsciousness in a day or two. Localized twitchings or generalized convulsions may occur. The blood pressure may be normal or low and the pulse is commonly feeble. Respirations are shallow and the temperature is usually low, between 95 degrees and 97 degrees. Gross ketonuria is common and the blood sugar is extremely low in some cases (3 and 14 mg. per 100 ml. in two cases). Death may follow after twelve to thirty-six hours, but some patients recover spontaneously. The exact nature of this condition has not been fully elucidated but it is apparently associated with severe hypoglycemia in most cases.

Differential Diagnosis

According to Sheehan and Summers,[17] "a confident diagnosis of complete hypopituitarism can usually be made easily from a short clinical examination. The special tests are chiefly of value for confirmation of the diagnosis, assessment of the severity of the disease, and for academic study." However, several conditions may be confused with pituitary insufficiency; the most important of these will be discussed.

Nephrosis.—Mistakes are made in this condition chiefly because of the facial appearance; they usually are rectified easily on further study.

Severe Anemia.—Sometimes these patients are treated for anemia for years. They usually have achlorhydria and a diagnosis of pernicious anemia has frequently been made, although the anemia may be normocytic. The only way to eliminate this mistake is to have a high "index of suspicion."

Myxedema.—It is understandable that this condition causes confusion, since patients who have hypopituitarism often have myxedema. Results of many of the laboratory studies may be compatible. Some authors routinely inquire into the possibility of postpartum necrosis and always obtain a roentgenogram of the sella turcica to exclude a tumor.[11]

Addison's Disease.—Usually patients who have pituitary insufficiency are not pigmented, but this is not invariable and thus difficulty may arise. Conversely, patients who have Addison's disease are usually, but not always, pigmented. For this reason one condition should always be excluded when making a diagnosis of the other. Perkins and Rynearson[11] pointed out that they treated several patients diagnosed as having Addison's disease for years before it was discovered that they had pituitary insufficiency. Sheehan and Summers[17] stated that about 50 per cent of female patients with Addison's disease lose their pubic hair. This produces further confusion. Evidence of thyroid and gonadal deficiency in hypopituitarism should help to clarify the diagnosis.

Anorexia Nervosa.—This condition is characterized by anorexia in a young woman caused by a deep-seated psychic disturbance. Because of insufficient intake of food the patients lose weight, experience secondary amenorrhea and present a low BMR and other signs and symptoms that cause confusion with pituitary insufficiency. A helpful feature in differentiating this condition at least from postpartum necrosis is that these women are usually nulliparous. Sheehan and Summers[17] proved that cachexia and extreme weight loss, such as are characteristic of anorexia nervosa, are not part of the syndrome of hypopituitarism.

Pathologic Findings

In cases of severe pituitary insufficiency necropsy will reveal pronounced atrophy of all the other endocrine glands in almost all cases. The findings of Sheehan and Summers[17] will be summarized.

Adrenals.—The cortex is extremely atrophic but not fibrotic. The medulla remains almost unaffected. The combined weight of the two glands averages 4.7 gm., the normal being 10 to 15 gm.

Thyroid.—Most commonly there is great atrophy of the acini. In extremely severe cases fibrosis may be extensive and in some it appears replaced by a mass of dense scar tissue. The mean weight is 7 gm., normal being 20 to 30 gm.

Gonads and Genital Tract.—Atrophy of the ovaries and superinvolution of the uterus, vagina and vulva occur. The changes are similar to those occurring after the menopause, although they are greater in severity. Microscopically the ovaries show a few primordial follicles, but developing graafian follicles or corpora lutea are absent. Their combined weight is about 4 gm., while the normal is 10 to 15 gm.

The testes show no evidence of spermatogenesis; the seminiferous tubules exhibit hyaline thickening of the basement membrane and the interstitial secretory cells have disappeared completely.

Other Organs.—There is striking atrophy of most of the viscera. This appears to be a true trophic atrophy, since nearly all the patients are of normal weight.

Laboratory Findings

There is nearly always a normochromic anemia of moderate degree, but much less severe than is usually expected from the pallor of the patient as already indicated. Eosinophilia of a mild nature is frequent, as is lymphocytosis.[9]

The serum sodium and potassium are usually normal except when the patient is in a state of adrenal crisis, when the sodium will be reduced and the potassium increased. The serum calcium tends to be in the low range of normal and the serum phosphorus to be in the high range of normal. The blood urea nitrogen and plasma proteins are not altered. The blood cholesterol is usually normal or slightly elevated. This is in contrast to the usual finding of an elevated cholesterol in primary myxedema.

The BMR is always low, usually in the range of —25 to —40 per cent. Treatment with thyroid extract produces a rise to about the normal level.

Gastric analysis most often reveals achylia or achlorhydria, but free acid may be found.

The urea clearance ranges about 50 per cent of normal. The glucose tolerance test is of no value in diagnosis but the curve tends to be prolonged and rather flat.[4]

Some of the special tests of endocrine function are of great value in the diagnosis of pituitary insufficiency and these will be commented upon in more detail.

The most important criteria for the laboratory diagnosis of pituitary deficiency are listed by Wilson[21] as: (a) low BMR; (b) insulin sensitivity; (c) "hypoglycemia unresponsiveness"; (d) negligible urinary excretion of seventeen-ketosteroids and (e) positive results of the water test.

Insulin Tolerance Test.—This involves the study of the patient's response to injected insulin. Patients with pituitary insufficiency characteristically have "hypoglycemia unresponsiveness," which is manifested by a delay in the return of the blood sugar to normal.[4] The dose of insulin injected intravenously is usually 0.1 unit per kilogram of body weight. In the normal individual the blood sugar returns to fasting level in one and one-half to two hours, but in hypopituitarism the return to normal is delayed beyond two hours. A similar curve is obtained in Addison's disease.[11,17,18] In primary myxedema there is a slow fall or insulin resistance, which is in contrast to the rapid fall in hypopituitarism. This rapid decline in blood sugar is a demonstration of the pronounced insulin sensitivity of these patients. Most authors consider that this is a dangerous test, especially in Addison's disease and pituitary insufficiency, and recommend that the dose of insulin be reduced to 0.03 unit per kilogram of body weight. Deaths from insulin have been recorded in these patients, because of their extreme sensitivity.[17] Oelbaum,[9] in a study of several patients with pituitary insufficiency, did not perform the test if the fasting blood sugar was 70 mg. or less.

Excretion of 17-Ketosteroids.—If the disease is severe, the twenty-four-hour urinary excretion of these substances is greatly reduced or absent. The excretion may be lowered by many nonendocrine conditions but usually not to such low values as are seen in hypopituitarism. In primary myxedema the level may be less than 0.5 mg. per 24 hours "in rare instances."[11] The values listed in Table I are hypothetic but experience with many determinations has been in general agreement.

TABLE I. URINARY 17-KETOSTEROIDS: HYPOTHETIC 24-HOUR VALUES IN MG.[4]

Normal male	14.0
Normal female	9.0
Female with Addison's disease	0.0
Male with Addison's disease	5.0
Panhypopituitarism (either sex)	0.0
Gonadectomized male	9.0
Gonadectomized female	9.0

Water Test.—This test is based upon the fact that patients with adrenal insufficiency fail to show adequate diuresis within a given period after oral ingestion of large quantities of water. The test is divided into two parts. In the first part the night volume of urine is measured, the patient being requested to eat or drink nothing after the evening meal. The following morning the patient is given a specified quantity of water to drink (9 ml. per pound) during a forty-five minute period. Specimens of urine are then collected at hourly intervals for four hours. If the volume of any single hourly specimen voided during the morning is greater than the volume of urine voided during the night, the response to the test is negative; such a response indicates the absence of Addison's disease. If the volume of the largest hourly specimen voided during the morning is less than the volume voided at night, the response to the test is positive, and Addison's disease may or may not be present.

To establish the diagnosis the second part of the procedure should be instituted. This involves the calculation of a numerical factor by substituting in a formula that has been devised. If the value of this factor is greater than thirty, the patient probably does not have Addison's disease. If the value is less than twenty-five, the patient probably has Addison's disease, provided that nephritis has been excluded.[6,7]

ACTH Test.—In the normal person, an intramuscular injection of 25 mg. of ACTH results in at least a 50 per cent decrease in circulating eosinophils in a four-hour period. According to Thorn and associates[20] this indicates a "satisfactory adrenal cortical response and in all probability eliminates the possibility of adrenal cortical insufficiency." Recently there has been a growing awareness of false negative responses to this test. At present the intravenous test is felt to provide a more accurate test. Here the patient receives 20 units of ACTH intravenously over an eight-hour period. Changes in circulating eosinophils and urinary steroids are used as indices of response.[22]

Salt Deprivation Test.—This is based upon the fact that normal persons have the ability to conserve sodium on a low-sodium diet. Patients with adrenal insufficiency have a constant excessive loss of sodium in the urine. These patients, when given a low-sodium diet, will be precipitated into an adrenal crisis in two to six days. The test must not be prolonged to actual development of crisis. A decrease in serum sodium and chloride, an increase in hematocrit, an increased blood urea nitrogen and a significant reduction in blood pressure constitute a positive test. The absence of this response renders such a diagnosis untenable. This is a dangerous test and should not be performed unless results of all other tests are equivocal.[18]

TABLE II. VARIATION IN DEGREE OF DAMAGE TO HORMONE PRODUCTION.

	Case					
Hormone	1	2	3	4	5	6
Gonadotrophin	+++	+++	0	+++	+++	0
Thyrothrophin	+++·	0	0	++	++	++
Adrenocorticotrophin	++	0	+	+++	+++	++

0 = No damage.
+ = Mild damage.
++ = Moderate damage.
+++ = Severe damage.

The above tests are the most important ones involved in making a diagnosis of pituitary insufficiency. Measurement of uptake of radioactive iodine and determination of serum protein bound iodine provide other measurements of decreased thyroid function. Attempted measurements of gonadotrophic and thyrotrophic hormone usually reveal their absence, which is, of course, direct evidence of anterior pituitary failure.

Table II, taken from Oelbaum,[9] summarizes results of laboratory investigation of several patients with postpartum hypopituitarism and emphasizes the variability of endocrine dysfunction in these cases.

Treatment

Theoretically the most desirable means of treating insufficiency of the anterior pituitary gland is to replace the missing hormones. At the present time, however, there has been no success in treating patients with anterior pituitary extracts. Perkins and Rynearson[11] considered this is partly because the available extracts lack potency, and perhaps because of the development of antihormones. Present therapy then is directed at replacement of the hormones normally supplied by the target glands of the anterior pituitary.

Adrenal Insufficiency.—Attempts to promote adequate adrenal function with ACTH have been made by many investigators,[2,3,6,19] and although response in some cases has been very good,[3] in general it has been unsatisfactory. It is postulated that the lack of good results in most cases is due to longstanding adrenal cortical atrophy. In disease of recent origin, however, ACTH would appear to be the drug of choice, constituting a more nearly physiologic means of treatment. It should be noted that this therapy may not be completely innocuous, since Summers and Sheehan[19] reported the occurrence of hemiparesis in two of their patients treated with ACTH. No explanation was offered for this and other authors do not report similar experiences. McCullough and associates[8] considered that these untoward effects may have been related to the large doses of ACTH used (100 mg. per day) and pointed out that some patients have been maintained satisfactorily on 25 mg. a day.

At present cortisone seems to be the most satisfactory method of dealing with the adrenal deficit. It has the advantage of being effective orally. The dosage must be individualized, 12.5 mg. per day being effective in one case reported by McCullogh's group, while 50 mg. per day was required in another. It controls the tendency to hypoglycemia quite well, and also relieves the anemia, which is otherwise extremely refractory in some cases. The blood pressure usually rises. Thyroid medication is almost always administered in conjunction with cortisone. It is interesting to note that frank myxedema has developed in patients treated with cortisone alone and that this was corrected by the addition of thyroid extract.[14]

McCullogh and associates were convinced that cortisone was chiefly responsible for the great improvement in two of their cases.

Thyroid Insufficiency.—All authors stress the danger of giving thyroid extract alone to these patients because of the possibility of stimulating acute adrenal insufficiency. Thyroid apparently increased the relative degree of adrenal insufficiency and several deaths have been reported in patients treated for primary myxedema who in reality had pituitary insufficiency.

Thyroid can be given safely and advantageously in conjunction with cortisone or ACTH, as mentioned previously. The sensitivity to cold is dispelled and the BMR is raised to normal levels. The apathy disappears and patients become active and interested in things about them. The electrocardiogram returns to normal. Elevated cholesterol levels tend to decline. Most patients require no more than 1 or 2 grains daily.

Gonadal Insufficiency.—Virtually all men and women with pituitary insufficiency are markedly improved by the use of androgens, according to Perkins and Rynearson.[11] This observation seems to be well substantiated by other investigators. The anabolic property of these hormones apparently constitutes their chief benefit, although their sex-hormone function is also desirable. Growth of axillary and pubic hair is stimulated and libido returns. Increase in appetite and sense of well-being occurs. Excellent results have been obtained in some cases with the use of testosterone alone.[5,8,10] In fact, McCullough and associates stated that "in some patients we have observed the addition of cortisone causes little or no further symptomatic response and is not worth continuing." This has been observed even in patients who have readily measurable evidence of adrenal defect. Methyl testosterone seems to be more effective than testosterone propionate. Here again the dosage must be individualized and doses of from 10 to 100 mg. per day have been used.

In premenopausal women, estrogen replacement therapy is also utilized, such as stilbestrol, 1 mg. a day for twenty-five of each thirty days.

Sheehan and Murdock[16] considered that if a patient can become pregnant again there is an improvement in her condition during pregnancy. They stated that after delivery normal menstruation may begin, and the patient subsequently remains free of any evidence of hypopituitarism. This they attribute to hypertrophy of the remaining pituitary tissue under the influence of pregnancy and its failure to regress after delivery. Cases of this nature are unusual and may occur in incomplete pituitary failure when sterility is not present.

Diet.—The diet should contain liberal amounts of salt and some authorities recommend giving supplementary sodium chloride. If defects in electrolytes persist despite therapy with cortisone, thyroid and testosterone, 2 to 4 mg. of desoxycorticosterone (linguets) a day may be given. This tends also to alleviate symptoms of weakness if they persist and also to elevate the blood pressure. Cook and co-workers[15] said that the diet should be high in calories and carbohydrate, and moderate in protein, making certain that the intake of carbohydrate is not very high in any individual meal but is spread out over the twenty-four-hour period by the use of multiple interval feedings. Others[12] stated that a regular diet without frequent feeding is sufficient.

Pituitary Coma.—Most of these cases are associated with severe hypoglycemia; intravenous administration of glucose usually results in some improvement but it must be maintained for a prolonged period (several days).[17] Cortisone apparently had a very beneficial effect in one patient reported on by Allott and Simmons.[20] The patient was semistuporous and febrile. Prompt recovery occurred after administration of 200 mg. of cortisone although glucose and desoxycorticosterone were also utilized. Immersion in a warm bath was utilized in one patient who had associated hypothermia; an apparently favorable effect ensued. It should be noted that some of these patients recover spontaneously over a period of many days or weeks when supportive therapy alone is given.

In summary, the reported results of therapy in pituitary insufficiency are almost universally successful and in some cases they are striking. However, Cook and associates cautioned that treatment is not simple and often proves to be unavailing despite all efforts of the physician.

References

1. Allott, E., and Simmons, J.: Brit. M. J., 2:568-569 (Sept.) 1951.
2. Cook, J.; Bean, W.; Franklin, M., and Embick, J.: A.M.A., Arch. Int. Med., 87:517-532, 1951.
3. Eckholm, R.: Acta Med. Scandinav., 144:1, 61-70, 1952.
4. Fraser, R.; Albright, F., and Smith, P.: J. Clin. Endocrinol., 1:297-306 (April) 1941.
5. Hurxthal, L.: Lahey Clinic Bull., 8:5, 137-41 (July) 1953.
6. Knowlton, A.: Jailer, J.: Hamilton, H., and West, R.: Am. J. Med., 8:269-284, 1950.
7. Levy, R., and Becker, G.: Illinois M. J., 102:1 (July) 1952.
8. McCullogh, E.; Skillern, P., and Schoffenberg, C.: Cleveland Clin. Quart., 21:31-38 (January) 1954.
9. Oelbaum, M.: Brit. M. J., 2:4776 (July 19) 1952.
10. Oelbaum, M.: Quart. J. Med., 21:83, 249-64 (July) 1952.
11. Perkins, R., and Rynearson, E.: J. Clin. Endocrinol. & Metab., 12:574, 1952.
12. Proof, J., and Craig, W.: Western J. Surg., Obst. & Gynec., 61:3 (March) 1953.
13. Robinson, R.: Power, M., and Kepler, E.: Proc. Mayo Clin., 16:577-582 (Sept.) 1941.
14. Schrock, C.: Sheets, R., and Bean, W.: J. Clin. Invest., 30:174-180, 1951.
15. Sheehan, H.: Quart. J. Med., 32:277-307, 1939.
16. Sheehan, H., and Murdock, R.: Lancet, 2:132, 1938 (cited by Cook).
17. Sheehan, H., and Summers, V.: Quart. J. Med., 18:319, 1949.
18. Soffer, L.: Bull. N. York Acad. Med., 29:2 (Feb.) 1953.
19. Summers, V., and Sheehan, H.: Brit. M. J., 2:564-568 (Sept.) 1951.
20. Thorn, G.; Faraham, P.; Prunty, F., and Hills, A.: J.A.M.A., 137:1005-1009 (July) 1948.
21. Wilson, L.: Lancet, 1:5 (Jan. 31) 1953.
22. Wolfson, W. Q.: A.M.A. Arch. Int. Med., 92:108-147 (July) 1953.

Laboratory Aids to Medical Practice

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LABORATORY DIAGNOSIS OF PITUITARY FUNCTION

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IN SPITE of the encouraging advances in laboratory techniques for the diagnosis of endocrine disease, there is available at the present time only one direct measure of pituitary function, namely, the demonstration of gonadotropic activity in the blood and urine. The clinician's diagnostic acumen remains, therefore, his most effective tool in the evaluation of pituitary function; with the aid of such relatively simple determinations as the basal metabolic rate, blood cholesterol and perhaps the number of eosinophils in the blood, accurate, although indirect, assessment of pituitary disorders is possible.

There are six established pituitary hormones for which one theoretically might be called on to test, namely corticoptropin (ACTH), somatotropin (STH), thyrotropin (TSH), follicle-stimulating hormone (FSH), luteotropin (LTH) and luteinizing hormone (LH). Although all of these substances have been isolated in highly purified form, practical laboratory determination is available only for the so-called gonadotropins. For didactic purposes, gonadotropins include FSH, LH and LTH, while gonadotropin as determined by the bio-assay procedures using rats or mice has a proved content of only FSH and LH. Whether LTH is involved in the results of this determination is not known. Thus the titer of gonadotropin, while it is not dependent solely on the determination of FSH, as claimed by some, represents a reliable clinical laboratory method for estimation of pituitary gonadotropic activity. Perhaps the main diagnostic application of this procedure is in cases of retarded sexual maturation and certain sexual dystrophies wherein selective pituitary gonadotropic hypofunction cannot be differentiated from failure of end-organ response. In general, titers of gonadotropin are high in castrated persons, postmenopausal women, Klinefelter's syndrome or hypergonadotropic eunuchoidism with aleydigism and in Turner's syndrome or primary ovarian agenesis. Gonadotropin is decreased or absent in destructive processes of the pituitary, such as Sheehan's syndrome.

For practical purposes, laboratory evaluation of the activity of the other pituitary hormones is done through indirect methods. The control of the number of eosinophils in the circulating blood by the adrenal corticosteroids was initially thought to provide a simple evaluation of the integrity of the pituitary-adrenal axis and thus of corticotropic activity. After the administration of epinephrine, a decrease in eosinophils occurs; it was presumed that the pituitary had been stimulated to produce excessive corticotropin, which in turn augmented the production of corticosteroid, resulting in eosinopenia. Unfortunately, later experience has invalidated the epinephrine test because of the demonstration of a direct eosinopenic action of epinephrine. Indeed, the pronounced normal diurnal variation in the number of eosinophils in the blood renders the direct stimulation of the adrenal glands by means of the ACTH test a much less reliable gauge of adrenal activity than was formerly believed.

The determination of corticotropic activity in the blood is a relatively involved bio-assay procedure that is not yet available for general laboratory use. No corticotropic activity can be demonstrated in normal persons or patients with Cushing's syndrome, whereas corticotropin is consistently present in the blood of patients who

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have Addison's disease or the adrenogenital syndrome; it is present in some patients with acromegaly.

Perhaps the best tests for the evaluation of the adequacy of pituitary corticotropic activity in the absence of known primary adrenal disease are the determinations of the 24-hour urinary excretion of 17-ketosteroids and corticosteroids. In the presence of diminished amounts of these steroids, parenteral administration of corticotropin (ACTH) will cause an increase of the urinary steroids in patients who have pituitary failure. The rate of recovery of steroid production will, of course, depend on the degree of adrenal inactivity imposed by loss of normal pituitary stimulation. On the other hand, no method is available for the direct measurement of excessive corticotropic activity in the presence of a responsive adrenal. The hypothetic result of such a disorder would be Cushing's syndrome or the adrenogenital syndrome. Although the majority of patients who have Cushing's syndrome are thought to be suffering from a primary disorder of the adrenals, the few who have undergone remission after irradiation of the pituitary may be examples of corticotropic hyperactivity.

The accurate assessment of thyrotropic activity remains largely a function of clinical diagnosis. Pituitary myxedema is the clinical counterpart of decreased thyrotropic activity. While this secondary type of myxedema may be accompanied by other stigmas of hypopituitarism, it may present a difficult problem in differentiation from primary thyroidal failure. In addition to the presence of normal or decreased values for blood cholesterol, the administration of thyrotropin to these patients will restore sufficient thyroidal function to cause significant increase in the uptake of radioactive iodine. In primary failure of the thyroid one would expect no alteration of thyroidal function after administration of thyrotropin. The many theories involving excessive amounts of thyrotropin or the presence of abnormal types of this hormone in exophthalmic goiter go untested because of lack of a direct technique for the measurement of TSH. Investigations still in the experimental stage involving the action of TSH on the metamorphosis of tadpoles have not proved adequate in the clinical laboratory.

The effects of excessive production of somatotropin are usually so dramatic that little need exists for dependence on the clinical laboratory. Often, however, it is desirable to know whether or not the process is still active in acromegalic patients. The presence of hyperglycemia and hyperphosphatemia is a reliable indicator of a continued increase in somatotropic activity.

The complex protein nature of the pituitary hormones and the minute quantities required for the performance of their spectacular tasks continue to defy their direct practical laboratory examination, with the exception of the gonadotropins. However, a high degree of accuracy in clinical endocrinologic diagnosis is attained by the astute physician despite the meager help available from the laboratory.

INJURIES TO THE DIAPHRAGM

(Continued from Page 800)

diaphragmatic hernia. Surg. Clin. North America, 26:152-162 (Feb.) 1946.
8. Downing, W. C.: Personal communication.
9. Gibson, F. S.: Diagnosis of diaphragmatic hernia with acute obstruction. J.A.M.A., 93:1719-1724 1929.
10. Gross, R. E.: Surgery of Infancy and Childhood pp. 428-444, Philadelphia: W. B. Saunders Co., 1953.
11. Hamilton, J. E. and Phillips, T. W.: Traumatic hernia of the diaphragm, Am. J. Surg., 78:686 (Dec.), 1949.
12. Harrington, S. W., Diaphragmatic hernia. In Cyclopedia of Medicine, Surgery, Specialties, 4:559.
13. Harrington S. W.: Esophageal hiatus hernia. Rocky Mountain M. J., 1952.
14. Harrington, S. W., Traumatic diaphragmatic hernia. Surg. Clin. North American 30:961, 1950
15. Harrington, S. W., Various Types of Diaphragmatic Hernia Treated Surgically, Surg. Gynec. & Obst., 86:735-755 (June), 1948.
16. Hedblom, C. A.: Diaphragmatic hernia. Ann. Int. Med., 8:156, 1934.
17. Hochberg, L. A.: The Thoracic Surgical Patient. New York: Grune and Stratton, Inc., pp. 120, 254-262, 1952.
18. Isaac, F. Wilkins, F. B. and Weinberg, J.: Radiology, 55:527-533, 1950.
19. Johnson E. K. and Mangrardi, J. L.: Traumatic pericardiophrenic laceration with diaphragmatic herniation. New York State J. Med., 53:1234 (May 15) 1953.
20. Kinsella, T. J., Personal communication.
21. Klertsch, W. P., Munger, A. D. and Johnson, W. F.: Diaphragmatic hernia with complete evisceration of the liver. Ann. Surg. 130:1079, 1949.
22. Strode, E. C. and Vance, C. A.: Herniation of the right diaphragm secondary to trauma, Ann. Surg., 137:609 (May) 1953.
23. Sweet, R. H.. Thoracic Surgery, Philadelphia: W. B. Saunders Co., pp. 330-331, 1950.
24. White, J. M., Jr. and Sladczyk, G.: Traumatic rupture of the diaphragm with transthoracic repair, Texas State J. Med., 47:644 (Sept.) 1951.
25. Williams, D. B.: New Orleans M. & S. J., 101:544 (May) 1949.

President's Letter

A TIME FOR REFLECTION

This time of year—the time of harvest and gathering of crops—is the traditional time to be thankful. And yet it makes us wonder at times why it should be necessary to set aside a special day just to be thankful.

For, a special day to be thankful should not be necessary at all—we should be thankful day by day. The current popular song "Count Your Blessings," which may be patterned after the old-time hymn, is certainly appropriate now.

Thanksgiving is a time for reflection on all the things for which we should be thankful. We of the medical profession have no cause for complaint on this line, in spite of certain setbacks in our path.

Recently I have had the opportunity of hearing a first-hand report on medical and hospital conditions in the Scandinavian countries and it made me sincerely grateful that I have had an opportunity to practice medicine without government interference. I am thankful too, that the profession here is alert to the dangers of such interference, and that we have fought for anything which eliminates such a threat.

We can be thankful for strong leadership—in government, in civic affairs, in schools, in churches and in all the voluntary organizations which make this country, itself, a respected leader in world affairs.

We can reflect on all the ordinary, mundane, commonplace things, which work for our benefit daily, and which we all take for granted as our right. But these things, so treasured in many other places in the world, are among the most important in our lives. Such things as the right to worship as we wish, the right to think and say things we feel, the right to meet in a group publicly, the right of privacy, and the right of protection against oppression, are all among this group of things for which we should be eternally thankful.

There are certain material things which cannot be overlooked in the counting of blessings. Necessities and luxuries both are provided in America in great abundance, and their availability has come to mean a tremendous rise in the standard of living over the years. For this, too, we should give thanks.

But most of all, we of the medical profession should thank our Maker for the opportunity of benefiting from the great good spirit of the vast majority of our colleagues. A trend has been developing—a trend toward a new and necessary emphasis on the value of the general practitioner and all that he has been endowed with by tradition. He has come into new and well-founded prominence, and his contribution to the extreme good health of this nation cannot be underestimated.

With all these blessings, a grateful nation officially gives thanks this month, and we can hope and pray that a watchful nation will make sure that these enormous advantages will forever bless us.

President, Minnesota State Medical Association

Editorial

ARTHUR H. WELLS, M.D., *Editor;* HENRY G. MOEHRING, M.D., and JOHN F. BRIGGS, M.D.

THE IMPORTANCE OF STATISTICS TO THE PHYSICIAN

IN the most general sense, statistics are observations or data affected by a multiplicity of causes. Such observations are the essence of the examinations we make in daily practice. A series of statistical facts comprises our case records. Some are qualitative, some quantitative. We cannot ignore our qualitative observations, yet when possible we substitute quantitative measurements for qualitative descriptions because we are interested in the degree or extent of any given abnormality and wish to record it as precisely as possible. Statistical methods are primarily concerned with evaluations of such quantitative observations, though methods are available also for utilizing qualitative information.

Given an accumulation of such observations, we require a means of talking about the data meaningfully. The tools available to us are the various statistical techniques, which are no more than logical summarizing processes. Statistical methodology is simply the language of observation. It tells us no more than is inherent in the original facts. However, it simplifies the handling of the data and enables us to draw reasonably exact conclusions.

It also protects us. We avoid ignoring any aspect of the data, and, if we don't abuse the method, we are prevented from overestimating or underestimating the significance of our results. Furthermore, if we pose a question we want to answer, and think through our experiment and methods of analysis before we set out to collect the data, we are helped greatly by the exact requirements set by the methods. They help us to crystallize our problems, to isolate what is relevant and exclude what is irrelevant.

Familiarity with the general concepts of statistical methodology is of obvious importance to us in compiling and presenting data for publication or in evaluating published reports. We are all concerned since today the critical reader of medical literature must be as informed as the critical writer.

Whether we are assembling data for publication or simply drawing deductions from our own clinical experiences, three recurring factors influence our impressions, evaluations, or conclusions—viz. the amount of variation in our observations, the extent to which we select our material consciously or unconsciously, and the adequacy of our control comparisons. A single description of a phenomenon not previously recognized is generally a contribution of value. However, beyond the initial description we are usually concerned with variations of the phenomenon in other cases. In most circumstances in medicine and biology, then, we record the variations in some measurement. In drawing conclusions from our observations, whether for purposes of simply describing the group or for purposes of contrasting one group with another, we must have clearly in mind the extent of variation of our data. The variability of the data is in fact as important to know as the typical finding. It is the variability primarily that determines the number of cases one must observe before making a generalization, though the extent of one's inductions is also important. There is no set minimum number of cases that one must use to make a given point. Generally in medicine approximately thirty to fifty cases will represent fairly well the range of variation to be expected with a typical quantitative variable. However for some purposes several hundred observations may be necessary and for others eight to ten may be sufficient. It is wise as a practice to be wary of conclusions drawn from a few cases.

Each of us accumulate clinical impressions from his daily experience. What we observe ourselves we accept most readily. Yet our personal impressions are very susceptible to error. In most instances the experience of any one of us is limited and does not cover the possible variations of a disease. Selective processes strongly influence us. The mere fact of the patient's seeking the physician is a step in the selective process, and hospitalization is a further step. Over and above such physical processes of selection which determine the nature and extent of our individual experiences are our psychological reactions. Even though our experiences be broad, our minds are selective. Without systematic tabulations, we tend to emphasize some items and exclude others. How many of us have not had the experience of gathering impressions as we collect our data and finding them erroneous in various respects when we make the final objective analysis? The errors we make in generalizing from our clinical experiences are thus both statistical and psychological, with the latter perhaps predominant.

Selection is intimately linked with the problem of control observations. The importance of both is most evident in clinical trials of therapeutic agents. The typical study compares a new therapy with some standard therapy or control situation, avoiding selection of cases in building the test group. We have introduced few therapeutic agents whose effects have been so phenomenal in contrast to previous failures as to obviate the need for a controlled study. For many illnesses we find multiple therapies advocated. This in itself is evidence of our need for controlled observations, since one medication or a related group of medications is generally sufficient when our treatment is specific. A positive claim for a new therapeutic agent is easily made but proved or disproved with difficulty. As a result we have vogues in therapy such as we have observed and are presently observing in the case of hypertensive vascular disease. We need more skeptics who will resist the claims of published reports and pharmaceutical advertisements and demand controlled observations before using a drug. It is sobering in this respect to read the analysis of Ross (J.A.M.A. 145:72, 1951) who analyzed 100 unselected articles then current in leading medical journals and, despite a liberal interpretation, found only one-fourth with adequate controls.

Under the best of circumstances, truly controlled studies are difficult to arrange in clinical medicine. What we desire is identity of the expermental and control groups except with respect to the agent being evaluated. If we are careful, we try to match our groups in all conceivable significant ways; however, because a multiplicity of interrelated factors is ever present in the background, we must recognize that unknown factors may operate despite our precautions, and the effect we observe may result from some unforeseen source of variation rather than the therapeutic agent. This is the primary reason that concurrent rather than retrospective controls are almost essential, and that random allocation of subjects to control or experimental groups is highly desirable. Given the completed study with all such precautions incorporated, it is still wise to seek alternative explanations for the differences observed before attributing them to the variable under consideration.

The interpretation of variations in symptoms, signs, and tests every day in arriving at clinical diagnosis is another circumstance where familiarity with statistical concepts may help us. This is an unexplored and difficult field for objective statistical evaluations. Yet as physicians we are required to weigh, relate, and combine several variables each time we make a diagnosis. The processes we use in arriving at our diagnoses are essentially statistical, though subconsciously so. If we could objectify some of the thought processes that pass under the name of clinical experience or ingenuity, we might all anticipate more diagnostic successes.

Consciously or unconsciously, we base our clinical diagnoses primarily on the patterns that various diseases manifest. In assembling the findings that make the pattern, the expert knows when to attach much or little significance to a particular finding. He weights the variables appropriately. Remembering a specific case is but linking the pattern to the case. For any given disease, the variations in patterns are great. The extreme variants come to us as unusual cases. Such instances with unusual manifestations, often involving a common disease, are among our most difficult diagnostic problems.

Our mistakes in diagnosis revolve about our interpretations of patterns. We may omit an important variable by failing to elicit a given finding.

This happens most commonly, not because we do not recognize the abnormality but because we simply do not look for it. It illustrates the value of a systematic examination. We may misinterpret the significance of various findings, overestimating some and underestimating others. Our emphasis or weightings are then inappropriate. Sometimes the full pattern is not manifested, and we are unable to classify the illness. Often we fail to think of all the possibilities that have patterns similar to that observed. In particular, we are less likely to think of the rare diseases. As we must have systematic examinations, so must we have systematic appraisals. Such systematization is equally valuable in prognosis where we commonly recognize the importance of individual variables yet do not utilize them fully in our evaluations.

Delineation and classification of the patterns of diseases will emerge from studies of interrelationships among the various manifestations we record. Such clearly defined patterns in diagnosis are prerequisite to objective checking systems. Given these checking systems, we may expect to minimize our errors of omission, which are perhaps the largest source of our mistakes in diagnosis and prognosis.

Leslie Zieve, M.D.

REDUCING NEONATAL MORTALITY

Let us consider these statistical facts:

1. More deaths occur during the first year of life than in the total of the next thirty-four years.
2. Although only about 6 per cent of Minnesota infants are born prematurely, the mortality among these babies is so high that it accounts for more than one-fourth of all infant deaths in this state.
3. Of all the deaths that occur during the first year of life, 75 per cent are within the first twenty-eight days, the *Neonatal Period,* the most critical period of life.
4. During the neonatal period, immaturity is responsible for more than one-third of all the deaths.
5. Infant and neonatal death rates have shown a continuing decrease during the past several decades.
6. The reduction in the number of deaths has been much greater during the postnatal period, between one and twelve months, than during the first month of life.

In view of these facts, if we are to reduce infant deaths further, we must reduce the number of deaths in the neonatal period. The chief cause of death in this early period is immaturity. It ranks eighth among the ten leading causes of death in all age groups in Minnesota. Other causes of death in the neonatal period are congenital anomalies, infection, and erythroblastosis. The challenge is to all physicians, but particularly to those practicing obstetrics and pediatrics. In Hennepin County, the challenge is being met in part by the current mortality study.

One-third of all neonatal deaths are associated with obstetrical complications. Adequate prenatal care for every pregnant woman thus becomes important. Necessary hospitalization and expectant supportive treatment continued as close as possible to term should help reduce neonatal mortality by reducing obstetrical complications. Caesarian sections that must be done before term for the benefit of the mother frequently result in neonatal deaths. There is always danger of obtaining an immature infant when elective sections performed because of pelvic disproportion or previous section are done too early. In the Hennepin County study, most of the deaths of infants delivered by section were due to abnormal pulmonary ventilation with resulting anoxia. (Almost 40 per cent of all neonatal deaths in Hennepin County in 1952 were due to abnormal pulmonary ventilation. This term was coined by Edith Potter and refers to deaths of infants who show no specific disease other than in the lungs; i.e., hyaline membrane disease and atelectasis.)

The significance of birth weight in survival is evident because less than 1 per cent (0.6 per cent) of full-term infants die during the first twenty-eight days of life while almost 20 per cent of all immature infants die during that period. The chance of a full-term infant's surviving the neonatal period is thus about thirty-two times greater than that of a prematurely born one. One-half of the immature infants who die, do so in the first twelve hours of life; 80 per cent of them die in the first forty-eight hours after birth.

Further reduction in neonatal mortality requires improvement in prenatal care beginning early in pregnancy. The intrauterine existence of the infant must be continued as close to term as possible to reduce the incidence of immunity. The prematurely born infant must be treated promptly and adequately. Prenatal, obstetric and newborn records must be complete and accurate. Every infant and fetal death should be studied by adequate autopsy. At the recent Fourth Governor's Conference on Children and Youth in Minneapolis, Dr. Martha Eliot, Chief, U.S. Children's Bureau, stressed the value of maternal and neonatal mortality studies in the reduction of mortality in Minnesota. A continued and more extensive neonatal mortality study as well as an intensive study of fetal deaths will help further to reduce infant and neonatal mortality in this state.

A. B. Rosenfield, M.D.

ARE INTERNSHIPS TOO EASY?

IT APPEARS that many of today's interns do not appreciate the reason for their twelve-months' tour of duty. They seem to consider their internship an obstacle in the way of better things. When we watch them arrive at the hospital in the morning, later than most of the staff consultants, and when we stop at the hospital on the way home from the office to see a sick patient and discover that most of the interns are already through for the day, we wonder whether anybody has ever stopped to tell these recent medical school graduates why they are required to spend twelve months working in a hospital.

Do these young physicians realize that in their internship they are finally given a chance to apply the various theories and principles to which they have been exposed during their medical school. After spending many years on the so-called background of their medical training, they are actually given a chance to heal the sick. Of course, in a good internship, this is done under supervision. The extent of the supervision varies considerably. In private hospitals, the actual care of the patient is usually done completely by the staff physician. The intern acts as an assistant and an observer. However, this need not detract from the value of this type of training, for the intern is immediately concerned with the day-to-day care and the detailed management of his sick patient. It is during this period of a doctor's training that he usually decides how he is going to do things when he has complete responsibility for the care of a patient.

This valuable period must necessarily be short. Those who are heading for a specialty must move on to residency training. Those who are planning to go into general practice must get ready to start that practice. These young doctors must progress to a situation where they are given more responsibility than is allowed a doctor just out of medical school. The intern will get the most out of his brief period if he is as busy as possible. When someone objects to an internship because it leaves little time for reading, he is actually stating that he has a worthwhile appointment.

An intern can learn most about the management of a hospital patient by following carefully the people for whom he is responsible. This can best be done by making rounds. The house officer has to make his own rounds in addition to those on which he accompanies the staff consultant. In addition to studying his own patients, an intern must take note of any rare or interesting condition in the hospital, even though such a patient may be another intern's responsibility. The doctor who has seen and remembers cases of the unusual is ahead of the one whose experience rests with only the routine situation.

Many hospitals tend to relieve the intern of such technical procedures as starting intravenous infusions and passing stomach tubes. This is not in the best interests of the man in training. These are technical procedures, and the only way to become skilled in them is to repeat them many times. Even if the doctor practices in a hospital where there are nurses or interns to do all this work for him, he will find that the difficult patients are always saved for himself. If he is not proficient in these procedures, he will be less skilled than his nurse.

The numerous histories and physical examinations cause as much objection by the interns as any part of their training. Medical educators will agree, however, that this is one of the most valuable parts of an intern's training. Undoubtedly, most mistakes in the practice of medicine are due either to an inadequate or to an unsatisfactory history and physical examination. Practice improves the ability to perform this task. It is during his internship that the doctor should learn the difference between a complete history and a detailed medical school type history. No matter what branch of the practice of medicine an intern enters, if he has learned how to take a good history and do a good physical examination rapidly, he will be a better doctor.

Although formal lectures and clinics are the least important part of his internship, he should make it a point to attend those given by the staff of his hospital. Usually, these illustrate a problem related to a patient under his care and, furthermore, they give him a chance to learn how the members of his staff feel about particular problems. By far the most important demonstration to which he will be exposed is that which occurs in the postmortem room. The percentage of necropsies has justly been given an important place in the rating of a hospital for training.

In order to get all these things done, the intern must have his day organized. His time off should be taken only after his work is done. Obviously, this is a full-time job. The innumerable calls and emergencies are going to disrupt the best laid plans of organization for the day. The day soon extends into night, and it is apparent that he is trying to crowd into twenty-four hours what just doesn't seem to fit there. That is as it should be. In order to carry out this crowded program, little time remains for extracurricular activities. However, a good internship is so important in the making of a good doctor that there should not be any extracurricular activities during this period. An internship should be considered not as a job but as a temporary way of life. This sounds like a grim prospect to some medical school graduates, but, in addition to the knowledge of medicine gained from such a program, the recent graduate also has the opportunity to decide whether he wants to spend his life in the hurly-burly of the practice of medicine or whether he would rather continue his career in one of the more academic fields of medical science.

Josiah Fuller, M.D.

ACCIDENTS, NUMBER 1 KILLER OF CHILDREN

EVERY doctor of medicine in Minnesota fully realizes that accidents are the Number 1 killer of our children. Although tuberculosis, contagious diseases, rheumatic fever and acute infections still take their annual toll of children, they no longer are at the head of the list. This improvement is very much to our profession's credit. Accidents as the killer of children, on the other hand, are on the increase. This situation constitutes a major challenge to us practicing physicians; we have a moral obligation to help eradicate accidents as well as we have eradicated diphtheria. This we can do through safety education, first of ourselves and later of the families in our individual practices.

During the next several months a series of articles will appear in Minnesota Medicine calling attention to safety education. Home accidents, a statistical review, is the beginning. Other contributions will include poisons, burns, farm accidents, aspiration and asphyxiation, gun shot wounds, motor vehicle accidents, drownings, et cetera.

In this way, your editorial board trusts that you will be repeatedly reminded of the physician's responsibility to reduce the ravage of children by accidents. Your self-scrutinizing question must be:

Are you administering the new safety vaccine?

Tague Chisholm, M.D.

MUNICIPAL BONDS AND THEIR ROLE IN A DOCTOR'S ESTATE

IN investing for income, municipal bonds should play an important part in a doctor's plans for his estate; first, during his earning years, and later in his estate when his professional earnings are no longer a factor. With our income tax structure still at high levels, the tax-exempt municipal bond constitutes a form of investment which provides "take-home" income that is most gratifying in comparison with other types of investment.

First, a doctor may be too busy with his practice to make an adequate study or may not want to take unusual risks in placing his funds in other forms of investment, so a program of purchase of good municipal bonds, second only to Federal Government securities in history of payment, generally speaking, offers a sound medium. Then, too, there are periods during a doctor's investment years as a stock buyer when the market may be topping off. At such a time, what could be more logical than a consolidation of profits by a switch of investment into tax-exempt bonds? An application on one's tax rate should be made to give a correct picture of the worth of what appears as large dividend income from equities.

There are currently so many forms of municipal obligations, it might be well first to consider limiting investment in those which are general obligations and payable from ad valorem taxes or payable from such primary revenue sources as water, electric, gas, and sewer service. There are others which have merit with adequate principal and interest coverage, but careful analysis should be made through an investment banker who has access to first-hand information. When one considers the assured "take-home" income of tax-exempt municipal bonds, it is not necessary to reach for the larger rates of yield available to be well compensated. The following will illustrate the ample advantage in contrast with taxable in-

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Medical Economics

Edited by the Committee on Medical Economics
of the
Minnesota State Medical Association
George Earl, M.D., Chairman

PHYSICIANS NUMBER ALL-TIME HIGH

A record graduation of 6,861 physicians during the past year by our nation's medical schools has boosted the ratio to an all-time high of one physician for every 730 persons in the United States. This ratio will be lowered even more in the next few years as the number of medical graduates is expected to rise due to the continued expansion of the country's medical schools.

This information is from the fifty-fourth annual report on medical education in the United States by the American Medical Association's Council on Medical Education and Hospitals. The report states that today's physician population has now reached approximately 220,100. Other highlights of the report are:

> "Enrollment of 28,227 is largest number of medical students in the history of U. S.
>
> "Freshman class enrollment of 7,449 also is a record.
>
> "More than 76 million dollars was spent during 1953-1954 for new facilities, remodeling or completion of buildings for medical instruction.
>
> "Budgets for medical schools during 1954-1955 total more than 143 million dollars.
>
> "21,328 physicians did volunteer teaching without pay during the year.
>
> "Ten new four-year schools are in construction or planning stages and will be in operation within the next few years."

In conjunction with this, figures have been released on the latest contributions to the American Medical Education Foundation—dedicated to give funds to American medical schools. According to a recent news bulletin from the American Medical Association, "Several large contributions from state medical associations have boosted the American Medical Education Foundation nearer to its goal of two million dollars in 1954."

States Increase Dues

Several state medical associations have voted to increase members' dues for contributions to the American Medical Education Foundation. Ohio took this action some time ago, and recently the state medical associations of Arizona and Utah have followed this example. A contribution of $100,000 was presented from the California Medical Association, and the Arizona Medical Association contributed $7,230 to the fund.

Since January 1, 1954, the Foundation has received a total of $968,000 and expected to reach the one million mark in September.

HOSPITAL EXPENSE SOARS RESULT: SICK HOSPITALS?

A recent article in the *Wall Street Journal* reports that American hospitals are suffering from a sickness found difficult to cure: soaring costs. And the result will probably be more fee raises for patients, to offset the deficit.

The American Hospital Association reports "it cost general hospitals $21.09 a day last year to care for the average patient whose bill came to $19.49 a day, the deficit being covered by charitable contributions and government grants. In some high-cost areas such as California and Connecticut, the average daily cost of institutional bed and board is edging close to the $30-a-day mark.

Reasons Cited

The article states that hospital deficits are swelling as the average deficit per patient is multiplied by an ever-growing stream of sick folk making use of hospital facilities. Admissions in 1953 totaled 20,183,827, almost triple the number 20 years ago.

Take Steps to Reduce Costs

Many hospitals are using industrial methods in an attempt to reduce costs of hospital care. Many are installing more efficient communication systems to save nurses from walking back and forth to patients' rooms so much. They're installing more automation in hospital laundries, too, and hiring lower-paid clerks to relieve nurses of much of their paper work. But the consensus among typical hospital superintendents is that any economies resulting from these innovations still cannot be expected to offset entirely their rise in labor costs.

"It's an unhappy fact, but about the only direction that costs can go is up," states a spokesman for the AHA. "We're taking some satisfaction that the increase in the last year ($1.60 a day per bed) is the smallest of any year since the war."

Shortened Stay Costly

The shortened stay of the hospital patient of today, now about eight days on the average compared with two weeks or more in 1929, also lessens a hospital's chances to break even. Why? A hospital superintendent says:

> "We used to make a little money on the patient while he was recuperating, just lying in bed not requiring much attention. Now, take an appendectomy, for example. The first day he is taking all sorts of tests; the second day he is operated on and requires all sorts of attention; the third day we have to watch him closely; then the next day he is up taking a few trial steps and we have to be careful that the stitches don't break. After that he goes down to pay his bill, a dead loss every day he has been in our hospital."

Labor Costs High

Another hospital superintendent says that it's the rising cost of hospital labor that has hospitals licked. They can't cut costs faster than the rise in wages.

The *Journal* states that "since 1946, the number of hospital employes per 100 patients has soared from 148 to 183 on the national average, while pay scales have doubled. The increase in employes results both from the more intensive care now given patients and the shortening of work weeks to meet the competition of private industry.

Opposite Views Expressed

Speaking at the convention of the American Hospital Association, Benson Ford, president of Detroit's 650-bed Henry Ford Hospital, stated:

> "There is room for improvement in the purely economic performance of most hospitals—particularly in the purchasing and house-keeping departments. . . . If it makes sense for industry to spend $20 or $30 to save $50 or $100, it should make sense in the hospital. And I suggest that the profit motive might be put to work in hospital administration with highly rewarding results."

But, a hospital superintendent reacted this way: "If Mr. Ford were a hospital superintendent he would be tearing out his hair at the end of the first month." The superintendent went on to describe the contrast in scope of a hospital's work and certain phases of industry:

> "He has about five basic models of cars to make. We have 150 or more models every day—human beings and sick ones at that, who can't be handled on an assembly-line basis. We're also called on to run a good-sized laundry, a restaurant, and a peculiar one at that with every diet different, provide a work shop for a group of very individualistic doctors, operate a collection agency, a labor recruiting office, and a purchasing agency all at the same time. No, we can't switch over 100 per cent to industrial methods. Our No. 1 job is to keep the patient happy and healthy."

Prepayment Helps

The rise in hospital expenses has been obscured to some extent by the phenomenal growth of prepayment insurance plans, which now cover two-thirds of the population. And, the *Journal* explains, even here distress is being felt as premiums have had to be increased or benefits cut because of rising claim costs. Prepayment does not seem to have been able to catch up with the rising costs in hospitals and the resultant rise in individual patient fees.

A Farm Bureau health section official, whose organization has signed up a large number for perpayment hospital contracts, sums up the situation thusly: "Farm people recognize the benefits they received from advances in medical science in the last decade, but there is a limit to anything and we view with great concern the current upward climb of costs."

INDIVIDUALISM VERSUS SOCIAL SECURITY NOTED

A recent article in *The Freeman*, by Helmut Schoeck takes special notice of American trends regarding individualism as contrasted to the collectivism of social security. The article states that "through tax deductions for insurance savings, Americans could build up their individual programs for true social security without surrendering to Welfare State paternalism."

Lamenting a little over what seems to be a decrease in the individualist tendency in America, Mr. Schoeck, quoted in a recent issue of *Insurance Economic Surveys,* reiterates the words of Dwight D. Eisenhower in 1949, when he was president of Columbia University. He calls Eisenhower's words among the clearest expressions of the individualist philosophy:

> "Millions of us, today, seem to fear that individual freedom is leading us toward social chaos . . . that we have reached the point where the individual is far too small to cope with his circumstances; that his lifelong physical security against every risk is all that matters. More than this, we hear that such security must be attained by surrendering to centralized control. . . . On every account the fearful men are wrong. More than ever before, in our country, this is the age of the individual. . . . The modern preachers of the paternalistic state permit themselves to be intimidated by circumstances. Blinding themselves to the inevitable growth of despotism, they — cravenlike — seek, through government, assurance that they can forever count upon a full stomach and warm cloak, or—perhaps—the sinister-minded among them think, by playing upon our fears, to become the masters of our lives."

According to the article, far less publicity was given to another, and quite contrary, development at Columbia University around the same time. It, too, is worth recollecting, the article states, for it represents on the highest level that march toward the Welfare State that would trample and obliterate the statement above quoted. This event was the publication of a report of The American Assembly on Economic Security for Americans, which organization was infiltrated by proponents of the all-inclusive Welfare State.

The report brings in the usual argument of the "increasing complexity of our economic system" that makes it impossible for the individual to take care of himself. Mr. Schoeck states: "Now it is difficult to imagine quite how a government agency in Washington, being responsible for the lifelong security and welfare of some 165 million citizens, should be better able to cope with 'complexities' than each family, community, and private enterprise insurance company. This would assume the existence therein of a divine mind, above and unlike the rest of us. And indeed the Report seems to envision just such a divine fatherly agency to cope with the dragon of complexity. For it goes on to declare that 'A security program, wisely conceived and fairly administered, can, indeed, provide the necessary economic foundation for high national morale.' "

Mr. Schoeck suggests a way for government and legislatures to truly help people in building up their own individual security program: It is full and unlimited deduction from taxable income of all premiums paid for health, income, life and annuity policies. In conclusion, he says:

> "I should like to add a question for those who applaud the maximization of 'social security' by compulsory coverage of even the well-to-do groups who don't need it. Did it ever occur to them that our left-wingers and radical equalitarians will have a much better argument for demanding a strict equalization of income and wealth as soon as everybody has his equal 'social security' provided by the central government?"

MUNICIPAL BONDS

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come from other sources, based on rates under present tax laws:

	Taxable Equivalent Yields Income Brackets—Thousands of Dollars			
Tax-Exempt Yields	10 to 12 38%	20 to 22 56%	26 to 32 62%	44 to 50 72%
2.25%	3.63%	5.11%	5.92%	8.04%
2.50%	4.03%	5.68%	6.58%	8.93%
2.75%	4.44%	6.25%	7.24%	9.82%
3.00%	4.84%	6.83%	7.89%	10.71%

Figures at the top of columns are the net taxable income brackets and the combined normal and top surtax rates applying to such brackets. The rates shown are applicable to taxpayers filing single returns. To find the taxable equivalent yield for taxpayers filing joint returns, divide the entire surtax net income by two. Further benefit accrues to the resident holder of Minnesota municipal bonds by exemption from our own state income tax.

At all times, it is well to remember that the eventual return *of* your money is more important than the return *on* it.

Bernard B. Knopp
The First National Bank of Saint Paul

Public Health

THE MINNESOTA PROGRAM ON ALCOHOLISM

Physicians have long realized that chronic alcoholism is primarily a medical rather than a social or moral problem. Minnesota's first health officer, Dr. Charles N. Hewitt, recognized the seriousness of the problem and the futility of the methods then used in dealing with the alcoholic. In his report to the State Legislature in 1872, Dr. Hewitt urged that alcoholism be recognized as a disease and that other means than fines and jail sentences be provided for its treatment.

Chronic alcoholism is also a public health problem by virtue of its "social magnitude, its essentially medical nature, and the fact that promising practical scientific methods of attack are now available."*

About 80,000 Minnesota residents are among the four million Americans directly affected by alcoholism. But alcoholism is a progressive condition that takes from ten to fifteen years to develop, the vast majority of alcoholics are between thirty and fifty-five years of age—a tremendously significant period in terms of the family, occupation, and social responsibility.

Although the Willmar State Hospital has for many years been designated as the treatment center for alcoholics in Minnesota, it was not until 1951 that the State Legislature formally recognized alcoholism as a medical and public health problem. A special commission was appointed in that year to study the matter. As a result of the findings of that commission, the Legislature in 1953 established a program to combat alcoholism.

This program centers in the Minnesota Department of Health. It is directed by a nine-member Advisory board. Goal of the program is the prevention of alcoholism, and emphasis is on the need for the early recognition and treatment of alcoholism, as in programs for the prevention of cancer. tuberculosis, mental illness, and other chronic diseases. The stigma currently attached to alcoholism frequently results in the concealment of the alcoholic by his family, friends, and employer. Public acceptance of the concept of alcoholism as a disease and the need for early recognition and treatment will do much to bring alcoholism into the open so that the alcoholic can receive the treatment he needs. As with any of the other chronic diseases, the earlier treatment is established the better are the patient's chances for recovery.

The Minnesota program has two major aspects—education of the general public, and provision of information to professional groups whose activities are likely to bring them into contact with the alcoholic or his family. Mass informational materials have been developed that are available to interested citizens without cost. These materials include a comprehensive bibliography of pamphlets suitable for the lay reader, several selected films that may be loaned to groups, and a radio series entitled "The Lonesome Road" which is now being used over broadcasting stations throughout Minnesota. Speakers are also available for talks to community groups.

Because health department personnel can reach directly only a very few persons affected by alcoholism, a special effort is made to work through physicians and other professional people who are likely to be consulted by families. Conferences have been held that have brought together representatives of several professional groups to discuss alcoholism. Because of the interest recently shown, it is hoped that increasing attention will be given to alcoholism by the various professional groups concerned with the problem.

Although the program described here is entirely educational, the need for improved and expanded treatment facilities for alcoholism is also recognized. Without adequate measures for successful treatment, the educational program is largely ineffective. The Willmar State Hospital returns over 50 per cent of its alcoholic patients to their communities either fully recovered or improved. With an adequate number of professional workers, this percentage could be increased significantly.

Also needed is a facility for women alcoholics. Despite a ratio of one woman to every five men alcoholics, treatment facilities have not been established in that proportion.

Although much remains to be done, great strides have been made in meeting the problem of alcoholism. With a progressively enlightened public opinion, progress will be accelerated.

NETTA W. WILSON
Senior Health Information Consultant,
Section of Public Health Education,
Minnesota Department of Health

**American Journal of Public Health,* Dec. 12, 1947.

In Memoriam

THEODORE L. CHAPMAN

Dr. Theodore L. Chapman, seventy-six, a leader in medicine for many years in northeastern Minnesota, and one of the founders of the Duluth Clinic, died September 23, 1954, in a Duluth hospital.

Dr. Chapman had practiced general surgery for nearly fifty years in Duluth before moving to Barnum at the time of his retirement a few years ago. He was born in Orange, Connecticut, received his preliminary education in Detroit and his medical education at Wayne Universty in Detroit. He served an internship and externship for two years at Harper Hospital there and then settled permanently in Duluth.

He was a member of the American College of Surgeons, the St. Louis County Medical Society, the Minnesota State Medical Association and the American Medical Association. He became a member of the Fifty club of the State Association in 1953. He was also a member of the Inter-Urban Academy of Medicine, the Duluth Athletic club and the Gold Island club. He served with Michigan's 33rd infantry battalion in Cuba during the Spanish American War and with the Army Medical corps as a first lieutenant in World War I.

His wife, Catherine, of Barnum, survives him; also, a son, Richard, Red Lodge, Montana; a daughter, Mrs. E. A. Wilkenson, Oakland, California; two brothers, Charles and Edward, Detroit; two sisters, Mrs. H. W. Crowell, Detroit, and Mrs. Alexander Harning, New York City, and four grandchildren.

MICHAEL F. HAYES

Dr. Michael F. Hayes died September 10, 1954, at Moose Lake Hospital. He was seventy-one years old, and had practiced at Nashwauk in Itasca county for nearly forty years.

Dr. Hayes was born in Fillmore county and went to school in Lanesboro. He received his Bachelor of Science and medical degrees from the University of Minnesota and served his internship at Minneapolis General Hospital.

After two years of practice at Lily, South Dakota, Dr. Hayes moved to Nashwauk where he remained until his retirement early this year. He was a member of the St. Louis County Medical Society, the Minnesota State Medical Association and the American Medical Association.

HENRY LUSSAN LAZAR

Dr. H. L. Lazar, Minneapolis urologist who retired from active practice in 1945, was found dead in Lake Minnetonka near the fishing dock at his home at Excelsior, October 13, 1954. According to the Hennepin county coroner's office, Dr. Lazar probably suffered a heart attack before he fell into the water. He had been fishing most of the afternoon and the drowning was not discovered until he failed to return home for dinner, about 5:00 p.m.

Dr. Lazar was born in New Orleans in 1881 and went to school at Magnolia, Mississippi, and at Tulane University, from which he received his medical degree in 1905. He took postgraduate training at the New York Post-Graduate College and was licensed to practice in Minnesota in 1924. He established himself in Minneapolis and practiced urology there until 1945 when a coronary thrombosis forced him to retire. He was an associate member of the Hennepin County Medical Society, the Minnesota State Medical Association and the American Medical Association.

His wife, the former Edna Leighton, survives him.

OWEN W. PARKER

The passing of Dr. O. W. Parker, at the age of eighty, calls for some special comment, as does that of every physician who for over half a century has been able to serve his people in his profession faithfully and unstintingly. A personal incentive obtains with this writer because, from my earliest years in Duluth, all his activities were so well known to me; and in addition, over a decade ago, he asked me (should I survive him) to write his obituary for this, our State *Journal*. Dr. Parker died Friday, September 17, 1954, at his home in St. Louis Park.

Owen Parker's life span covered that period of Minnesota's development in which this northeast section burst into ever-expanding action in terms of iron mining. Born at Beaver Dam, Wisconsin (1874), he finished his medical training at the University of Minnesota just at the turn of the century (1900). His year of internship in St. Luke's Hospital, St. Paul, brought him in touch with such inspiring surgeons as McLaren, O'Brien, Rogers and men of their type. It was an inspiring contact and it showed its influence through his long life of service in St. Louis County. Following his internship, he joined with the late Dr. Charles Shipman, in Ely, where some of the earliest deep mines began to employ that amazing conglomerate of European immigrants and where the tussle to protect them from injury and safeguard their health called for doctors with unusual courage, stability and sound medical judgment. These qualities were just what that young doctor and his charming wife had, and between them, they made a great health center out of the Shipman Hospital.

Doctor Parker made an early association with the late Dr. George Ayers, and later on, they were joined by a succession of competent men, including Dr. Harry N. Sutherland, thus maintaining a good outpost of medical service ever since. This type of total service to the community stemmed, in no small degree, from the self-sacrifice and sense of duty to further all the essential health activities, not only as a public official, but in leading the way in all movements where opinion and support need this personal guidance, for which Doctor Parker was eminently fitted.

To those of us who for years felt the glow of his friendship and comradeship, he was always there to listen appreciatively at all our meetings, although loathe to offer comment himself. He and Mrs. Parker (her death within the past year greatly depressed him) traveled widely. He reported his observations from such travel studies and contacts and took as a hobby, sorting out the details concerning the events leading up to (and what might be called the clinical charts pertaining) to the deaths of our martyred presidents. He did this most conspicuously in terms of the assassination of Abraham Lincoln, making intimate studies in and about Washington, D. C., for details.

After 1942, he moved to Duluth and was associated with us here at the Duluth Clinic for a period of two years during the stressfulness of World War II. He later helped out the medical staff at the Moose Lake Hospital. There he served faithfully, as usual, lending the charm, dignity and a sense of charitable motivation that his family and host of friends shall long treasure.

E. L. Tuohy, M.D.

DANA C. ROOD

Dr. Dana C. Rood, leader in medicine for many years on Minnesota's Iron Range, died at the age of ninety in Santa Barbara, California, October 6, 1954.

Dr. Rood was founder of Rood Hospital, the first major hospital on the Iron Range. It was located in Hibbing and is now today's Hibbing General Hospital. He practiced medicine for fifty years in Hibbing until he retired in 1946 and he was a member of the Fifty Club of the Minnesota State Medical Association. He was also a member of the St. Louis County Medical Society and the American Medical Association. He moved to California after his retirement and remained there until his death.

Survivors include his wife, Elizabeth; a daughter, Mrs. Marcia Rood Dwyer; a son, Dana C., Jr., and a granddaughter, Mrs. Lynn Davidson Hewitt.

RASMUS VALLIN WILLIAMS

Dr. R. V. Williams of Rushford, a life member of the Minnesota State Medical Association and a member of the Association's Fifty club, died October 16, 1954, in Methodist Hospital, Rochester. He was seventy-seven years old and, though he had moved to Rochester three years ago, he continued to go back to Rushford a few days each week to see patients.

Long active in Rushford community affairs, one of his last trips to Rushford was on July 3 when he sat on the reviewing stand for the Rushford Centennial parade.

He had served as health officer for a number of years and as member of the school board. He was also active in county medical society affairs and in the work of the Minnesota State Medical Association, having served for many years on the Committee on State Health Relations. He also served as vice president and then president of the Southern Minnesota Medical Association.

Dr. Williams was born in Lee county in Illinois and attended high school and college at Shabbona, Illinois. He received his medical education at Northwestern University and served an eighteen months' internship at Northwestern University, with ten months' postgraduate training at the University of Vienna in 1909-10. He practiced for one year in La Crosse, Wisconsin, and moved to Rushford in 1905.

Survivors are his wife, the former Mrs. Helen Smith Merriam; a daughter, Mrs. Judith Long of Rockford, Illinois; and a grandson, Roger Long, now serving in the U. S. Army at Fort Devens, Massachusetts.

FRACTURE OF THE SPINAL COLUMN AND PELVIS

(Continued from Page 807)

marked displacement of bone may produce extensive damage to the membranous urethra, rupture of the bladder, or both.

On admission, having established that there is a fracture of the pubic portion of the pelvic ring, one should be concerned at once about the condition of the urinary tract. If the patient voids clear urine there probably has been no damage done; if he cannot void, an attempt should be made to pass a catheter into the bladder. Bleeding from an injury of the bladder should stop in 6 to 10 hours; if it persists beyond this period, one should suspect injury higher in the urinary tract. Inability to pass the catheter usually denotes some damage to the urethra or bladder. If the general condition of the patient is satisfactory, one may temporize for a few hours to see if the bladder can be emptied voluntarily. When this cannot be done, the services of a urologic surgeon should be sought, for one must then open the abdomen and deal directly with whatever injury is found. At times it may be necessary to reduce and immobilize the pelvic bony injury before continuity of the urinary tract can be re-established.

References

1. Ivins, J. C.: Compression fractures of the spinal column. Minnesota Med., 33:154-156 (Feb.) 1950.
2. Watson-Jones, Reginald: Fractures and Joint Injuries. Ed. 3, vol. 1, 407 pp.. Baltimore: The Williams and Wilkins Company, 1943.

◆ Reports and Announcements ◆

MEDICAL MEETINGS

State

MINNESOTA STATE MEDICAL ASSOCIATION, Annual meeting, Minneapolis, May 23-25, 1955.

Physicians and Schools, First conference, University of Minnesota Center for Continuation Study, Minneapolis, December 2-3, 1954.

Sectional

American College of Surgeons, Sectional meeting, The Fort Harry, Manitoba, Canada, April 25-26, 1955.

National

American Academy of Dermatology and Syphilology, Thirteenth annual meeting, Palmer House, Chicago, December 4-9, 1954.

American Congress on Obstetrics and Gynecology, Sixth annual meeting, Palmer House, Chicago, December 13-17, 1954.

INTERNATIONAL ACADEMY OF PROCTOLOGY 1954-55 AWARD CONTEST

The International Academy of Proctology announces its annual cash prize and certificate of merit award contest for 1954-55. The best unpublished contribution on proctology or allied subjects will be awarded $100 and a certificate of merit. Certificates will be awarded also to physicians whose entries are deemed of unusual merit. This competition is open to all physicians in all countries, whether or not affiliated with the International Academy of Proctology. The winning contribution will be selected by a board of impartial judges, and all decisions are final.

The formal award of the First Prize, and presentation of other Certificates, will be made at the Annual Convention Dinner Dance of the Academy, March 26, 1955, at The Plaza Hotel, New York.

All entries are limited to 5,000 words, must be typewritten in English, and submitted in five copies. All entries must be received not later than the first day of February, 1955. Entries should be addressed to the International Academy of Proctology, 147-41 Sanford Avenue, Flushing 55, N. Y.

INTERNATIONAL ACADEMY OF PROCTOLOGY SEMINAR

Plan now to attend the Seventh annual convention of the International Academy of Proctology at the Plaza Hotel, New York City, March 23 to 26, 1955. The International, National, and Local Program committees are planning an unusual seminar on anorectal and colon surgery. There will be special emphasis on anorectal presentations, and on panel discussions, as requested by those who attended the Chicago meeting in 1954.

Plans are being developed for wet clinics and lectures at the Jersey City Medical Center under the dirction of Dr. Earl Halligan, surgeon-in-chief of the Medical Center.

Eminent speakers from all parts of the country and abroad will present interesting papers and motion picture demonstrations of their personal techniques. Mexico is expected to be very well represented at this meeting.

The delegates and trustees, and their wives, will have cocktails and dinner on Tuesday evening, March 22, 1955, the evening before the official opening of the scientific activities of the convention. Both members and non-members of the Academy, and their wives, should plan to attend the Saturday night, March 26, banquet.

The Women's Auxiliary has planned a very unusual program for the wives of the members and their guests.

Please remember that all physicians and their wives may attend the annual conventions of the International Academy of Proctology, whether or not they are affiliated with the Academy. There is no fee for attendance at these teaching sessions of the Academy.

THE NEW ORLEANS GRADUATE MEDICAL ASSEMBLY

The eighteenth annual meeting of The New Orleans Graduate Medical Assembly will be held March 7-10, 1955, with headquarters at the Municipal Auditorium.

Eighteen outstanding guest speakers will participate, and their presentations will be of interest to both specialists and general practitioners. The program will include fifty-four informative discussions on many topics of current medical interest, in addition to clinicopathologic conferences, symposia, medical motion pictures, scentific exhibits, round-table luncheons and technical exhibits.

The Assembly has planned another interesting post-clinical tour to follow the 1955 meeting in New Orleans. On Saturday, March 12, a party composed of doctors and their families will leave New York for Europe via Pan American World Airways "President Special." The itinerary includes France, Italy, Germany, Denmark, Sweden and England, and the arrangements have been made for medical programs in these countries. The tour ends in England and the group will return to New York, sailing April 7 from Southampton on the *S. S. Liberte,* or by Pan American World Airways.

Details of the New Orleans meeting and the post-clinical tour are available at the office of the Assembly, Room 103, 1430 Tulane Avenue, New Orleans 12, Louisiana.

ACADEMY TO MEET IN CHICAGO

The thirteenth annual meeting of the American Academy of Dermatology and Syphilology will be held December 4 to 9 in the Palmer House, Chicago.

Two new round-table panels will be added this year, one on "Cutaneous Testing" and the other on "Hereditary Dermatoses."

Seven special courses will be held the first two days at Northwestern University, the University of Illinois College of Medicine, in the Palmer house, and at Billings Hospital of the University of Chicago. The courses will include histopathology, mycology, X-ray and radium therapy, bacteriology and virology of the skin, anatomy and embryology, and special problems in dermatohistopathology.

Special lectures will be given by Dr. Arthur C. Curtis, professor of dermatology and syphilology at the University of Michigan, on "Essential Familial Hypercholesterolemia and the Secondary Xanthomas"; Dr. Sidney Farbero, professor of pathology at Harvard Medical School and director of research of Children's Cancer Research Foundation at Boston, who will discuss "Lipoidoses in Children."

Other special lectures will be delivered by Dr. Frederick Urbach, associate chief, cancer research (Dermatology), Roswell Park Memorial Institute, Buffalo, N. Y., on "Treatment of Xanthomas"; and another by Dr. Farber on "Biological and Chemical Approaches to the Treatment of Cancer." Fifteen symposia and 12 round-table panels are also scheduled.

INDUSTRIAL HEALTH INSTITUTE

The Institute of Industrial Health of the University of Cincinnati has announced that it will accept applications for Fellowships offered to qualified candidates wishing to pursue a graduate course in preparation for the practice of Industrial Medicine. Any registered physician who is a graduate of a Class A medical school, and who has satisfactorily completed at least two years of training in an AMA-accredited hospital may qualify. The course of instruction consists of a two-year period of intensive Industrial Medicine training, followed by a year of practical experience. Candidates will receive a degree of Doctor of Science in Industrial Medicine. Requests for additional information should be addressed to the Institute of Industrial Health, College of Medicine, Eden and Bethesda, Cincinnati 19, Ohio.

ANNUAL CONGRESS ON OBSTETRICS AND GYNECOLOGY

Every phase of maternal and newborn care will be covered at the Sixth American Congress on Obstetrics and Gynecology, to be held in the Palmer House, Chicago, December 13-17.

The five-day meeting, which is expected to draw an attendance of about 3,500, will be under the sponsorship of The American Committee on Maternal Welfare, Inc., and The American Academy of Obstetrics and Gynecology. Welcomes will be extended at the opening session, Monday, December 13, by Fred H. Falls, M.D., Chicago, president of the American Committee on Maternal Welfare, and Bayard Carter, M.D., of Durham, N. C., president of the American Academy of Obstetrics and Gynecology.

Philip F. Williams, M.D., former professor of clinical obstetrics and gynecology at the University of Pennsylvania Graduate School of Medicine, Philadelphia, and honorary general chairman of the congress, will speak in retrospect. R. Gordon Douglas, M.D., professor of obstetrics and gynecology at Cornell University Medical College and obstetrician and gynecologist-in-chief at New York Hospital, will look into the future.

Among the panel topics will be: "Indications for Hysterectomy," "Factors Determining Type of Cesarean Section," "Function of Personnel and Standards of Procedure in the Delivery Room," "Management of Prolonged Labor," "Blood Transfusions," "Preparation for Parenthood," "Consultation in Obstetrics," "Community Aspects of the Care of the Cancer Patient," "Obstetric Anesthesia," "Selection, Training, and Function of Obstetric Personnel," "Perinatal Mortality," "Organization and Operation of Maternal Mortality Committees," "Lesions to be Considered in the Differential Diagnosis of Carcinoma of the Cervix," "Carcinoma of the Cervix," "Toxemias of Pregnancy," "Obstetric Hemorrhage" and "Prenatal Care."

Symposia subjects will include: "Medical Complications of Pregnancy," "Acceditation of Hospitals and Residencies," "Basic and Graduate Education and Training of Obstetric Nurses and Supervisors," "Premarital Examinations and Counselling," and "Prevention and Management of Prematurity."

FLORIDA SEMINAR PLANNED

The Ninth annual University of Florida Midwinter seminar in Ophthalmology and Otolaryngology will be held at the Sans Souci Hotel in Miami Beach the week of January 17, 1954. The lectures on ophthalmology will be presented on January 17-19, and those on otolaryngology on January 20-22. A midweek feature will be the midwinter convention of the Florida Society of Ophthalmology and Otolayngology on Wednesday afternoon, January 19, to which all registrants are invited. The registrants and their wives may also attend the informal banquet at 8 p.m. on Wednesday. The Seminar schedule permits ample time for recreation.

The Seminar lecturers on ophthalmology this year are: Dr. William F. Hughes, Jr., Chicago; Dr. Phillips Thygeson, San Jose; Dr. James Allen, New Orleans; Dr. Walter H. Fink, Minneapolis, and Dr. Milton L. Berliner, New York. Those lecturing on otolaryngology are: Dr. Paul Holinger, Chicago; Dr. Lawrence R. Boies, Minneapolis; Dr. Edmund P. Fowler, Jr., New York; Dr. Arthur W. Proetz, St. Louis, and Dr. David D. DeWeese, Portland, Oregon.

TUBERCULOSIS AUTHORITIES SPEAK

Dr. Howard M. Payne, professor of medicine at Howard University, Washington, D. C., and Dr. James J. Waring, professor emeritus of medicine at the University of Colorado and director of the Colorado foundation for TB Research, were principal speakers at a three-day tuberculosis study course at the University of Minnesota Center for Continuation Study.

The men spoke at the Minnesota Tuberculosis and Health Association's dinner meeting at the University, which highlighted the ninth annual tuberculosis course for lay workers. Dr. Payne was a featured speaker at the 50th Anniversary meeting of the NTA in Atlantic City, and Dr. Waring received the Trudeau Medal for distinguished service from the NTA in 1953. The course was attended by county volunteer tuberculosis campaign workers and consisted of lectures by University faculty members, health officials and other tuberculosis authorities.

CONTINUATION COURSES

The University of Minnesota will present a continuation course in Pediatrics for General Physicians January 6-8, 1955, at the Center for Continuation Study. The program will stress the recognition and management of the many and varied urinary tract problems seen in youngsters. Guest speaker will be Dr. Henry Barnett, associate professor, Department of Pediatrics, Cornell University Medical School, an outstanding authority in this field. The program will be presented under the direction of Dr. Irvine McQuarrie, professor and head, Department of Pediatrics, University of Minnesota Medical School.

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Continuation Courses in *Ophthalmology* and *Otolaryngology* for Specialists in these fields will be held at the Center for Continuation Study on the University of Minnesota campus during the week of January 31 to February 4, 1955. Physicians may register for one or both courses; those registering for one only will have a portion of each day free for attending hospital clinics, visiting the new Mayo Memorial, etc. Guest speakers in ophthalmology will be Dr. P. J. Leinfelder, Professor of Ophthalmology, Iowa State University, Iowa City, Iowa, and Dr. Frank Newell, Associate Professor of Ophthalmology, Northwestern University Medical School, Chicago, Illinois. This course will be presented under the direction of Dr. Erling W. Hansen, Clinical Professor and Director, Division of Ophthalmology, University of Minnesota Medical School. Guests in otolaryngology include Dr. Howard House, Associate Professor of Otolaryngology, University of Southern California, Los Angeles, California, and Dr. Peter Pastore, Professor of Otolaryngology, Rhinology, and Laryngology, University of Virginia Medical College, Richmond, Virginia, and Dr. L. R. Boies, Professor of Otolaryngology and Director of Division of Otolaryngology, University of Minnesota Medical School, will direct this course.

DOCTORS, NEWSPAPERS HONORED

Distinguished service in the cause of tuberculosis control was recognized when five Minnesota physicians and 10 newspapers in St. Louis County were honored for having aided in the TB campaign for fifty or more years.

The ceremonies took place at the annual Iron Range meeting of Christmas Seal workers Virginia. The meeting was one of a number being held in commemoration of the fiftieth anniversary of the voluntary TB movement in the United States. The five doctors whose work against TB was cited were Dr. E. L. Touhy, Dr. A. T. Laird, Dr. A. L. McDonald and Dr. W. R. Bagley, all of Duluth, and Dr. R. P. Pearsall of Virginia.

Guest speaker at the meeting was Dr. S. Marx White of Minneapolis, who was introduced by Dr. Walter S. Neff of Virginia. Dr. White detailed the fifty-year history of the organized battle against tuberculosis and urged for increased interest in future projects.

Newspapers honored at the banquet meeting were the *Duluth News-Tribune, Duluth Herald, Duluth Scandinav, Mesabi Daily News, Labor World, Ely Miner, Hibbing Tribune, Tower News, Chisholm Tribune-Herald,* and *Eveleth News-Clarion.*

AMEF STATE CHAIRMEN TO MEET

The fourth annual meeting of the American Medical Education Foundation state chairmen will be held at the Sheraton Hotel in Chicago on Sunday, January 23, 1955. Specific details regarding the proposed program and reservation forms for accommodations at the Sheraton Hotel will be distributed with the November Bulletin to AMEF state chairmen and regional auxiliary chairmen.

John W. Hedback recently joined the staff of the Foundation in the capacity of associate executive secretary. Through many years of hospital public and community relations work, Mr. Hedback has gained valuable experience which will assist him in the development of state and local committees. The son of a physician, the late Axel E. Hedback, M.D., of Minneapolis, he has a keen interest in the problems of medical education.

AMERICAN BOARD OF PHYSICAL MEDICINE AND REHABILITATION

The next examinations for the American Board of Physical Medicine and Rehabilitation will be held in Philadelphia, June 5 and 6, 1955. The final date for filing applications is March 1, 1955. Applications for eligibility to the examinations should be mailed to the secretary, Dr. Earl C. Elkins, 30 N. Michigan Ave., Chicago 2, Illinois.

AMA SETS DEADLINE FOR PAPERS

The Council on Scientific Assembly announces the deadline for those who wish to participate in the Atlantic City Meeting, either by reading a paper or presenting a scientific exhibit. The deadline for section papers is December 15, 1954, and the deadline for scientific exhibits will be January 10, 1955.

Applicants should communicate with the Secretary or the Representative to the Scientific Exhibit of the Section in which they are interested. Further information may be obtained from the Secretary, Council on Scientific Assembly, American Medical Association, 535 North Dearborn St., Chicago 10, Ill.

Woman's Auxiliary

CIVIL DEFENSE AND THE AUXILIARY

The term "Civil Defense" to most of us is without specific meaning. It involves so much, and is so vague, that we find it easier to act like the ostrich. We shut our eyes and trust there will never be any need for it. And we do nothing.

All of us are familiar with insurance which protects us in enumerable ways—life, accident, illness, old age, fire, wind, theft—almost anything you may want, and we are willing to pay money for it. Civil Defense provides protection for us, also, and for our property, in the event of natural or enemy-inflicted disaster. This we do not have to buy. Instead, its effectiveness for our protection depends upon individual participation and work in helping to set up the program.

The Civil Defense director has an organized plan for all of us but without workers, and as one of them recently stated, "a director is as helpless as a football coach without players." The director needs the aid and co-operation of many, many citizens.

Auxiliary Can Help

As Auxiliary members we could do much to help. We represent a unique group of women who are already acquainted with medical and public health activities—and we have the time to help, too, if it is our will to do so.

We do not neglect to insure our homes against fire because we regard fire insurance as a necessity. Civil Defense is a fundamental necessity, too. Perhaps we evade our responsibility because we know so little about the program or because it seems so colossal, so overwhelming that we are unwilling to participate. Step-by-step training in a concentrated course of Civil Defense instruction might enable us to see the integral parts which make up the whole program and show us how to fit ourselves into the situation.

Our husbands know definitely what their roles in Civil Defense are going to be. Now, what about us wives? Our problem will differ according to where we live, as we integrate ourselves into the local picture, but individually it will be the same,—the individual desire for the safety and survival of our immediate families in case of disaster.

With this in mind, let's begin by setting up our own defenses at home. Let's obtain adequate knowledge about exactly what each person is expected to do in case of emergency. Let's get suitable training, if necessary, and thereby provide some assurance of survival and recovery for ourselves should the need arise. A possible study group program might include the following:

1. General preliminary study presenting information about various phases of Civil Defense.
2. Essentials of nutrition for survival; food preparation with limited facilities (no gas, electricity or safe water supplies); essential family food kits or emergency closets.
3. Shelter in diaster with particular attention to sanitation.
4. Essentials in home nursing.
5. Simple first aid training.

Training For Defense

After training for family self-preservation, it will be much easier to integrate ourselves into neighborhood and community Civil Defense, either by virtue of skills we are already trained for, or by attending classes in whatever we may be most interested in, such as practical nursing, nurses aide training, day nursery and child care, first aid, hospital aides of all sorts, clerical work, welfare and social service, air force observation, amateur radio operation, telephone operation, fire fighting. The list is endless.

Civil Defense is truly enormous in its scope. But it needs our support and co-operation to make it function.

This is a challenge!

What are you going to do about it?

Mrs. L. R. Boies, *President-Elect,*
Hennepin County Medical Society Auxiliary

RAMSEY AUXILIARY OPENS FALL SEASON

Activities of the Ramsey County Medical Society Auxiliary for October included a board meeting, the regular Auxiliary luncheon and attendance at the Minnesota Tuberculosis and Health Association annual dinner by a large number of members.

The board meeting was held October 25, with Mrs. W. P. Gardner, president of the organization, presiding. Board members assisted the cancer committee with the making of surgical dressings during the meeting. At the same time, the medical and surgical relief committee of the auxiliary reported packing sixteen boxes of medical supplies during September.

The luncheon meeting, first of the season, was held on October 25 at the Saint Paul College Club. F. Manley Brist, legal advisor of the Minnesota State Medical Association, was the speaker.

James J. Waring, M.D., emeritus professor of medicine, University of Colorado Medical Center, Denver, and Howard M. Payne, M.D., professor of medicine at Howard University, Washington, D. C., were principal speakers at the Tuberculosis and Health Association dinner attended by many members who have cooperated for many years in the program of the association.

Of General Interest

Dr. Walter A. Fansler, Minneapolis, presented the Annual Beaumont Oration at the 63rd meeting of the Michigan State Medical Society in Detroit. The subject of his address was "Indications for Roentgenological Studies of the Colon and Significance of Roentgenological Findings." Dr. Fansler also presented a paper on the topic "The Cause and Prevention of Unsatisfactory Results Following Ano-Rectal Surgery."

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Three Hopkins physicians, **Drs. R. H. Picha, J. J. Hersman** and **E. W. Chernick,** have purchased a residence in Hopkins and will build a clinic. **Dr. H. E. Drill,** also of Hopkins, plans to build a clinic.

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Improved hospital care for the aged was the subject **Dr. Charles W. Mayo,** Rochester, spoke on at a convention meeting at the 25th anniversary of Passavant Memorial Hospital, Chicago. Dr. Mayo talked on activities of the United Nations at a recent meeting of the Women's City Club, St. Paul.

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Dr. Milton Kaiser of New Ulm has been licensed as a lay preacher in the Methodist church. Dr. Kaiser received the license at the annual meeting of Methodist church officers and pastors of the Mankato District. Among those whose licenses to preach were renewed at the session was **Dr. George Reineke,** also of New Ulm.

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Dr. Charles A. Aling has been installed as president of the Hennepin County Medical Society, succeeding **Dr. John H. Moe,** who became chairman of the Board of Directors. Other officers are **Dr. Willis L. Herbert,** first vice president; **Dr. James A. Blake,** second vice president; **Dr. Conrad J. Holmberg,** secretary-treasurer, and **Dr. Tague C. Chisholm,** librarian. Board members include **Drs. Robert E. Priest, L. Haynes Fowler, Ralph H. Creighton, Thomas J. Kinsella, Leo C. Culligan** and **Silas C. Anderson,** with the officers and **Dr. Horatio B. Sweetser,** sixth district councilor of the Minnesota State Medical Association.

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Dr. L. McKendree Eaton of the Mayo Clinic has accepted a membership on the medical advisory board of the Myasthenia Gravis Foundation, Inc., New York. The group studies the muscular diseases, myasthenia gravis.

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A former Albert Lean, **Dr. Kenneth Hodges,** and his bride will make their home in Ancon, Panama, after a wedding trip in the East. Dr. Hodges' bride is the former Margaret Joan McKenzie, a member of the University Hospitals nursing staff. Dr. Hodges, a University of Minnesota Medical School graduate, is interning at Gorgas Hospital.

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Speakers at an October business meeting of the Wabasha County Medical Society were **Dr. C. G. Ochsner** of Wabasha, and **Drs. J. M. Janes** and **E. C. Burke** of the Mayo Clinic, Rochester. The meeting was held in Lake City. Auxiliary members were entertained by Mrs. E. C. Bayley.

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Back from a stay in Oak Ridge, Tennessee, is **Dr. Leslie Zieve,** director of the radioisotope unit at the Veterans Administration Hospital and associate professor of medicine at the University of Minnesota. Dr. Zieve studied techniques of using radioisotopes in biochemistry.

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Dr. D. M. O'Donnell of Ortonville was specially honored by residents of his home town recently when "Dr. D. M. O'Donnell Day" was officially declared by Mayor Ed Martinson. Sponsored by hundreds of Ortonville friends, a television set, a testimonial presentation book signed by donors and a check were presented to Dr. O'Donnell, who celebrated his sixty-ninth birthday. Dr. O'Donnell has practiced in Ortonville forty years.

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New president of the Trudeau Society, medical branch of the Mississippi Valley Tuberculosis conference, is **Dr. E. P. K. Fenger,** Edina. Dr. Fenger was installed at the group's annual meeting in Kansas City, Kansas.

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Dr. E. W. Humphrey, founder of the Moorhead Clinic, recently observed his fiftieth year of practice there. Dr. Humphrey, who is seventy-five years old, is still engaged in active practice.

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Eighty-five new nurses and several teachers of the Swedish Hospital nursing staff were entertained recently by **Dr. and Mrs. H. S. Linner** of Red Cedar Point, Excelsior.

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Three antelope and three deer were bagged when two Blue Earth physicians, **Dr. R. H. Baker,** and **Dr. C. E. Wilson,** with **Dr. Arthur Bulbulian** of Rochester, hunted recently at Moorcroft, Wyoming.

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Dr. Paul Lober, pathologist at University Hospitals, participated in sessions of the International Pathologists convention in New York City.

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Dr. Donald D. Metz and his bride, the former Beverly Jean Briggs of St. Paul, are at home in Buffalo Lake after a wedding trip in the West. Dr. Metz, a medical school graduate of Marquette University, Milwaukee, took his internship at Ancker Hospital, St. Paul.

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Dr. W. F. Mercil of Crookston attended a meeting of the Central Obstetrical and Gynecological Association in St. Louis, Missouri.

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Dr. Walter C. Alvarez of Minneapolis was the principal speaker at the annual Community Health Day in Minneapolis. Sponsored by the Hennepin County Medical Auxiliary, the all-day program featured such speakers as **Dr. R. F. D. Johnson, Dr. C. W. Lillehei, Dr. Richard Varco, Dr. R. M. Elliot, Russell Nye, Dr. M. Elizabeth Craig, Dr. Leo Rigler** and **Dr. N. Kenneth Jensen. Mrs. Harold Benjamin,** Auxiliary president, welcomed visitors of the day's program, and **Thomas Cook** was master of ceremonies.

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Three Minnesota physicians have been named to enter the Mayo Foundation as Fellows. Majoring in medicine is **Dr. Donald C. Bell,** Minneapolis, and **Dr. Richard H. Lindquist** and **Dr. Robert B. Litin,** both of Minneapolis, are majoring in surgery.

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At home in Minneapolis are **Dr.** and **Mrs. Owen H. Wangensteen.** Mrs. Wangensteen is the former Sarah Ann Davidson of St. Paul. Dr. Wangensteen is a member of the faculty of the University of Minnesota School of Medicine.

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Dr. James Rogers Fox of the University of Minnesota has returned from Europe where, sponsored by the American Medical Association and the World Medical Association, he conducted a survey of medical facilities and payments plans in Great Britain, the Scandinavian countries and Switzerland. Dr. Fox also spoke to medical groups in Italy, Greece and Spain.

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Dr. F. R. Kotchevar of the East Range Clinic in Eveleth is making plans to open a new clinic in Aurora. Construction work on the building has begun.

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Three Duluth physicians were featured in a panel discussion before the St. Mary's Hospital Council on Volunteers. The men, who presented information and suggestions to help hospital volunteer workers in performance of their duties, were **Dr. R. O. Bergan, Dr. R. P. Buckley** and **Dr. E. A. Zupanc.**

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Dr. L. M. Randall and **Dr. S. W. Harrington,** both of Rochester, were speakers at the Pan-Pacific Surgical Association meeting in Honolulu, Hawaii.

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"The Value of Psychometrics in Medical Practice" was the title of a paper read by **Dr. Gordan R. Kamman,** St. Paul, before the regional meeting of the American College of Physicians in Indianapolis.

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Work of the Duluth Scottish Rite infant welfare clinics were outlined at an October dinner meeting of the Rite bodies by **Dr. N. E. A. Leppo** of Duluth.

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Chisholm teenagers are responsible for a new television set in the home of **Dr. A. D. Klein.** The TV set was presented Dr. Klein, member of the Chisholm board of education and community physician, by Chisholm Teen-Age Canteen officials.

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Dr. Jaime Paris and **Dr. Robert G. Siekert,** Rochester, have been appointed as consultants at the Mayo Clinic. Dr. Paris was a Mayo Foundation Fellow in internal medicine and Dr. Siekert was a Fellow in neurology and psychiatry.

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Dr. George Earl, St. Paul, was guest speaker when the Southwestern Minnesota Medical Society met at Worthington for its regular fall meeting. Dr. Earl's topic was "Economics in Medicine." Members of the Society Auxiliary were entertained at the home of **Dr. and Mrs. John Stam.**

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Dr. Frank H. Krusen of Rochester has been named to receive the 1954 Ambassador award of the Pennsylvania State Medical Society. The award was presented to Dr. Krusen during "Pennsylvania Week" in Philadelphia in October. Dr. Krusen heads the section on physical medicine at Mayo Clinic. He has been previously honored by both the State of Minnesota and by President Dwight Eisenhower for his contributions to the employment and welfare of handicapped persons.

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Dr. C. Walton Lillehei of the University of Minnesota's Department of Surgery discussed the diagnostic approach to the acute abdominal condition at one in a series of fall seminars at Slayton. The meeting of southwest Minnesota physicians was held at the Murray County Memorial Hospital.

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Promoted to the rank of professor and associate director of the Mayo Foundation for Medical Education and Research is **Dr. Raymond D. Pruitt.** A native of Wheaton, Dr. Pruitt will succeed **Dr. Kendall B. Corbin,** who is returning to scientific work on the foundation staff. Dr. Pruitt, a former Rhodes scholar, was a Fellow on the Foundation in 1940 and had been consulting physician of the Mayo Clinic since 1943. Dr. Pruitt's appointment was made by the University Board of Regents.

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Dr. Mario Fischer of Duluth, director of the Duluth Health department, participated in a panel discussion before the annual meeting of the American Public Health Association in Buffalo. With Dr. Fischer in the discussion of a cancer education project in Duluth were **Mrs. Betty Bond** and **Mrs. Marilyn Beyer** of his staff.

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Dr. Thomas M. Seery of the Austin Clinic recently attended a refresher course in internal medicine held at the Cook County Hospital in Chicago. Dr. Seery received a part of his internship at the Hospital.

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Dr. Howard M. Payne, professor of internal medicine at Howard University in Washington, D. C., spoke on the topic "Diagnosis and Therapy of Chronic Pulmonary Disease" at a joint meeting of the St. Louis County and Iron Range Medical Societies in Virginia. Dr. Payne was introduced by Dr. Reno W. Backus of Nopeming, chairman of the Tuberculosis committee of the Society.

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Dr. Russell Bagley and **Dr. John McNeil,** both of Minneapolis, have been elected as staff anesthesiologists to the medical staff of Northwestern Hospital, Minneapolis.

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Dr. James C. Masson, retired chief of surgery of the Mayo Clinic, was honored by about 20 of his former first assistants at a dinner given during the Alumni Association meeting at Rochester. Dr Masson, a native of Ontario, Canada, entered the Clinic in 1912 as an intern at St. Marys Hospital. He became chief of surgery in 1935.

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Speaker at the first 1954 general meeting of the Mount Sinai Hospital Auxiliary last month was **Dr. Lyle Hay,** chief surgeon at Mount Sinai. Dr. Hay's topic was "The Advantages to Our Hospital of a Teaching and Research Program."

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Dr. James A. Blake, Hopkins, has been given the merit award of the Minnesota Academy of General Practice. The citation pointed out the "extraordinary zeal and steadfast devotion" with which Dr. Blake has served the Academy. He is executive secretary of the group. Also honored by the Academy were **Drs. Robert Quello,** Minneapolis, **H. J. Jeronimus,** Duluth, **Paul M. Smith,** Lake Crystal and **B. A. Flesche,** Lake City, and **Dr. James A. Cosgriff, Sr.,** Olivia, president at the October meeting of the Academy. Chief luncheon speaker was **Dr. Albert M. Snell,** Palo Alto, California.

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Representing Minnesota as state president, **Dr. David P. Anderson,** of Austin, attended the annual conference of the American Cancer Society last month in New York. Dr. Anderson heads the Minnesota Division of the Society.

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Captain L. B. Kucera, former Faribault physician, has been named regimental surgeon for the 28th Infantry regiment stationed at Fort Carson, Colorado. Capt. Kucera is also commanding officer of the Medical company. He was taken into the Army in July.

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Honored by the Minneapolis committee of the Save the Children Federation was **Dr. Richard P. Saunders,** president of the federation. He recently returned from a survey trip to Europe.

Dr. Kenneth Buresh of Westbrook is now on active duty with the U. S. Army in Texas. Dr. Buresh will have a two-year tour of duty.

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Dr. Louis A. Buie, head of the Mayo Clinic section on proctology, was honored recently by about twenty-five former Mayo Foundation fellows who studied under him. The physicians' meeting was arranged by **Dr. Raymond J. Jackman** of Rochester and **Dr. Richard Hopping,** Newark, N. J. A special guest was **Dr. Buie's** son, **Louis, Jr.,** a fellow in general surgery, who entered the Air Force November 1. The men were members of the Foundation Alumni Association, which had its annual meeting in Rochester.

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Dr. David P. Anderson, Austin, has been elected president of the Minnesota division, American Cancer Society. Other officers elected are **Walter C. Briggs,** Minneapolis, first vice president; **Stanley Hawks,** Minneapolis, second vice president; **Dr. John J. Bittner,** Minneapolis, third vice president; **L. W. Hill, Jr.,** St. Paul, treasurer; **Mrs. R. D. Brubacher,** St. Paul, secretary; and **Mrs. Russell Hanson,** Benson, state commander and chairman of volunteer services.

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Dr. G. C. Kimmel of Red Wing attended a weeklong meeting last month of the American Academy of Pediatrics.

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A native of Ireland, **Dr. Harold J. Swan** of Minneapolis, has been awarded the first fellowship to be awarded by the Minnesota Heart Association. **Dr. Swan** will do heart and circulation research. **Dr. Charles N. Hensel,** president of the group, and **Dr. Maurice B. Visscher,** head of the department of physiology at the University of Minnesota, presented the award to **Dr. Swan.**

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Four Virginia physicians have been named as consultants to the medical staff of the International Falls Memorial hospital. The new medical appointees are **Dr. Harrison E. Law,** surgery; **Dr. Jack R. Pierce,** obstetrics and gynecology; **Dr. Walter S. Neff,** internal medicine, and **Dr. David A. Sher,** pediatrics.

* * *

Dr. John Stam of Worthington spoke on the topic "Children, Our Privilege and Responsibility" at a meeting of the County Public Health Nursing Service and guests at Westbrook recently. He was introduced by **Dr. Elmer Schutz** of Worthington, chairman of the Nursing Service board.

* * *

Dr. Merrill D. Chesler, Minneapolis, was speaker at the regional Seminar in Therapeutics held at Union Hospital, New Ulm, last month. The topics covered were "Treatment of Congenital and Acquired Deformities in Children" and "Useful Applications of Skin Grafts and Flaps." Cases were demonstrated with colored slides.

At home in St. Paul after a wedding trip through the western states are **Dr.** and **Mrs. William R. Jahnke.** Mrs. Jahnke is the former **Phyllis Jean Tucker** of St. Paul. The couple were married in House of Hope Presbyterian church.

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Dr. Thomas J. Dry, Rochester, talked on new trends in treatment of heart disease by surgery at a joint meeting in October of the St. Louis County Medical Society and the Duluth District Dental Society.

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Main speaker at a regional meeting of the Institute of Food Technologists at Ames, Iowa, was **Dr. Ralph Holman,** associate professor of physiological chemistry at the Hormel Institute, Austin. His topic was "Essential Fatty Acid Deficiency and Metabolism."

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Dr. Neil R. Kippen of Breckenridge, attended the annual meeting of the Royal College of Surgeons at Winnipeg. Dr. Kippen received the degree of Fellow of the Royal College of Surgeons. He was admitted to the College in 1953.

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Dr. A. J. Lenarz of Browerville, county coroner, suffered a heart attack last month and was admitted to St. Joseph's Hospital in St. Paul. **Dr. M. E. Mosby** has temporarily taken over Dr. Lenarz' practice.

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Dr. Henry W. Meyerding of Rochester, a retired Mayo Clinic staff member, has been re-elected to the board of directors of the Minnesota Tuberculosis and Health Association.

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Nineteen cancer research projects will be conducted during the next year at the University of Minnesota as a result of grants announced totaling $140,750. Included in the nineteen are six new projects, which were granted to **Dr. Franz Halberg, Dr. Robert A. Good, Dr. David Mitchell, Dr. Lester L. Bissinger, Dr. F. John Lewis** and **Dr. James Hugh Casey.** Four awards to projects receiving first funds from the American Cancer Society were given to **Dr. K. W. Stenstrom, Dr. Claude Hitchcock, Dr. Donald Ferguson** and **Dr. Helmut Gutmann.**

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New editor of the journal *Current Researches in Anesthesia and Analgesia* is **Dr. T. H. Seldon,** Rochester. The appointment was made at a meeting of the board of trustees of the International Anesthesia Research Society in Cleveland.

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The 1929 Medical School class of the University of Minnesota has presented $2,900 to the alumni association for special projects. **Dr. Elmer Ceder** of Minneapolis, class member, made the presentation. Officers elected by the medical group are **Dr. William C. Bernstein,** St. Paul, president; **Dr. Byron Cochrane,** St. Paul, first vice president; **Dr. Virgil Lundquist,** Minneapolis, second vice president, and **Dr. Shelley Legaard,** Minneapolis, secretary-treasurer.

Dr. A. J. Wentworth of Mankato, president of the Blue Earth County Christmas seal organization, has been elected to the board of directors of the Minnesota Tuberculosis and Health Association. The election took place at the annual meeting in St. Paul.

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"The Medical Treatment of Hypertension" was the topic of **Dr. T. W. Parkin's** speech before the Southwestern Minnesota Medical Society last month. Dr. Parkin is a member of the Mayo Clinic's department of medicine.

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Dr. Charles W. Mayo presented the $10,000 Mutual of Omaha Criss award and gold medal to **W. Earl Hall,** editor of the *Mason City Globe-Gazette.* The presentation, which is made to the person or persons considered to have made the most outstanding contribution in the field of safety or health, was made at the annual meeting of the board of directors of the National Safety Council in Chicago. The award is given in honor of the late Dr. C. C. Criss, founder of the company.

* * *

Members of the White Bear Lake Area Council for Handicapped Children heard **Dr. Harold Flanagan,** St. Paul, at their meeting last month at White Bear Lake. Dr. Flanagan emphasized the social and emotional problems of handicapped children.

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Guest speaker at a workshop meeting of Christmas Seal workers from Todd, Morrison, Cass, Aitkin and Crow Wing counties was **Dr. Philip Soucheray** of St. Paul. The meeting was held in Brainerd.

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The principal cause of obesity in college students is eating, **Dr. William M. McConahey** of Rochester told a meeting of the North Central district of the American College Health Association. The sessions were held at Luther College in Decorah, Iowa.

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Dr. Dale C. Cameron, medical director of the Minnesota Department of Public Welfare, was luncheon speaker at a two-day meeting in Rochester of the Minnesota Association of Nursing Homes. Other speakers were **Glen Taylor,** executive secretary of the Minneapolis Hospital Association, **Dr. Viktor O. Wilson,** director of the Rochester-Olmsted County Health unit and **Dr. Gordon Martin,** consultant in physical medicine and rehabilitation at Mayo Clinic.

NEW LOCATIONS

Dr. Robert J. Meyer, who has practiced in Minneapolis for the past three years, has opened offices in Golden Valley. Dr. Meyer was graduated from the University of Minnesota Medical School in 1950.

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Dr. John T. Olive, Jr., has joined the staff at the Mankato Clinic. Dr. Olive, who has recently been discharged from the Army Medical Corps, was graduated from St. Louis University Medical School in 1948. He specialized in pediatrics at the University, graduating in 1953.

Dr. George W. Stahl, a former resident of Sheboygan, Wisconsin, and a graduate of the University of Wisconsin, has opened offices for the specialized practice of urology in Austin.

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Dr. Harold Kaplan, a graduate of the University of Minnesota, is now located in the Medical Arts Building, Minneapolis.

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Dr. Norman R. Haugen, who has been with the Bratholdt Clinic at Watertown, has joined the Crookston Clinic staff as general practitioner. Dr. Haugen received his medical degree from the University of Minnesota.

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Dr. George Smith, who has served in the Air Force the past twenty-one months, has returned to the United States from Iceland. His family has returned to the Smith home in Hutchinson. Dr. Smith, after his discharge, will resume practice with Dr. E. W. Lippmann and Dr. Dan Huebert.

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Dr. Celia A. McNeely has opened a practice in Herman, taking over for Dr. R. M. Watson, who is associated with the Arneson-Watson Clinic in Morris.

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Dr. V. A. Schulberg, a resident of Los Angeles, California, for the past two years, has returned to Arlington to join Dr. D. C. Olson in practice.

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Dr. O. M. Porter, who practiced in Atwater a number of years, has returned to Atwater after practicing in Willmar and Sisseton, South Dakota.

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Dr. N. Rysen has joined Dr. M. I. Hauge in his Clarkfield Clinic. Dr. Rysen, a graduate of the University of Tennessee College of Medicine, interned at Northwestern Hospital, and has been at Anoka State Hospital for 15 months.

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Dr. K. A. Law of Millheim, Pennsylvania, has become associated with the Wood Lake Clinic. He is a graduate of Jefferson Medical College, Philadelphia, and took his internship at Methodist Hospital in Philadelphia.

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Dr. David F. Struxness, who has been associated with the Alexandria clinic, has begun practicing at Glencoe. He is a graduate of the University of Minnesota Medical School, and served his internship at General Hospital.

MINNESOTA BLUE CROSS-BLUE SHIELD NEWS

The Blue Shield Professional Relations department is embarking on a more active and extensive campaign. This has been made possible by the employment of one additional person and the return to the organization of a former employe whose past training and activities include special qualifications for this work. With these two additional workers, it is planned that calls will be made upon doctors and upon their office nurses, receptionists and secretaries.

Personal service to the physicians and their office staffs is to be the keystone of the program as planned. Personal calls will be made to discuss Blue Shield's policies, practices and developments with the doctors. Information about claims can more effectively be disseminated and questions about them can better be answered by a visit than by a letter. This personal attention principle will be emphasized with both the doctors and their office personnel.

With this as a foundation for an active and energetic public relations program, features of professional relations programs which have been effective in other Blue Shield plans are to be explored and added. These include news releases, literature, and attendance of staff and medical meetings by Blue Shield representatives.

To assure effectiveness of the program, records are to be maintained of all suggestions made by both the physicians and members of their office staff. This, it is expected, will provide a means of molding the professional relations activities in whatever manner will serve the profession best.

Blue Cross is continuing to interest all Minnesota residents in the non-profit plan for hospital care. Blue Cross has found through the use of radio and television that they are effective media for getting the Blue Cross story across to Minnesota people. The majority of persons listen to some type of news program, hence the sponsorship of only this type of program.

Commencing October 5, 1954, the "Bill Ingram News" program can be heard each Tuesday, Wednesday and Thursday from 12:00 to 12:15 p.m. on KSTP-TV, Channel 5.

Charles McCuen will continue to present "News at Noon" each Monday, Tuesday and Thursday on Channel 4, WCCO-TV from 12:00 to 12:15 p.m.

Also heard over WCCO-TV, Channel 4 is the late evening news program, "All the News Tonight" with Charles McCuen, broadcast each Tuesday and Friday evenings from 10:00 to 10:12 p.m.

Holding its own on radio and continuing to bring in a good share of inquiries from Minnesota people is the Saturday night edition of "Night Time News" over WCCO radio. Rolf Hertsgaard, the well-known news broadcaster in the midwest area, is brought to you each Saturday night from 10:00 to 10:15 p.m.

In the Duluth area, which has its own CBS station, Blue Cross is presenting the "Bill Krueger News" on KDAL, Duluth, three times each week at 10:00 to 10:15 p.m. The sponsorship of the "Bill Krueger News" will alternate Monday, Wednesday and Friday one week and Tuesday, Thursday and Saturday the following week.

BOOK REVIEWS

Books listed here become the property of the Ramsey, Hennepin and St. Louis County Medical Libraries when reviewed. Members, however, are urged to write reviews of any or every recent book which may be of interest to physicians.

A TEXTBOOK OF PATHOLOGY, E. T. Bell, M.D., 7th Edition, 1008 pages. Philadelphia: Lea and Febiger, 1952.

The seventh edition of Dr. Bell's book has had adequate changes and additions to keep it well abreast of the recent changes in medicine. The same enjoyable manner of combining pertinent clinical features of a disease with the pathological discussion has been used in this edition.

The section on kidney pathology stands out above the rest of the book because of the author's years of active research in this field.

This book continues to be a "must" in the adequate preparation of each medical student and the first reference for the busy practitioner."

D. W. Koza, M.D.

CARDIOVASCULAR SURGERY. Gerald H. Pratt, M.D., F.A.C.S., Associate Clinical Professor of Surgery, New York University College of Medicine; Attending Surgeon and Chief of the Vascular Clinic Saint Vincent's Hospital, City of New York; Diplomate of the American Board of Surgery; Consultant to the U. S. Naval Hospitals, Meadow Brook Hospital, Long Island; All Souls Hospital, Morristown, New Jersey. 843 pages. Illus. Plates. Price, $15.00. Philadelphia: Lea & Febiger, 1954.

This present volume is an extension and enlargement of an earlier text prepared by Dr. Pratt. To Minnesota readers it may be of interest that Dr. Pratt is a nephew of the late Dr. Robert Emmett Farr of Minneapolis, and in the preface to this volume, Dr. Pratt gives credit to Dr. Farr for guidance in his early years and for outlining his course through medical training. In this same preface, Dr. Pratt states: "This book's purpose is to bring to surgeons, internists and students a summary of accepted or acceptable treatment for cardiovascular lesions."

This text starts with a section on nomenclature, case history and physical examination of the patient. The nomenclature developed by the committee of the American Heart Association is discussed and presented. This is followed by a brief discussion of the essential history required for evaluation of a cardiovascular problem as well as the general medical problem. The essentials of a physical examination and the specific tests required for such an examination are briefly presented.

Under the section of laboratory examinations, included in this chapter, Dr. Pratt has an excellent discussion of the requirements of the internist, the pediatrician and the surgeon who are to deal together with the surgical cardiac patient and points out the necessity of having attached to such a group, someone well trained in cardiopulmonary physiology who has had angiocardiographic and heart catheterization training.

The second section deals with what Dr. Pratt described as ancillary surgical procedures. Anesthesia, both for the patient with cardiovascular disease requiring general surgery and for such patients requiring operations on the heart or cardiovascular system is discussed quite briefly but succinctly. There is also a brief discussion of controlled hypotension anesthesia and of hypothermia.

A short chapter is devoted to "Blood and Fluid Balance," and another short chapter to "Cardiac Arrest and Resuscitation."

Section Three is one hundred pages in length and is devoted to cardiac surgery starting off with a discussion of chest dynamics and cardiac physiology. Dr. Pratt also covers briefly in this section the technical points in a thoracotomy and the postoperative care of a patient who has had a thoracotomy.

The subsequent five chapters of this section are devoted to cardiac surgery and surgery of the pericardium. This is an excellent section. It deals first with penetrating wounds of the heart and then with nonpenetrating wounds of the heart in a very brief but very satisfactory manner. It is most gratifying to see in the opening paragraph of the section on nonpenetrating wounds of the heart that fifteen per cent of all fatal chest injuries are due to crushing trauma of the heart.

The section on "Surgical Treatment of Congenital Heart Lesions" deals very well with these problems starting with the classification of congenital heart disease by Bing. Pulmonary stenosis is presented with the Blalock and Pott's procedures which are well illustrated. There is also an ample section on the Brock procedure of direct valvulotomy for pulmonary stenosis. Patent ductus and coarctation of the aorta are both treated in summary fashion. There are brief descriptions of the various anomalies of the aortic arch, the anomalies of the pulmonary vessels, and intraseptal defects. No effort is made to give any surgical procedures for the correction of these latter defects.

The section on acquired cardiac valvular disease covers mitral stenosis quite nicely and aortic stenosis less completely. The discussion of the surgery in the treatment of coronary occlusion and the cardiac patient as a surgical risk is timely, although Dr. Pratt is unable to find any surgical procedure which he feels is of sufficient value to warrant description in any detail.

Starting on page 159, the book becomes a text covering quite fully diseases of the peripheral vascular system, venous, arterial and lymphatic. It is interesting to page through this text and to read the various sections which Dr. Pratt has included as proper fields of endeavor for a cardiovascular surgeon. Inasmuch as the blood vessels go to every part of the body and inasmuch as their failure to properly carry out their transport role results in disease of the tissue wherever this occurs, logically the cardiovascular surgeon could undertake the enucleation of an eye for thrombosis of the ophthalmic artery as well as the amputation of a toe for ischemic gangrene and the coverage in this text is almost that broad.

In the section on occlusive arterial disease the usual arterial diseases of the extremities are beautifully covered and in addition, consideration is given to the arterial occlusive diseases which occur within the abdomen along with an excellent discussion of arterial disease as it occurs in the diabetic. A chapter is devoted to amputations, the indications, the types and the techniques which are required in the management of arterial disease of the extremities.

In the section on angiospastic diseases and syndromes, many conditions considered by the dermatologist and the internist as their special province such as Raynaud's Disease, scleroderma, cold allergy, spastic anemia and hyperhidrosis are considered. Also with such conditions as Sudeck's atrophy which the orthopedist usually claims is presented. Likewise, in the excellent section on traumatic injury to the arteries the field of the general surgeon is invaded. A discussion of the surgical treatment of hypertension with the operative description of the various sympathectomies that are used today by the neurosurgeons invades the field covered by this latter specialty. Finally, the sections on skin grafting and the section on roentgen ray visualization cover fields normally felt by the plastic surgeon and the radiologist as provinces reserved for their attention.

I do not feel that this broad coverage is a criticism either of Dr. Pratt or of his excellent text. I mention it in this review only to point out that this book has much to offer many different disciplines, as the approach in each instance is presented in this text from the vascular standpoint and oftentimes this approach is stimulating and rewarding. It is also interesting in that it illustrates how, as knowledge develops, certain compartmentalization occurs, which seems reasonable and necessary and is rather jealously maintained by the various devotees. Then a new body of knowledge accumulates and is systematically organized such as has happened in the development in the last fifteen years of cardiovascular surgery and the extension of this body of knowledge into the practical therapeutics of these conditions cuts across many different fields not out of primary intent to invade other areas of specialization but as the logical development of the new specialization. Dr. Pratt's book is much more worthwhile because of its broad coverage and because it ties together as a systematic whole pretty much what is known about the pathology and treatment of diseases of the cardiovascular system particularly from the surgical standpoint.

The last two chapters of this excellent text take a look to the future and a glance at the present. Chapter 44 deals with "Radioactive Isotopes and Atomic Energy in the Diagnosis and Treatment of Cardiovascular Diseases" and Chapter 45 recognizes the daily influence of compensation problems in the practice of medicine and surgery. It is devoted to a discussion of the relationship of trauma and occupational hazards to cardiovascular diseases.

In conclusion, this is an excellent book, beautifully bound, well illustrated, broad in its coverage, brief and concise and to the point all the way through, well written and each chapter beautifully documented with extensive bibliography. Most surgeons should own a copy for their personal library. It will be of value to residents and interns and it should be readily available to all who are engaged in the healing of the sick.

NATHAN KENNETH JENSEN, M.D.

DRUGS IN PEPTIC ULCER

(Continued from Page 788)

of help, they can in no wise substitute for the fundamental effects of rest and of freedom from hurry, worry and emotional tension in the control of peptic ulcer.

References

1. Benjamin, F. B., Rosiere, C. E. and Grossman, M. I.: A comparison of the effectiveness of banthine and atropine in depressing gastric acid secretion in man and the dog. Gastroenterology, 15:727-735 (Aug.) 1950.
2. Gambill, E. E., Morlock, C. G., Butt, H. R. and Wollaeger, E. E.: A study of the effect of an orally administered enterogastrone preparation on the clinical course of patients with duodenal ulcer. Gastroenterology, 14:228-234 (Feb.) 1950.
3. Grimson, Keith: A clinical trial of banthine in cases of peptic ulcer. Gastroenterology, 14:583-586 (Apr.) 1950.
4. Kirsner, J. B. and Palmer, W. L.: Newer gastric antisecretory compounds. J.A.M.A., 151:798-805 (Mar. 7) 1953.
5. Lepore, M. J., Golden, Ross and Flood, C. A.: Oral banthine, an effective depressor of gastrointestinal motility. Gastroenterology, 17:551-559 (Apr.) 1951.
6. McHardy, G. G., Browne, D. C., Marek, F. H., McHardy, Robert and Ward, Swan: Clinical evaluation of methantheline (banthine) bromide in gastroenterology. J.A.M.A., 147:1620-1626 (Dec. 22) 1951.
7. Plummer, Kemp, Burke, J. O. and Bradford, Sarah C.: The effect of methantheline bromide (banthine) on the insulin induced secretion of gastric acid, mucoproteose and mucoprotein: physiologic implications. Gastroenterology, 18:218-223 (June) 1951.
8. Ruffin, J. M., Texter, E. C., Jr., Carter, D. D. and Baylin, G. J.: Role of anticholinergic drugs in treatment of peptic ulcer. J.A.M.A., 153:1159-1161 (Nov. 28) 1953.
9. Smith, C. A., Woodward, E. R., Janes, C. W. and Dragstedt, L. R.: The effect of banthine on gastric secretion in man and experimental animals. Gastroenterology, 15:718-726 (Aug.) 1950.
10. Texter, E. C., Jr., Baylin, G. J., Legerton, C. W., Jr. and Ruffin, J. M.: Effects of a new cholinergic blocking agent (SKF-1637) on gastric motor and secretory activity. Am. J. M. Sc., 224:612-618 (Dec.) 1952.
11. Texter, E. C., Jr., Legerton, C. W., Ruffin, J. M., Atwater, J. S., Cayer, David, Cheney, F. D., Jackson, R. A., Oren, B. G. and Rumball, J. M.: Prolonged drug therapy in peptic ulcer. I. An evaluation of banthine as an adjunct to conventional ulcer therapy. South. M. J., 46:1062-1068 (Nov.) 1953.

MANAGEMENT OF MASSIVE HEMATEMESIS OF UNDETERMINED CAUSE

CHARLES E. REA, M.D.
Saint Paul, Minnesota

THE PURPOSE of this paper is to review the treatment of our cases of massive hematemesis in which the source of bleeding was not known preoperatively. Also, an evaluation of blind gastrectomy in such cases will be made.

Given a patient with massive hematemesis, the cause of the bleeding is usually due to either a duodenal ulcer, gastric ulcer, carcinoma of the stomach, gastric erosions, or esophageal varices. When any one of these lesions is found, the surgeon can institute definitive treatment. However, most surgeons have had the experience of operating upon patients in whom hematemesis could not be controlled by conservative means, and in whom no obvious cause for the bleeding was found. This occurs in as high as one-quarter or one-third of the patients operated upon for hematemesis so the problem is of more than academic interest.

When a surgeon operates upon a patient who has had a massive hematemesis, and the source of bleeding is not known preoperatively, he usually opens the stomach and duodenum to inspect the interior for the bleeding source. Palpation of the stomach and duodenum without opening these organs is a rather unsatisfactory procedure. If a duodenal ulcer is found, but is not actively bleeding, a search should be made of the stomach to be sure there are no bleeding lesions there. We routinely open the stomach and duodenum in all such cases because, on two occasions while a chronic duodenal ulcer was found, the bleeding was from an unsuspected gastric ulcer.

If, on inspecting the stomach and the first portion of the duodenum, the source of bleeding is still not found, a careful search of the upper end of the stomach, especially around the esophageal opening, and also the entire duodenum should be made. Gilchrist and Chun report a series[2] of eleven cases. In three instances fatal bleeding occurred from lesions 3.5 to 7 centimeters from the esophagus. In our experience, we have had a hemangioma in the second portion of the duodenum and also a leomyosarcoma near the ligament of Treitz. Neither of these lesions was discovered until autopsy but the futility of doing the conventional gastric resection with removal of 75 per cent of the stomach in such cases is apparent.

If, after careful examination of the entire stomach and duodenum no lesion is found, many surgeons perform a gastric resection, removing about 75 per cent of the stomach. This was popularized by Wangensteen[3] in 1945. More recently Cooper and Ferguson[1] have reported their experiences with this procedure. In their eighteen patients who received operations as an emergency measure for the control of upper gastro-intestinal hemorrhage, six showed no evidence of the source of bleeding even after thorough exploration. In each instance a resectable mucosal lesion or acute ulceration was established as the source of bleeding when the stomach was studied by the pathologist. In five of the patients, a subtotal gastric resection was done. In the one patient in whom a resection was not done the patient subsequently bled to death from a resectable superficial gastric erosion high on the lesser curvature of the stomach.

If one suspects gastric erosions, one should

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probably do an infradiaphragmatic vagotomy at the same time as the resection. It is interesting that of three patients with demonstrable bleeding gastric erosions that have come to our attention, one occurred after intensive hormone therapy for the menopause, the other after cortisone therapy for arthritis and the third in a woman five months pregnant. The hormonal factor may be of etiological significance.

TABLE I.

No. of Cases	Lesion	Gastric Resection	Benefit	Comment
1	Hemangioma—2nd portion duodeum	+	0	Lesion found at autopsy
1	Leiomyosarcoma—3rd portion duodeum near ligament of Treitz	+	0	Lesion found at autopsy
2	Gastric erosions	+	+	Not recognized at operation
3	Duodenal ulcers	+	+	
2	Gastric ulcers	+	+	Associated with inactive chronic duodenal ulcer
1	Bleeding esophageal varices	+	+	Esophagogastrectomy performed
2	No definite lesion found	+	+	
12				

In our series, there were two patients in whom multiple gastric erosions were found by the pathologist in the resected stomach. In both of these cases a three-quarter gastric resection had been done but the lesions were not diagnosed at the time of exploration.

If no lesion is found on examining the open stomach and duodenum to account for the massive hematemesis, why not simply close the incision without a resection? It appears that a gastric resection is justified in these instances from a review of our cases (Table I). Although in two cases gastric resection was of no value, in eight cases it was of definite value as the bleeding source was removed. In two cases it was of questionable value as no definite lesion was found, although the bleeding was controlled. If, after a blind gastric resection, the patient still bleeds, what should be done? Most surgeons would try conservative measures at first, but if these are of no avail, further resection of the stomach is usually resorted to. It should be emphasized that a careful search of the remaining stomach and the duodenum to the ligament of Treitz should be made because an obscure lesion, previously undiagnosed, may be found in these regions.

Summary

In most cases of massive hematemesis in which the source of bleeding is not known preoperatively, the cause will be definitely found at the time of operation. This occurred in eight of our twelve cases. However, in one-third of the cases a definite lesion was not found. In two of these four cases a gastric resection was performed and the cause of the bleeding was not found in the resected stomach. In the two other instances, lesions in the duodenum were found which were not amenable to gastric resection and in which a gastric resection would have been useless under any circumstance.

While our series is small, the following conclusions seem valid. A gastric resection is of value if the bleeding lesion is found in the stomach or duodenum (eight out of twelve of our cases). If the lesion is not found and a blind resection done, the surgeon may experience further difficulty in about half of the cases (two out of four of our cases).

References

1. Cooper, D. R., and Ferguson, L. K.: Gastric resection for upper gastro-intestinal hemorrhage of undetermined cause. J.A.M.A., 151:879 (March 14) 1953.
2. Gilchrist, R. K., and Chun, N.: Severe hemorrhage in presumed peptic ulcer. Arch. Surg., 69:366, 1954.
3. Wangensteen, O. H.: The ulcer problem. Canad. M.A.J., 53:309, 1945.

RECURRENT PEPTIC ULCER

GEORGE A. HALLENBECK, M.D.

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ALTHOUGH surgical treatment is ordinarily quite effective in eliminating the pathologic process and controlling the symptoms in properly chosen cases of gastric and duodenal ulcer, failures occur in some instances. One complication of such operations which usually requires further surgical treatment is recurrence of peptic ulceration. The present discussion is concerned with this problem.

Brief Résumé of the Pathologic Physiology of Peptic Ulceration

Peptic ulceration may develop in any part of man's gastrointestinal tract which can be bathed with acid peptic gastric juice: the stomach, the duodenum, the small intestine near a gastroenteric anastomosis, the ileum in the presence of a Meckel diverticulum containing ectopic gastric mucosa and occasionally near sites of ectopic gastric mucosa elsewhere. Continuous hypersecretion of highly acid gastric juice has been observed in patients with duodenal ulcer both during exacerbations of the disease and during its quiescent phase.[15] The duodenal content of patients with duodenal ulcer, both during fasting and after eating, tends to be more acid than does that of normal people.[3] However numerous other contributing factors may be, the facts remain that the presence of acid gastric juice is almost always necessary for the continued existence of benign peptic ulcers and that such ulcers almost invariably heal if the ulcerated area can be protected from the action of gastric juice by nonsurgical or surgical means.

Understanding of the problem has been greatly aided by experiments upon animals, too numerous to be presented in detail here, but which constitute a rewarding chapter in the field of experimental surgery. Peptic ulceration can be produced in dogs by surgical procedures, such as the Mann-Williamson operation,[16] which deprive the small intestine near a gastroenteric anastomosis of the neutralizing effect of bile and pancreatic juice or by methods, such as injection of histamine in beeswax,[11] which cause continuous hypersecretion of acid gastric juice. Results of many studies along these lines consistently support the hypothesis that peptic ulceration will occur in experimental animals when there is inadequate neutralization or excessive production of gastric juice. It is logical to apply these premises to man; indeed, they constitute the physiologic basis for present-day medical and surgical treatment of peptic ulcer. Recurrent ulceration, which follows surgical treatment for duodenal ulcer much more often than that for gastric ulcer, usually occurs in the jejunum adjacent to the site of gastrojejunostomy and can be looked upon as the result of failure of the original operation to reduce the rate of secretion of gastric juice below the level which can produce ulceration in the jejunum of a particular patient.

Prevention of Recurrence of Peptic Ulceration With Reference to Various Standard Operative Procedures

Gastroenterostomy.—The surgical procedure, gastroenterostomy, presents an entirely logical mechanical solution to the problem of pyloric obstruction and the operation has produced many brilliant results in this situation. During the first 3 decades of this century, it came to be used frequently for duodenal ulcer, with or without symptoms of obstruction, and was commonly followed by at least temporary healing of the ulcer and alleviation of symptoms. The physiologic basis for the improvement so often noted is not entirely clear. There is no reason to believe that gastroenterostomy reduces gastric secretion; in fact, experiments indicate that in the dog the operation can result in an increased secretion of gastric juice.[12,27]

Read at the annual meeting of the Northern Minnesota Medical Association, Willmar, Minnesota, September 10, 1954.

From the Section of Surgery, Mayo Clinic and Mayo Foundation.

The Mayo Foundation is a part of the Graduate School of the University of Minnesota.

Despite the many good results which followed gastroenterostomy when the operation was applied as a routine for patients with duodenal ulcers, the procedure fell into disrepute because of the distressingly high incidence of subsequent gastrojejunal ulceration. Data from a series of papers reviewed by Portis[20] indicate a reported incidence of gastrojejunal ulceration following gastroenterostomy varying between 1.4 per cent and 34 per cent. The first figure is far too low and a commonly quoted estimate of the incidence is 16 per cent. The interval between gastroenterostomy and a secondary surgical procedure for gastrojejunal ulcer may be long. In a study of 186 cases reported by Walters, Chance and Berkson,[24] this interval was 11.2 years. Thus the shorter the follow-up, the lower will the observed incidence probably be. Likewise, a lower incidence for recurrence can be expected if the operation is not used for younger people, especially men, with very pronounced ulcer diatheses, and a higher recurrence rate will follow inclusion of such patients in a series.

Both clinical experience and laboratory data provide information to help minimize occurrence of gastrojejunal ulceration after gastroenterostomy. The first warns the surgeon to avoid use of the operation in young and middle-aged people with very active ulcer symptoms and to consider its usefulness peculiar to a few older people whose symptoms are primarily due to cicatricial pyloric obstruction and whose physical status is so poor that more extensive operations would entail too great a risk. The second, based on the observation that, in dogs, establishment of a gastrojejunal stoma high in the stomach is followed by increased secretion from a Heidenhain pouch whereas establishment of a stoma in the antrum is not,[27] suggests that the stoma should be made low in the stomach. Ample laboratory data also indicate that anastomosis between proximal and distal loops of jejunum is to be avoided because it deviates neutralizing secretions from the stoma and promotes formation of gastrojejunal ulcers.[7]

Gastric Resection.—Use of gastric resection as treatment for duodenal ulcer grew from dissatisfaction with the high incidence of recurrence of ulcers following gastroenterostomy in most patients who require surgical treatment for the disease. Furthermore, the current practice of limiting recourse to surgery for patients with definite complications of duodenal ulcer has decreased the number of surgically treated patients with disease mild enough to permit expectation of good results with gastroenterostomy. Improvements in preoperative and postoperative care together with standardization of operative techniques, has lowered the risk of gastric resection to acceptable levels. The purpose of gastric resection is to reduce the total quantity of gastric juice secreted daily to levels which render subsequent ulceration at the gastroenteric stoma unlikely while still leaving enough stomach to permit eating with comfort and to allow maintenance of body weight.

Several techniques have been developed for reestablishment of gastrointestinal continuity after gastric resection. One would think that some type of gastroduodenostomy would most nearly restore normal anatomic relationships, render subsequent ulceration least likely and probably favor best digestion of food; this procedure has many partisans at present. Actually, the deformity of the duodenum often precludes satisfactory end-to-end gastroduodenostomy and the fixation of the duodenum commonly seen in patients with severe duodenal ulcer would often limit the extent of resection to unsafe levels if the operation were forced. Furthermore, the incidence of recurrent ulceration after the Billroth I procedure for duodenal ulcer has often been reported higher than that for resection with gastrojejunostomy. For these reasons, one of the variants of the Billroth II procedure in which the remaining stomach is joined to the jejunum is commonly used. Judging from the variations in preference shown by individual surgeons for postcolic or antecolic anastomoses, for the Hofmeister or "full" Polya operation and so forth, it seems fair to state that several procedures can give satisfactory results. There are, however, certain principles which must be followed if recurrent ulceration is to be kept at a minimum. These principles are:

1. *Removal of an adequate amount of stomach.* In the dog, 75 per cent of the stomach must be removed to prevent formation of ulcer in response to injections of histamine in beeswax, as Lannin,[14] in Wangensteen's laboratory, has

shown. In man, lower resections are followed by a higher incidence of recurrent ulceration than are higher resections. Figures of more than 9 per cent are common in series of patients from whom half or less of the stomach is removed,[22] values of around 4 to 5 per cent often accompany descriptions of removing two-thirds of the stomach[1] and recurrence rates of less than 1 per cent have been reported by surgeons who describe removing three-fourths of the stomach.[9,10] Because it has not seemed to me that increasing the extent of resection from two-thirds to three-fourths significantly increases the severity or incidence of postgastrectomy symptoms, I favor higher resections and try to remove three-fourths of the stomach.

2. *Complete removal of the pyloric (antral) mucosa.*—Experiments on dogs, particularly those of Dragstedt and his colleagues,[5,19] conclusively show that, under certain circumstances, the antrum may liberate a hormone which stimulates any remaining acid-secreting gastric mucosa to secrete large quantities of juice. Clinical experience amply proves that leaving some or all of the antrum in place is followed by an increased incidence of peptic ulceration.[13,17] One must conclude that though the duodenal ulcer itself can be safely left in place if this is technically advantageous, the antral mucosa must be totally removed if recurrence of ulcer is to be avoided.

3. *Use of as short a proximal loop of jejunum as is practicable.* Merendino and his associates,[18] in Wangensteen's laboratory, found that with resection of 75 per cent of the stomach of dogs, stomal ulcers developed in response to injections of histamine and beeswax if the proximal loop of jejunum was long but not if this portion was made short. The ability of jejunal contents distant from the duodenum to neutralize acid is less than that of contents obtained nearer the duodenum.[4] The corollary of this observation is that the afferent loop of jejunum should be made as short as practicable.

4. *Avoidance of any procedure such as enteroenterostomy which diverts duodenal contents away from the anastomosis.* This concept has been presented above with reference to gastroenterostomy.

If what has been recommended is followed carefully, it should be possible to keep the incidence of recurrent ulceration to no more than 3 per cent, and even lower figures have been reported.

Gastroenterostomy With Vagotomy.—At present, gastroenterostomy with vagotomy is enjoying extensive clinical trial. The purpose of adding vagotomy, of course, is to eliminate the cephalic phase of gastric secretion, stimulation of which is mediated by the vagus nerves. Though longer periods of observation will be needed to establish the incidence of recurrence of ulceration after this combined operation, present indications are that it lies between 5 and 8 per cent.[23] It is impossible to be certain how many of these recurrences are associated with incomplete vagotomy, but not all are.[2] If the pattern of recurrence of ulceration after the combined operation were to be similar to that after gastroenterostomy alone, the incidence of gastrojejunal ulcer would rise as the years pass. Vagotomy alone, as a primary procedure for duodenal ulcer, has been abandoned because of the high incidence of recurrences, failures of the stomach to empty properly, and diarrhea.

Other Procedures Currently Being Evaluated.—Though experience with certain other procedures is still insufficient to permit more than tentative conclusions, they deserve mention.

Hemigastrectomy and vagotomy are appealing because the procedure eliminates the cephalic and antral mechanisms for stimulating gastric secretion while leaving the reservoir function of the stomach less impaired than is the case with subtotal gastric resection. In a preliminary report, Farmer and Smithwick[8] noted that achlorhydria followed this procedure more regularly than was the case with either gastroenterostomy and vagotomy or with subtotal gastric resection. It is too early to determine the incidence of recurrence which will follow this operation. The procedure is based upon sound physiologic principles and deserves careful trial.

Subtotal gastrectomy with vagotomy has been appraised by Druckerman and his associates,[6] who reported no recurrent ulcers in a group of 165 patients who had undergone this operation, as compared with twelve recurrent ulcers (5.5 per cent) in a group of 220 patients who had undergone partial gastrectomy alone. Morbidity, largely postoperative diarrhea, was greater in the group which had undergone gastric resection with vagotomy.

Wangensteen[25,26] has recently been trying two operations which have as their principle the removal of sufficient acid-secreting gastric mucosa that even though the antrum, with its potentiality of stimulating secretion of acid gastric juice, is preserved, so little acid juice can be secreted that ulcerogenesis is prevented. The first of these is segmental gastric resection, which consists of sleeve resection of the body of the stomach with preservation of the pyloric antrum and of a rim of stomach near the esophagus constituting only 5 to 10 per cent of the organ. The remaining upper remnant of stomach and the antrum are anastomosed and pyloroplasty is carried out to insure satisfactory gastric emptying. The procedure protects dogs against histamine-induced ulcers and, in 1953, Wangensteen had noted no recurrent ulcers in a group of ninety patients, some of whom had undergone the operation as early as 1949. The second of the procedures which Wangensteen[26] has been trying is termed "tubular gastric resection with transverse gastroplasty." In this operation a large portion of the acid-secreting gastric mucosa is removed in one piece, consisting of the anterior and posterior gastric walls and the intervening greater curvature of the stomach. The lesser curvature is left intact and vagus nerve fibers passing down it are not cut. Transverse closure of the resulting defect, supplemented by pyloroplasty if obstruction is present, completes the procedure. Tubular resection protects dogs against formation of ulcers when histamine in beeswax is given. It is too early to know what the incidence of recurrent ulcer may be after this procedure but Wangensteen[25] has reported none in a group of approximately ninety patients so treated over a period of a year and a half.

Diagnosis of Gastrojejunal Ulceration

Recurrent ulceration is less common after gastric resection for gastric ulcer than after the same operation done for duodenal ulcer. For example, Priestley and Gibson[21] reported the antecedent lesion to be gastric ulcer in only 1.4 per cent of 283 cases of gastrojejunal ulcer studied. The disease occurs predominantly in men. In a series of 301 patients with gastrojejunal ulcer studied by Walters, Chance and Berkson,[24] 91 per cent were men and 9 per cent were women.

In general, whenever gastroenterostomy or gastric resection has been performed for peptic ulcer and symptoms of ulcer again develop or hemorrhage from the stomach takes place, gastrojejunitis or gastrojejunal ulcer should be suspected. Roentgenologic demonstration of these lesions tends to be more difficult than is the case with primary gastric or duodenal ulcers. Thus, in one series of cases,[24] a positive roentgenologic diagnosis of gastrojejunal ulcer was made in only 50 per cent of cases of ulcer occurring after gastric resection and 37 per cent of those following gastroenterostomy. In some instances, gastroscopic examination may provide a positive diagnosis but a negative gastroscopic examination is not of significance because the ulcer may be hidden from view in the jejunum.

Gastrojejunocolic fistula, variously reported to occur in from about 6 to 15 per cent of patients with gastrojejunal ulcer, is demonstrated by roentgenologic examination of the colon with the aid of barium much more regularly than by similar examination of the stomach.

Hemorrhage occurs in approximately 50 per cent of cases and is not usually fatal.

In many instances, the diagnosis of gastrojejunal ulcer will be made only at the operating table and the patient's signs and symptoms of recurrence must often provide the indications for surgical operation in the absence of objective preoperative evidence for ulcer.

Treatment of Gastrojejunal Ulcer

Although trial of nonsurgical treatment for ulcer may be justified in some cases in which recurrent symptoms develop after operation for peptic ulcer, and continued nonsurgical treatment may be advisable in a few cases in which the patient's general condition precludes surgical operation at any but a prohibitive risk, the majority of patients are best treated surgically.

When gastrojejunal ulcer follows gastroenterostomy, the choice will lie between vagotomy alone, provided there is no evidence for obstruction, and gastric resection with or without vagotomy. Walters, Chance and Berkson[24] have compiled the most recent data from the Mayo Clinic in this regard and have reported that 78 per cent of twenty-seven patients obtained excellent results after vagotomy alone (no mortality) whereas 86.5 per cent of 111 patients obtained excellent results after the anastomosis made at gastroenterostomy had been disconnected and gastric resection performed (mortality rate 0.7 per cent). These data give gastric resection a slight edge over vagotomy for gastrojejunal ulcer following gastroenterostomy.

There are undoubtedly patients in whom the added security of vagotomy, performed in addition to gastric resection, compensates for the increased morbidity which the procedure is reported to impose and there remains room for the surgeon, giving consideration to the severity of the ulcer diathesis, to exercise his judgment in this regard. Simple disconnection of the gastroenteric anastomosis is mentioned only to condemn it, for results are usually poor.

In the presence of gastrojejunal ulcer following gastric resection, re-resection of the stomach is accompanied by a much higher operative mortality rate and gives poorer results than vagotomy alone. In the series of Walters, Chance and Berkson,[24] 70.5 per cent of seventy-eight patients who had gastrojejunal ulcers after gastric resection reported excellent results after vagotomy. The operative mortality rate was only 1.1 per cent. There was no difference in end results whether vagotomy was performed transthoracically or transabdominally.

Thus, data from the Mayo Clinic led to the conclusion that disconnection of the gastroenteric anastomosis, together with gastric resection, sometimes accompanied by vagotomy, is the procedure of choice for gastrojejunal ulcers following gastroenterostomy and that vagotomy alone is best for such ulcers following all but minimal gastric resections.

Patients with gastrocolic fistulas may be seen when in a state of good nutrition or may be encountered with moderate or severe degrees of malnutrition. Patients in poor condition become so not so much because of loss of foodstuffs into the colon as because of passage of feces into the stomach, with consequent gastroenteritis and anorexia. Because of this, establishment of a temporary colonic stoma proximal to the gastrocolic fistula has been proposed as a preliminary operation before definitive surgical treatment of the fistula and has been found effective in relieving the anorexia and improving nutrition. Though I would not hesitate to use this procedure if the general condition of a patient was poor enough that closure of the fistula and an appropriate operation for the ulcer diathesis would entail too much risk, experience at the clinic has indicated that most of the patients seen recently can be treated satisfactorily by a single operation. Thus, Walters, Chance and Berkson[24] have described twenty patients with gastrocolic fistulas treated between 1945 and 1952 with procedures of one stage. In sixteen of these cases the antecedent operation had been gastroenterostomy. In twelve of these, excision of the fistula was followed by gastric resection; in four by vagotomy. In four patients, the gastrocolic fistula followed gastric resection. Three of these were treated by gastric re-resection and vagotomy, one with excision of the fistula and vagotomy. There were no operative deaths. Of fifteen patients who could be followed, thirteen had excellent results.

Perspective on the Problem

This discussion, concerned as it is with the problem of recurrent ulceration after surgical treatment for peptic ulcer, should not lead to the erroneous conclusion that this combination is the only problem to be considered. The sacrifice of large amounts of stomach is followed by crippling postgastrectomy symptoms in a few patients and by less serious but very annoying symptoms, such as inability to eat more than small amounts at one time and inability to maintain acceptable body weight, in some others. Vagotomy is sometimes followed by delayed emptying of the stomach, epigastric bloating discomfort, vomiting and diarrhea; these may be only mildly annoying or in a few instances may be described as worse than the original disease. A low incidence of recurrent ulceration is only one, though a very important, criterion of a successful operation for peptic ulcer. It is fair to state that present-day surgical treatment for peptic ulcer is superior to that of even fifteen years ago with regard to mortality rate and incidence of undesirable by-products of operations, and that results compare favorably with those obtained from treatment of a host of other surgically treated lesions. Yet the fact that some patients, perhaps 10 or 15 per cent of those operated upon, obtain results that fall short of reasonable satisfaction, properly prompts surgeons constantly to re-evaluate the procedures and to seek better means of treating this very common disease.

References

1. Allen, A. W. and Welch, C. E.: Subtotal gastrectomy for duodenal ulcer. Ann. Surg., 124:688-703 (Oct.) 1946.
2. Brooks, J. R. and Moore, F. D.: Vagotomy for duodenal ulcer: a final survey after 10 years. New England J. Med., 249:1089-1097 (Dec. 31) 1953.
3. Comfort, M. W.: Acidity of the duodenal contents in health and disease: a review of clinical investigations. Gastroenterology, 4:135-146 (Feb.) 1945.
4. Cross, F. S.: The loss of buffering capacity of alkaline solutions after passage through long and short duodeno-jejunal loops. In: Proceedings of the Forum Sessions, Thirty-seventh Clinical Congress of the American College of Surgeons (November) 1951. Philadelphia: W. B. Saunders Company, 89-93, 1952.
5. Dragstedt, L. R., Woodward, E. R., Oberhelman, H. A., Jr., Storer, E. H. and Smith, C. A.: Effect of transplantation of antrum of stomach on gastric secretion in experimental animals. Am. J. Physiol., 165:386-398 (May) 1951.
6. Druckerman, L. J., Weinstein, V. A., Klingenstein, Percy and Colp, Ralph: Duodenal ulcer treated by subtotal gastrectomy with and without vagotomy: six year comparative study. J.A.M.A., 151:1266-1269 (Apr. 11) 1953.
7. Exalto, J.: Quoted by Lannin, B. G., 14.
8. Farmer, D. A. and Smithwick, R. H.: Hemigastrectomy combined with resection of the vagus nerves. New England J. Med., 247:1017-1022 (Dec. 25) 1952.
9. Gaviser, David: Clinical investigation and evaluation of four hundred sixteen cases consecutively operated upon for peptic ulcer. Surgery, 24:873-916 (Nov.) 1948.
10. Graham, R. R.: Technical surgical procedures for gastric and duodenal ulcer. Surg., Gynec. & Obst., 66:269-287 (Feb. 15) 1938.
11. Hay, L. J., Varco, R. L., Code, C. F. and Wangensteen, O. H.: Experimental production of gastric and duodenal ulcers in laboratory animals by intramuscular injection of histamine in beeswax. Surg., Gynec. & Obst., 75:170-182 (Aug.) 1942.
12. Kanar, E. A., Schmitz, E. J., Sauvage, L. R., Storer, E. H. and Harkins, H. N.: The secretory response of the stomach to gastroenterostomy, as measured by a Heidenhain pouch. In: Proceedings of the Forum Sessions, Thirty-eighth Clinical Congress of the American College of Surgeons (September) 1952. Philadelphia: W. B. Saunders Company, 12-17, 1953.
13. Kiefer, E. D.: Jejunal ulcers and recurrent hemorrhages: after partial and subtotal gastrectomy for peptic ulcer. J.A.M.A., 120:819-824 (Nov. 14) 1942.
14. Lannin, B. G.: Experimental evaluation of a satisfactory operation for ulcer. Surgery, 17:712-741 (May) 1945.
15. Levin, Erwin, Kirsner, J. B., Palmer, W. L. and Butler, Carrie: A comparison of the nocturnal gastric secretion in patients with duodenal ulcer and in normal individuals. Gastroenterology, 10:952-964 (June) 1948.
16. Mann, F. C. and Williamson, C. S.: The experimental production of peptic ulcer. Ann. Surg., 77:409-422 (Apr.) 1923.
17. McKittrick, L. S., Moore, F. D. and Warren, Richard: Complications and mortality in subtotal gastrectomy for duodenal ulcer: report on a two-stage procedure. Ann. Surg., 120:531-554 (Oct.) 1944.
18. Merendino, K. A., Lannin, B. G., Kolouch, Fred, Jr., Baronofsky, Ivan, Litow, S. S. and Wangensteen, O. H.: Length of afferent duodenojejunal loop in gastric resection; a factor in stomal ulcer. Proc. Soc. Exper. Biol. & Med., 58:226-230 (Mar.) 1945.
19. Oberhelman, H. A., Jr., Woodward, E. R., Zubiran, J. M. and Dragstedt, L. R.: Physiology of the gastric antrum. Am. J. Physiol., 169:738-748 (June) 1952.
20. Portis, Bernard: Indications for surgical treatment of peptic ulcer. In: Portis, S. A.: Diseases of the Digestive System. Philadelphia: Lea & Febiger, 599-608, 1941.
21. Priestley, J. T. and Gibson, R. H.: Gastrojejunal ulcer: clinical features and late results. Arch. Surg., 56:625-637 (May) 1948.
22. Rienhoff, W. F., Jr.: An analysis of the results of the surgical treatment of 260 consecutive cases of chronic peptic ulcer of the duodenum. Ann. Surg., 121:583-599 (May) 1945.
23. Walters, Waltman and Chance, D. P.: Vagotomy as a prophylactic and curative procedure in peptic ulcer. J.A.M.A., 153:993-997 (Nov. 14) 1953.
24. Walters, Waltman, Chance, D. P. and Berkson, Joseph: A comparison of vagotomy and gastric resection for gastrojejunal ulceration: a follow-up study of 301 cases. Surg., Gynec. & Obst. (In press.)
25. Wangensteen, O. H.: Evolution and evaluation of an acceptable operation for peptic ulcer: including description of the technic of tubular gastric resection with transverse gastroplasty, and extrapleural sternotomy for operations in the attic of the abdomen. Rev. Gastroenterol., 20:611-626 (Sept.) 1953.
26. Wangensteen, O. H.: Segmental gastric resection for peptic ulcer. J.A.M.A., 149:18-23 (May 3) 1952.
27. Zubiran, J. M., Kark, A. E., Montalbetti, A. J., Morel, C. J. L. and Dragstedt, L. R.: Quantitative studies in the effect of gastrojejunostomy on gastric secretion. In: Proceedings of the Forum Sessions, Thirty-eighth Clinical Congress of the American College of Surgeons (September) 1952. Philadelphia: W. B. Saunders Company, 5-11, 1953.

STAPHYLOCOCCAL SEPSIS: CONTROL WITH ANTIBIOTICS

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THE problems encountered in the management of staphylococcal infections are increasing in their relative importance when compared to the problems in the control of other infections, particularly those caused by other Gram-positive bacteria. The treatment of severe staphylococcal infections, such as staphylococcal septicemia and acute endocarditis, is one of the most difficult problems in the management of infectious diseases.

The magnitude of the problem is better understood by considering that over 1000 strains of coagulase-positive staphylococci were isolated from 700 patients at the University of Minnesota Hospitals in a recent period of ten months. Most of these cultures were from localized infections. In the last two and one-half years we have studied approximately fifty patients with staphylococcal septicemia having a fatality rate of approximately 57 per cent. This high fatality rate is discouraging, since it has occurred under circumstances where many different antibiotics were available.

Staphylococci manifest considerable resistance to antibacterial agents which remain highly effective against other bacterial species. We have not encountered highly resistant strains of group A hemolytic streptococci, pneumococci, gonococci or the spirochete of syphilis. Strains of staphylococci which are now causing infections in hospitalized patients are so frequently resistant to penicillin, streptomycin and the tetracycline compounds (chlortetracycline, oxytetracycline and tetracycline), that a choice of the proper antibiotic for the treatment of severe staphylococcal sepsis becomes extremely difficult.

In order to control these highly fatal infections, certain helpful concepts may be formulated by considering the following pertinent points:

1. *Ubiquity of Staphylococci.*—The wide distribution of staphylococci and their presence on the skin results in frequent contamination of specimens which are submitted to the bacteriological laboratory. The presence of staphylococci in a blood culture must be evaluated as to whether the organisms represent contamination or the cause of an infection. This can be a very difficult problem. Helpful information is gained by the coagulase test, because if the culture coagulates plasma, it is probably a pathogenic strain. The coagulase test does not resolve the situation entirely, for coagulase-positive strains obtained from the skin of patients may present false information. This dilemma does not occur with other pathogens to the same degree that it does with staphylococci. Extreme precautions must be used in collecting and handling specimens. When obtaining blood for culture, the skin should be thoroughly cleaned with soap and water and dried with alcohol. Then it should be painted with tincture of iodine. The specimen is collected and the iodine can be removed with Richardson's solution. Attention to these precautions will contribute much to the proper evaluation of the patients' disease.

2. *Enzymatic and Toxic Properties of the Organism and Its Metabolic Products.*—The properties of the staphylococci are related to the manifestations of disease. During multiplication these micro-organisms produce potent toxins and enzymes which play major roles in the pathogenesis of the disease. Toxins arise which allow the bacteria to penetrate the tissue, cause necrosis, destroy leukocytes and fibrin, and hemolyze red blood cells. There is evidence that the pathogenic staphylococci are able to survive and multiply in the phagocytes. There is also a coagulating mechanism which probably is important in localizing the infections and causing formation of the abscesses. Some strains produce an enzyme, peni-

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TABLE I.
STAPHYLOCOCCUS AUREUS ISOLATED FROM INFECTIONS AT MINNEAPOLIS GENERAL HOSPITAL

	Resistant to >50 Units or Micrograms per ml.		
	1951	1952	1953
Penicillin	62.5%	57.2%	67.7%
Streptomycin	48.0	48.8	65.3
Oxytetracycline	38.0	47.5	62.7
Chlortetracycline	23.0	33.2	62.7
Chloramphenicol	25.0	2.5	0.65

TABLE II.
STAPHYLOCOCCUS AUREUS ISOLATED FROM INFECTIONS AT MINNEAPOLIS GENERAL HOSPITAL

	Resistant to >3.1 Units or Micrograms per ml.		
	1951	1952	1953
Bacitracin	10%	11.5%	7.8%
Neomycin	0	25.0	4.5
Erythromycin	—	0	21.0
Carbomycin	—	0	20.9

cillinase, which can quickly and completely inactivate penicillin.

3. *Tissue Reactions of the Invaded Host.*—The outstanding feature of staphylococcal infections is the formation of abscesses. Metastatic abscesses occur frequently and appear in the kidneys, liver, spleen, myocardium, lungs and brain with resultant complications of severe degree.[16] In the formation of an abscess, large numbers of polymorphonuclear leukocytes migrate to the site of bacterial localization, resulting in an inflammatory reaction with walling off of the infection. In the center there is a relatively avascular area with exudate, necrotic tissue, and large numbers of bacteria surrounded by the inflammatory wall. This is significant from the viewpoint of therapy. The amount of antibiotics which can diffuse into the center of the abscess is not known, yet this is the site where inhibition and destruction of the bacteria must be attained. Frequently a patient improves following therapy, treatment is discontinued, and relapse occurs. Awareness of the protection afforded the staphylococci in the abscess will be helpful in the management of this infection.

4. *Selection and Propagation of Strains Highly Resistant to Antibiotics.*—As mentioned, the problem of resistance to antibiotics is a challenging question. All strains have a measurable degree of resistance to antibiotics, but for practical application in therapy, arbitrary criteria for the delineation of degrees of resistance have been generally accepted. For this purpose strains of staphylococci are classified as follows: (a) sensitive, inhibited by 1.0 unit or microgram per ml. or less; (b) moderately resistant, inhibited by 1.0 to 10.0 units or micrograms per ml., and (c) resistant, inhibition by more than 10 units or micrograms of antibiotics per ml.

The incidence of penicillin-resistant strains in infections acquired outside of hospitals is less than in patients who acquire infections in hospitals. Approximately 25 per cent of staphylococcal infections isolated from out-patients show penicillin-resistant strains, and this figure has remained approximately the same for the last six years.[3,4,7,9,15,18] The incidence of penicillin-resistant strains causing infections in hospitalized patients has increased. At the time of introduction of penicillin in the treatment of infections, strains of resistant staphylococci were rare.[12,17] At the present time approximately 75 per cent of the staphylococci isolated from infections in hospitalized patients are resistant to the antibiotic.

A summary of the percentages of strains of staphylococci which were found to be resistant *in vitro* to over 50 units or micrograms of antibiotic per ml. for the years 1951 through 1953 is found in Table I. These strains were isolated from patients with staphylococcal infections at the Minneapolis General Hospital. Sixty-two and five-tenths per cent in 1951, 57.2 per cent in 1952 and 67.7 per cent in 1953 were not inhibited by 50 units of penicillin per ml.*

Results with streptomycin have been similar. A survey in 1949 revealed no strains of staphylococci to be resistant to this antibiotic. At Minneapolis General Hospital the percentage of strains which were resistant to 50 mcgm. per ml. increased from 48 per cent in 1951 to 65.3 per cent in 1953.

There has been increasing percentage of strains which are resistant to the tetracycline group of antibiotics. With oxytetracycline (terramycin), there was an increase from 38 per cent in 1951 to 47.5 per cent in 1952 to 62.7 per cent in 1953 of strains resistant to this high concentration. Results for chlortetracycline (aureomycin) are similar.

The data in regard to chloramphenicol (chloromycetin) are quite different from that of other antibiotics. There has been a decrease in the percentage of chloramphenicol-resistant strains since 1951. There was a reduction in the use of this antibiotic in 1951 and the percentage of strains which were resistant to 50 micrograms per ml. decreased from 25 per cent in 1951 to 2.5 per cent in 1952 and to 0.65 per cent in 1953. All strains of staphylococci which were obtained in 1953 were resistant to one microgram per ml. and 95 per cent were inhibited by 12.5 micrograms per ml.

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*Acknowledgment is made of the technical services of Miss May Collin in performance of the sensitivity tests.

A summary of the percentages of strains which are resistant to 3.1 mcgm. per ml. of other antibiotics is found in Table II. These strains were also isolated at the Minneapolis General Hospital from 1951 through 1953. Approximately 10 per cent of the strains were moderately resistant to bacitracin each year. Bacitracin was used topically in most cases, and employed parenterally only occasionally in the treatment of patients with staphylococcal septicemia. There has been little evidence of increasing resistance to neomycin, except in 1952, when 25 per cent of the strains exhibited moderate resistance. This was not true in 1953 when only 4.5 per cent were moderately resistant to neomycin.

There is evidence of staphylococci manifesting increased resistance to erythromycin. In 1952 all strains that were studied at the Minneapolis General Hospital were sensitive. In 1953, 21 per cent of all strains examined were resistant to 3.1 mcgm. per ml. The majority of these strains required 200 to 400 mcgm. per ml. for inhibition of growth. Carbomycin (magnamycin) has not been used except in a few patients, but every strain of staphylococcus which has demonstrated resistance to erythromycin in this study, has also been resistant to carbomycin in similar concentrations. This indicates a cross resistance between these two antibiotics.

These data indicate the increasing incidence of antibiotic-resistant strains of pathogenic staphylococci in hospitalized patients. Newer antibiotics are being discovered, but they too are losing effectiveness and the present large numbers of antibiotics adds to the perplexity of the problem of management.

An attempt to explain the mechanism of the appearance of resistant strains is presented in Figure 1. There can exist in a population of antibiotic-sensitive staphylococci a few cells which are naturally resistant. During therapy with antibiotics there is destruction of the susceptible cells and survival of these relatively few resistant cells. The resistant organisms multiply rapidly and replace the antibiotic-sensitive staphylococci. This gives rise to an antibiotic-resistant culture of staphylococci.

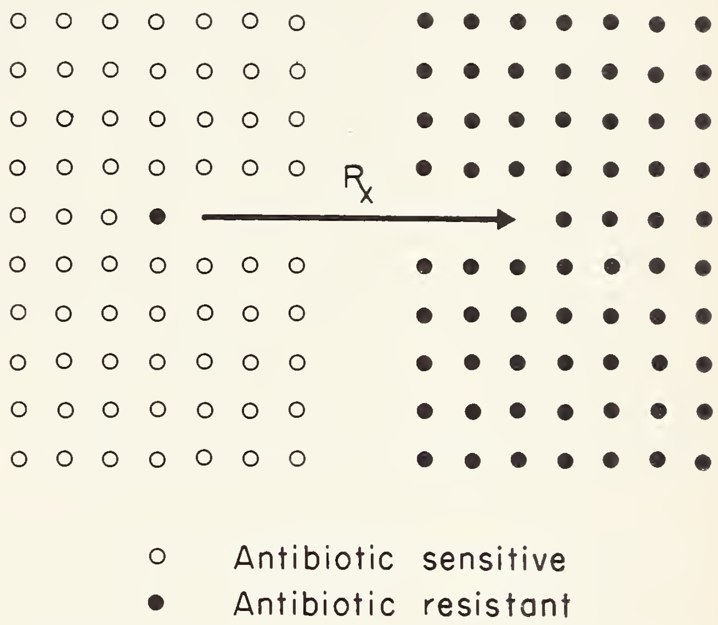

Fig. 1. Mechanism of the appearance of resistant strains of antibiotics.

5. *Epidemiology of Staphylococcal Infections.*—A knowledge of the mechanisms of spread of these bacteria is important, because if therapy of staphylococcal disease is so difficult, every precaution must be exercised in preventing such infections. The primary reservoir of staphylococci is the human nasopharynx. The carrier rate of pathogenic staphylococci in the general population is 25 to 50 per cent, whereas in hospital personnel the carrier rate is 50 to 75 per cent.[1,2,5,10,11,14] Nasal carriers are usually skin carriers of the same strains.[19] The spread of staphylococci from the nose to the intact and abraided skin occurs. The organisms are spread to bedclothes, dressings, dust and instruments.[6] Antibiotic-resistant strains find habitation in wounds and also in the noses and on the skin of hospital patients and personnel.[13] The cycle may be reduplicated many times in a hospital environment. To break this cycle great precautions must be taken in the care of indwelling venous catheters, surgical techniques, dressing of wounds

and burns, the instrumentation of the genitourinary tract and the treatment of staphylococcal infections of the skin.

The problem of antibiotic-resistant staphylococci appearing in a large hospital has been studied by Lepper et al[8] in the Municipal Contagious Disease Hospital of Chicago. Before introducing erythromycin in the wards, the carrier rate of erythromycin-resistant staphlococci in the hospital staff was determined. No strains were found that were resistant to this antibiotic. Erythromycin was then used for the treatment of many infections. After five months 75 per cent of the strains isolated from personnel required over 100 micrograms per ml. for inhibition. These results demonstrate the selection of strains of staphylococci which survive the action of antibiotics and consequently are resistant.

6. *Therapeutic Procedures.*—The concepts of therapy of severe staphylococcal infections must be based upon the factors which have been discussed. The most important point is to know when a staphylococcal infection is present. To begin therapy empirically with antibiotics before making an effort to obtain the etiologic agent is to invite trouble. A blood culture should be obtained from the febrile patient before therapy is started and changes can be made in management later when more information is obtained.

The abscess is a focus which may be impermeable to antibiotics. Bacteria may exist in the abscess for long periods of time, although high concentrations of antibiotics are present elsewhere in the body. The logical approach to this problem is the maintenance of high concentrations of properly selected antibiotics in the tissues for long periods of time, and surgical drainage when possible. This is in contrast to the treatment of infections caused by beta hemolytic streptococci, which usually produce an infiltrative type of infection rather than localized abscesses.

In some instances combined therapy is essential. The rationale of using at least two antibiotics in the treatment of severe staphylococcal disease is based on principles of genetic concepts of resistance. Since resistant cells are either present at the beginning of therapy or they arise by mutation during therapy, it is logical to assume that the chances of the resistant cell being resistant to two antibiotics are less than its chances of being resistant to a single antibiotic.

The antibiotics should be selected by an *in vitro* test of the susceptibility of the causative organisms to the antibiotic. The information gained by use of the tablet or disc method may be misleading in the case of staphylococci. The preferred technique for the staphylococci is the tube dilution method.

The management of the patient during the interval of waiting for results of cultures and sensitivity studies must necessarily be empirical. A presumptive etiological diagnosis of a bacterial infection and the most probable species of bacteria is made on the basis of history, physical examination and preliminary laboratory results. No rules can be made for the use of antibiotics. The tetracyclines or chloramphenicol are useful during this period, since they are active against many of the Gram-negative bacteria and some of the Gram-positive cocci. Concurrent use of one of these antibiotics with penicillin may be a satisfactory choice until bacteriological results are known.

Penicillin is the choice drug providing the causative organism displays *in vitro* sensitivity. Under these circumstances the following is suggested for stapylococcal septicemia: a total daily dose of at least 2 to 4 million units of crystalline penicillin G in aqueous solution is administered intramuscularly in divided doses every 4 hours. In addition, "booster doses" of 1 million units in 10 ml. of saline may be administered intravenously every twelve hours to produce high peak levels. The insoluble penicillin or deport penicillin should not be used in any staphylococcal infection for the prolonged low levels may allow the appearance of resistant forms. Therapy should be continued for a minimum of two weeks after the patient is afebrile. If the patient has normal renal function, bacitracin may be used simultaneously, but a total daily dose of 100,000 units in divided doses should not be exceeded and the urine should be examined daily for albuminuria. Bacitracin should be discontinued if nitrogen retention appears.

If the causative organism is resistant to penicillin and sensitive to erythromycin, then erythromycin should be given orally in total daily doses of 2 to 3 gm. or 500 mg. intravenously every 12 hours. Bacitracin can be administered concurrently with erythromycin.

Since there has been a decrease in the incidence of chloramphenicol-resistant strains of staphylococci in the last two years, this antibiotic may be valuable in the treatment of severe staphylococcal infections. In case the causative agent displays resistance to penicillin and to erythromycin, chloramphenical can be given orally in a total daily dose of 2 to 3 gm. or 500 mg. every eight hours intravenously. Bacitracin can be administered concurrently.

The other antibiotics, such as the tetracycline group, can be used when indicated as a result of sensitivity tests. Streptomycin is not desirable for long-term use because of the rapid appearance of resistant organisms. Neomycin can be used parenterally in a total daily dose of 1 gm. per day but should only be used as a last therapeutic resort, since irreversible deafness and renal damage can occur following the use of this antibiotic for periods necessary in the treatment of staphylococcal sepsis.

In addition, there should be good nursing care, adequate dietary intake, blood transfusions in anemic patients and surgical drainage of suppurative areas when possible.

Summary

In summary, generalized staphylococcal infections are severe and the fatality rate is high. Control and management of these infections should include measures of prevention. The toxic and enzymatic properties of the microorganisms, resistance to antibiotics, and the survival of organisms in abscesses are some of the problems to be surmounted. Treatment will be more successful when the identity of the causative agent and its sensitivity to antibiotics are known. Management should be immediate, aggressive and continued until all signs of infection have subsided.

References

1. Allison, V. D., and Hobbs, B. C.: An inquiry into the epidemiology of Pemphigus neonetorum. Brit. M. J., 2:1, 1947.
2. Barber, M., Hayhoe, F., and Whitehead, J. E. M.: Penicillin-resistant staphylococcal infections in a maternity hospital. Lancet, 2:1120, 1949.
3. Barber, M., and Whitehead, J. E. M.: Bacteriophage types in penicillin-resistant staphylococcal infection. Brit. M. J., 2:565, 1949.
4. Birstingle, M. A., Shooter, R. A., and Hunt, M. R.: Sensitivity to five antibiotics of strains of *Staph. pyogenes* isolated from out-patients. Brit. M. J., 2:253, 1952.
5. Dowling, H. F., Lepper, M. H., and Jackson, G. G.: Observations in the epidemiological spread of antibiotic-resistant staphylococci with measurements of the changes in sensitivity to penicillin and aureomycin. Am. J. Pub. Health, 43:860, 1953.
6. Duquid, J. P., and Wallace, A. T.: Air infection with dust liberated from clothing. Lancet, 2:845, 1948.
7. Forbes, G. B.: Infection with penicillin-resistant staphylococci in hospital and general practice. Brit. M. J., 2:569, 1949.
8. Lepper, M. H., Moulton, B., Dowling, H. F., Jackson, G. G., and Kofman, S.: Epidemiology of erythromycin-resistant staphylococci in a hospital population—effect on therapeutic activity of erythromycin. Antibiot. Ann., 308, 1953-1954.
9. Linsell, W. D.: The antibiotic sensitivity of pathogenic staphylococci. J. Clin. Path., 5:165, 1952.
10. McFarlan, G. M.: Incidence of pathogenic staphylococci in the nose. Brit. M. J., 2:939, 1938.
11. Miles, A. A., Williams, R. E. O., and Clayton-Cooper, B.: The carriage of Staph. aureus in man and its relation to wound infection. J. Path. & Bact., 56:513, 1944.
12. North, E. A., and Christie, R.: Observations on sensitivity of staphylococci to penicillin. M. J. Australia, 2:44, 1945.
13. Rountree, P. M.: Cross infection of surgical wounds. M. J. Australia, 2:766, 1951.
14. Rountree, P. M., and Barbour, R. G. H.: Nasal carrier rates of *Staph. pyogenes* in hospital nurses. J. Path. & Bact., 63:313, 1951.
15. Rountree, P. M., Barbour, R. G. H., and Thomson, E. F.: Incidence of penicillin-resistant and streptomycin-resistant staphylococci in a hospital. Lancet, 1:435, 1951.
16. Skinner, D., and Keefer, C. S.: Significance of bacteremia caused by *Staph. aureus.* A study of 122 cases and a review of the literature concerned with experimental infection in animals. Arch. Int. Med., 68:851, 1941.
17. Spink, W. W., Ferris, V., and Vivino, J. J.: Comparative *in vitro* resistance of staphylococci to penicillin and to sodium sulfathiazole. Proc. Soc. Exp. Biol. & Med., 55:207, 1944.
18. Summers, G. A. C.: Penicillin-resistant staphylococci—distribution among out-patients. Lancet, 1:135, 1952.
19. Williams, R. E. O.: Skin and nose carriage of bacteriophage types of *Staph. aureus.* J. Path. & Bact., 53:259, 1946.

ACUTE POISONING IN CHILDREN

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THE ACCIDENTAL death of a child is the dramatic and tragic finale of someone's mistake. In no other form of accidental death does this fact appear more obvious than in that resulting from acute poisoning. And yet in the United States each year over 1,000 deaths are due to accidental poisoning in children. This is just the number of deaths recorded. It is unfortunate that there are no accurate statistics as to the frequency of poisonings, but it has been estimated that one per cent of all pediatric hospital admissions each year are for accidental poisonings. Even this does not give one a complete picture, for there are many cases of poisoning that are treated in the home, in the physician's office, and as out-patients in the hospitals. There are no statistics to show how many of these represent near catastrophies. Nor are there any figures available to show the number of sequelae of poisoning such as esophageal strictures, blindness, cerebral damage, permanent liver damage, etc. During the year of 1952 the Minnesota Department of Health reported eleven deaths due to poisoning in the one to fourteen years age group. During the years 1951-1952 a private hospital in Minneapolis treated 168 cases of poisoning in children under the age of five years.

It is a well-known fact that causes of death in children have changed markedly in the last twenty years. In 1930 the mortality tables showed pneumonia and communicable diseases to rank first and second, respectively, as causes of death in the one- to four-year age group. Accidents ranked third. With the advent of the sulfonamides, and later the antibiotics, deaths from infectious diseases dropped. In 1954, accidents are the leading killers of our children.

The causes of fatal injuries are illustrated in Figure 1. Although poisonings represent what apparently is a small percentage of deaths, it is well to remember that the incidence of poisoning in children is inadequately represented by mortality tables. Statistics comparable to those illustrated were obtained by the Minnesota Department of Health for the year 1952.

The incidence of poisoning is greatest in the one-year to four-year range. Its peak is in the two-year to three-year age group (Fig. 2). Recalling the development of the child, it is during this period that the motor development is very rapid, enabling the child to walk, climb, pull open drawers, doors and cabinets. A child of this age is an explorer, an adventurer, and a taster. If he can pick it up, he will smell it and/or put it in his mouth.

The manner in which a child may become poisoned may be one of several. It may be by (a) inhalation, such as carbon monoxide or carbon tetrachloride; (b) by absorption through the skin as in the analine dye, mercury, or boric acid; (c) by injection as in errors in dosage of narcotics or sedatives or (d) by ingestion. The latter accounts for the majority of accidental poisonings in children. It may be the inherent nature of a child to put things into its mouth, but parents, and yea, we physicians, encourage this practice. Parents, in an attempt to administer medications to their children resort to various antics and systems of reward to achieve this end. We as physicians have condoned the practice of the pharmaceutical houses in their attempt to disguise their medications to have "eye appeal" and to "taste good like candy."

Symptomatology

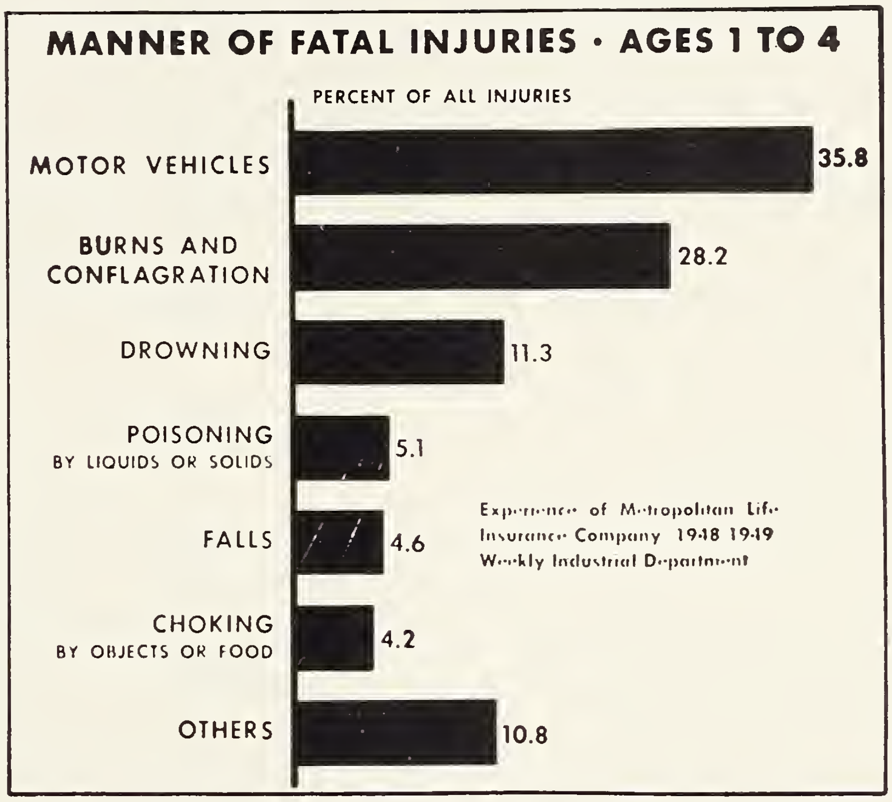
The symptoms of acute poisoning will vary with the poison consumed, but in general will fall into one or more of the following groups.

1. Gastro-intestinal. Nausea, vomiting, and abdominal pain are most common symptoms encountered.

2. Respiratory. This may range from mild hyperpnea as seen in salicylate intoxication to complete respiratory collapse with loss of con-

sciousness as found in barbiturate or nicotine poisoning.

3. Circulatory. This may be manifested by cyanosis, hemorrhagic manifestations, and shock.



Experience of Metropolitan Life Insurance Company 1948 1949 Weekly Industrial Department

Fig. 1. Types of fatal injuries in the one-year to four-year age group.

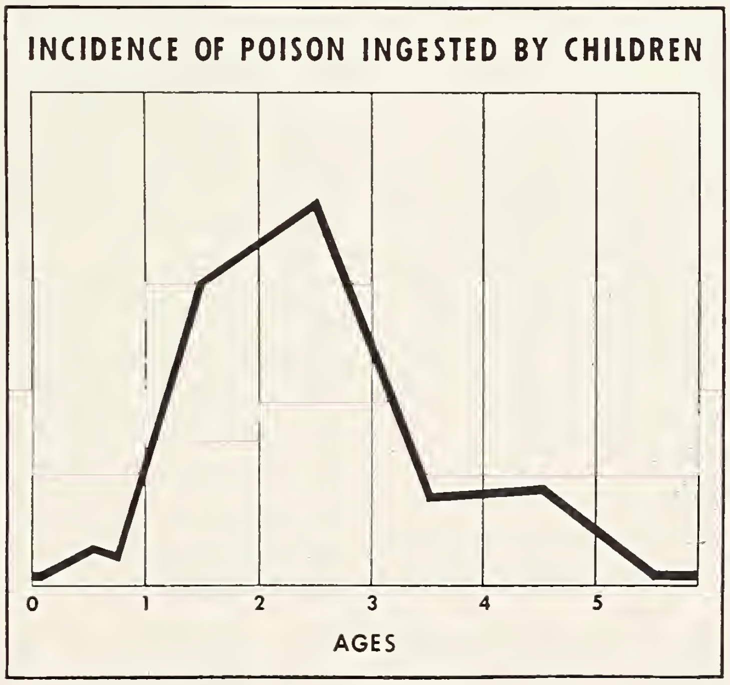


Fig. 2. Incidence of poisonings according to age.

4. Corrosive action. Evidence of erosion of oral and pharyngeal mucosa will occur with the injection of lye, phenol, alkalies and acids.

In any child who presents one or more of the foregoing symptoms, and in whom adequate evidence for their cause cannot be determined, acute poisoning should be considered. The absence of a history of poisoning does not rule out its possibility. It is well to remember that children are too young and usually too frightened to give an adequate and accurate history. The latter may often be said of the parents as well.

Treatment

The treatment of poisoning should be immediate. The child should be dispatched with the greatest expediency to a point, hospital or office, where treatment can be instituted immediately on arrival. It is of great value to have the parent bring the poison container. It may list the ingredients. Too frequently the anxious and overwrought parent forgets or destroys the container only to add additional time before definitive treatment may be started. The treatment may be divided into three stages: (1) removal of the poison; (2) administration of an antidote, and (3) supportive therapy.

Removal of Poison

The method of choice in ingested poisons is gastric lavage. In all cases or suspected cases of poisoning, lavage should be done immediately. There are certain exceptions to this rule. These exceptions are in patients who have consumed lye, ammonia, acids, strong alkali, and phenol. It is also to be avoided in the unconscious or convulsing patient. In the former group it is avoided because of the danger of perforation of the esophagus, and in the latter group because of the danger of aspiration during the procedure. It is the opinion of the author that lavage should be avoided in hydrocarbon (kerosene, fuel oil, and so forth) ingestion because of the increased incidence of bronchopneumonia following the procedure.

The technique of gastric lavage is a simple and well known one. With the child "mummied" or wrapped tightly in a sheet and placed on its side in Trendelenburg position, a mouth gag is introduced and the stomach tube passed. The larger the diameter of the tube that can be passed, the more easily and quickly can this procedure be carried out. Because of the possible and frequent occurrence of emesis during the procedure, suction should be available and used frequently to keep the nasopharynx free of mucus and vomitus. Using a small amount at a time, about 200 cc., the stomach is repeatedly lavaged with the solution of choice using a minimum volume of one liter. If the desired solution is not readily available, one should not delay the procedure. It is wiser to use water than to lose valuable time. Before the tube is withdrawn, the specific or universal antidote is placed in the stomach. The tube should be pinched before its withdrawal to prevent possible aspiration of material in the tube.

The use of emetics should be mentioned. In general, emetics should be avoided because they are usually inadequate to empty the stomach, and they tend to give one a false sense of security. They are usually difficult to get the child to take, and as a result much valuable time may be lost. If used, it should be only as a preliminary emergency measure and should be followed by lavage. Various emetics are available, but the one that is most readily available and effective is warm soapy water—dish water. Others that may be used are mustard water, milk, and egg white.

Antidotes

When one turns to one's therapeutic armamentarium in search for an antidote for a certain poison, one suddenly realizes that there is NOT an effective antidote for every known poison. As a matter of fact, specific antidotes are relatively few. Until the introduction of BAL (British anti-lewisite, 2, 3-dimercaptopropanol) there was no specific antidote for heavy metal poisoning. BAL is of value in heavy metal poisoning (mercury and arsenic) if administered early. It has no value if tissue damage has begun. BAL is dispensed as a 10% solution in peanut oil and is to be used for intramuscular injection only. The dosage is 2.5 mgm/Kg/ injection and given at four-hour intervals for twelve injections, then every six hours for four injections. BAL may produce toxic reactions such as lacrimation, salivation, nausea, vomiting, tachycardia, rise in blood pressure, and pain at the site of injection. The use of ephedrine sulfate in doses of 25 mgm. orally with each injection will help to offset some of these unpleasant side effects.

When no specific antidote is available, or when the nature of the poison is unknown, the use of the Universal Antidote is indicated. This should be placed in the stomach before the removal of the lavage tube. The constituents of this antidote are as follows:

Charcoal	2 parts
Magnesium oxide	1 part
Tannic acid	1 part

The charcoal will absorb the phenols and strychnine; magnesium oxide will neutralize the acids; the tannic acid will precipitate the alkaloids, glucosides, and some heavy metals. The function

TABLE I.

Poison	Common Source	Acute Symptoms	Treatment		
			Lavage	Antidote	Supportive
Strychnine	Cathartics, insecticides, rodenticides.	Central nervous system stimulant, hyperreflexia, opisthotonos, convulsions.	If no convulsion 1: 10,000 potassium permanganate.	No specific, use universal antidote.	Sedation, oxygen.
Arsenic	Insecticides, rodenticides, plant sprays, some paints.	Construction of throat, abdominal pain, projectile vomiting, diarrhea ➤, rice water stools ➤, bloody stools, convulsions and/or coma.	1% sodium thiosulfate.	BAL.	Morphine for pain, stimulants, treat for shock.
Lye	Draino pipe cleaner, toilet bowl cleaner.	Burning pain in mouth and stomach, mucous membranes soapy and ulcerated, bloody vomitus, collapse.	No and no emetic.	Dilute vinegar solution or lemon juice, olive oil for pain.	Fluids.
Sodium Hyposulfite	Sani-Flush, Clorox, bleaching agents, washing powders.	Vomiting, corrosive burns of lips, mouth, and tongue.	If no ulceration weak vinegar soln.	No specific.	Fluids.
Salicylates	Aspirin, oil of wintergreen.	Hyperpnea, listlessness, vomiting, dizziness, mental confusion, acidosis, hemorrhagic manifestations.	Milk or 1: 10,000 potassium permanganate.	No specific, instill saline cathartic in stomach after lavage.	Oral or parenteral fluids, watch electrolyte balance.
Barbiturate	Sedatives.	Somnolance ➤, stupor ➤, coma, respiratory and circulatory collapse.	If conscious.	Picrotoxin 1 to 10 mgm IV at 15 to 30 minute intervals. Instill saline cathartic in stomach after lavage.	Fluids, stimulants, oxygen.
Hydro Carbons	Kerosene, gasoline, naphtha, benzine, cleaning fluids. Fuel oil.	Burning pain in mouth, nausea and vomiting, drowsiness, confusion, fever. Aspiration is common ➤, bronchopneumonia.	No.	No specific.	Oxygen, fluids, antibiotics.
Fluoride	Insecticides.	Excess salivation, abdominal pain, hematemesis, shallow respirations—respiratory collapse.	Calcium chloride or milk.	No specific.	Fluids, calcium lactate IV.
Carbon Tetrachloride	Cleaning fluids, fire extinguishers, solvents for oils and fats.	Nausea and vomiting, headache, inebriation, convulsions. Late effect is liver necrosis.	1:10,000 potassium permanganate.	No specific.	Oxygen, fluids, high protein and carbohydrate diet.
Nicotine	Insecticides, plant sprays, tobacco, Black Flag.	Nausea and vomiting, mental confusion, salivation, abdominal cramps, convulsions, coma, respiratory failure.	Tannic acid or 1:10,000 potassium permanganate.	No specific.	Oxygen, stimulants.
Mercury	Antiseptics, fireworks, insect spray, also tablets.	Abdominal pain, vomiting, bloody diarrhea, circulatory collapse, kidney damage ➤, oligures ➤, anuria.	Copious amounts of milk.	BAL.	Fluids.
Analine Dye	Yellow and orange crayons, shoe polish	Apathy, dyspnea, cyanosis due to methemoglobinemia.	Water.	1% methylene blue IV (.2cc/Kg).	Oxygen, whole blood.
Ammonia	Household ammonia.	Burning in mouth and stomach, nausea and vomiting, abdominal pain, respiratory failure.	No.	Weak acids, olive oil.	Fluids.
Phosphorus	Rodenticides, roach poison, fireworks, imported matches*.	Nausea and vomiting, abdominal pain, diarrhea, shock, garlic odor to breath, liver and kidney damage.	2% copper sulfide then 1:10,000 potassium permanganate.	No specific.	Fluids.
Camphor	Camphorated oil, moth repellents.**	Odor to breath, headache, excitement ➤, delirium ➤, convulsions.	Water.	No specific.	Sedation, no opiates.
DDT	Insecticides.	Nausea and vomiting, weakness, vertigo, disorientation, coma.	Water.	No specific.	Fluids, sedation, Ca gluconat.
Cyanide	Rodenticides, metal polish, silver polish.	Odor of bitter almonds on breath, giddiness, headache, cyanosis, coma acts rapidly.	Early, 1: 10,000 potassium permanganate.	Immediate treatment may be of some value. Principle of treatment is to produce methemoglobin which in turn combines with the cyandie to form nontoxic cyanmethemoglobin. Inhale amyl nitrite pearl. Follow with 10cc of 10% Na nitrite IV.	

*Matches made in the U.S.A. are now made with insoluable phosphorus trisulfide and are relatively harmless.
**Many moth repellents now contain paradichlorobenzene which is relatively nontoxic.

of this antidote is to render the poison innocuous or unabsorbable. One cannot err in using this as an adjunct to all other therapy.

There are over 400 agents that have been known to cause acute poisonings. However, only a few of these are responsible for the majority of deaths. Although it is not the purpose of this paper to discuss the treatment of each poison, Table I outlines the most common poisons, their sources, symptoms and treatment.

Supportive Therapy

Close observation of all patients who have ingested poison is imperative regardless of the initial therapy. Hospitalization for at least a twenty-four-hour period is advisable, for delayed action such as shock, electrolyte imbalance, cyanosis, et cetera, may follow in spite of what may seem adequate therapy at the time. The judicious and propitious use of oxygen, blood, electrolytes, stimulants and analgesics may not only be lifesaving, but will also render the patient more comfortable.

Prevention

A discussion of poisoning in children is not complete without emphasis on prevention. It has been shown that accidents are now the greatest killer of our children. We have seen that deaths from accidental poisonings contribute significantly to this mortality. Of equal importance is the morbidity. A discussion of prevention of poisonings naturally overlaps into the field of all accident prevention in children. Significant advances in accident prevention have been made by such organizations as the National Safety Council, American Academy of Pediatrics, Children's Bureau, life insurance companies, and locally the Minnesota Department of Health. However, the campaign for the prevention of poisonings can be brought directly into the office of the individual physician. He can and should become the most ardent campaigner for this cause. He can awaken the parents to the possibility of poisonings in the home. He should lay the facts before the parent—the morbidity, the mortality, the serious sequelae. Emphasize the fact that 99 per cent of accidental poisonings are due to the neglect of the parents. A child cannot read the poison label. Specific instructions by the physician to the parents to carry out a household inspection on their arrival home will be rewarding. Have them open drawers, doors, cabinets in a search for "poison possibilities." The Minnesota Department of Health has available pamphlets and brochures for distribution to parents. This "gospel of prevention" should be given when the child begins to crawl, or preferably before. The physician may enhance the prevention program in another way. He can discourage the use of candy-like medications such as aspirin, and advise against use of over-the-counter and dubious home remedies.

The role of the physician in prevention is well summarized in the words of Dr. H. F. Dietrich: "If we don't accept this responsibility, remember that accidents may be the killers and parents may be the accomplices, but we the medical profession will be the accomplices before the fact."

A Poison Control Program

Although the primary responsibility of prevention and treatment of acute poisonings lies with the parents and the physician, the community can and should take an active part in such a program. A poisoning control program similar to that instituted by the University of Illinois for the city of Chicago could be constructed. The purpose of such program would be to furnish physicians and hospitals with current information on the diagnosis and treatment of poisonings. A current index could be compiled and maintained that would list the constituents of all proprietary drugs, patent medicines, home remedies, and household articles such as cleaning fluids, bleaches, insecticides, metal polishes, and so forth. This information would be available on the call of any physician or hospital. In addition to this, all cases of poisoning treated in the doctor's office, as an out patient in the emergency room, or in the hospital would be reported to this control center on a specially developed standard form. The compiling and analysis of this information would yield important data as to the type and frequency of poisoning. It would permit a better evaluation of treatment. It would allow when indicated, follow-up contacts to help correct those situations predisposing to the accident. It would expedite proper legislation to control the sale and distribution of poisonous materials. In addition to the foregoing, a poison control program would promulgate a campaign of prevention.

The nucleus of this program would be a committee composed of representatives—physicians when possible—from the city health department, state health department, department of pharmacology and toxicology of the University, department of pediatrics of the University, and of the department of internal medicine of the University. Until such time as a central treatment center for the community could be established, represented on this committee should be physicians delegated by staff of each hospital in the community. These physicians in turn would head the committee on poisonings in his respective hospital. In this manner all hospitals would be active participants in the control program. The above

(Continued on Page 897)

RAUWOLFIA

PAUL DE KRUIF
Holland, Michigan

IT WAS my first intention to make the title of this talk, "Rauwolfia—or how not to blow your top." But now it's just Rauwolfia. Your periodical, MINNESOTA MEDICINE, has asked me to submit the present talk for publication—and the vernacular in the first title would look bad in the table of contents, and might even scare the editors away from printing the story. For me there is a vanity that what I write for plain people might also be of some interest to the doctors. So, if this evening's entertainment turns out to be a bit stuffy, you now know why. Yet, it's going to be hard to be stuffy about this ancient and humble little shrub, *Rauwolfia serpentina.* The powder from its root is making miracles, even though they're slow and unspectacular. Rauwolfia is giving general practitioners a new, unexpected, unhoped-for chance to save life, to restore broken-down people to work, and, maybe, to prolong notably their span of productive vitality.

To this Rauwolfia hints a dividend. It gives family doctors a new weapon against anxiety. What all the psychoanalytic gobbledegook of neuropsychiatric specialists has had a hard time accomplishing, to the plain general practitioner Rauwolfia gives something powerful and tangible. Psychotherapy in pill form.

But forgive me, I'm getting ahead of my story. What originally excited me about Rauwolfia was an article by Ford and Moyer, of Houston, Texas, in GP published in November, 1953. It told how Rauwolfia has at last put the treatment of high blood pressure—including severe forms of it—into the hands of general practitioners. Four years ago this was beyond the power of the smartest internists; and the stakes the family doctors are going to play for in this particular grim game of life-or-death, are high—a fifth of all deaths in our country are due, directly or indirectly, to the consequences of hypertension.

And now Rauwolfia is going to put plain GP's into the front line in such a fight? With real power? What a story.

Yes, and what a tough story to write. When you set out to peddle hope among 17,500,000 people buying the Reader's Digest, you'd better be sure. And here's the trouble. Hypertension, of course, *can* be harmless; many people live out a normal life span unaware they have it. And there are big-shot internists, notably in New York, who'll tell you of old ladies—pressure 210/120 for years—living hale and hearty till eighty-five. Then dying—of cancer, not hypertension.

Certain of these big clinicians believe it best hypertension be kept a secret from their patients. Out of one side of their mouths these internists imply high blood pressure isn't going to kill you. Out of the other they say knowing you've got it will scare your pressure higher, and that might be dangerous. This confused me.

Then too, what about this new-fangled, old drug, Rauwolfia, and the new treatment it makes possible? In 1953 the eminent *Lancet,* of Great Britain, editorialized that chemical treatment of hypertension was so difficult that clinicians could still do no more than keep their patients' pressure a secret.

On the other hand there was information—grim and reliable—that people with essential hypertension with onset at age thirty-two, on the average only lived till fifty-one. Then dying—of the consequences of high blood pressure. Now at Reader's Digest, if you want to get a story into it, there's one thing you'd better not be, and

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Presented at the annual meeting of the Northern Minnesota Medical Association, Willmar, Minnesota, September 10 and 11, 1954.

EDITOR'S NOTE: The editor considers it a great privilege to publish an article written by Paul de Kruif, dean of medical science writers. In spite of the widely-experienced irritations due to inquiring patients stimulated by his many contributions in the lay press, physicians have learned and gained much from his writings. There is probably no single person in our time who has presented the benefits of good medical care to the public more ably than Paul de Kruif.

Paul de Kruif is roving editor for *Reader's Digest.*

that's confusing. The project seemed stymied before it got started. What to do?

It was my good luck to find a couple of clinical investigators in the hypertension field who talked not out of both sides but straight out of the middle of their mouths. These two fellows—whose discoveries will make the burden of the story—became my briefing-officers. And now, physicians that you are, you must bear with me if I talk very simply.

Forgive me, too, if I digress for a moment to explain why I'm going to stick to A B C's. At a welcome Travelers broadcast, not long ago, the M.C., Tommy Bartlett, asked me: "How come you can write complicated medical science to millions of people?"

"Only because I'm so dumb," I answered.

"What do you mean dumb?" asked Tommy.

"To get to understand that highbrow stuff," I said, "being really dumb, I've got to boil it down to the littlest words. When it's clear to me, dumber than dumb, it'll be clear to *anybody,* no matter how low their I. Q."

So, in what follows, forgive me if I talk up to you instead of down to you. Getting going on the investigation for the story, I boiled down heavy scientific publications to the simplest questions, asked of my briefing-officers.

"If so many can live years, not knowing they're hypertensive, how can you fellows be sure a given case is dangerous?" I asked them. They both agreed they could not be, but there were clues. It's more likely to be serious when it runs in families or starts early in grownup life.

"How do you diagnose serious hypertension?" I asked them. That was tough, they agreed. It starts insidiously. Only a minority have early symptoms (notably fatigue) sending them to a doctor. Most hypertension is stumbled on at insurance, and other routine examinations.

But what makes high blood pressure dangerous? My briefing-officers explained the heart has to pump harder against a chronic, rising pressure. Till at last its muscle may fail. And when the pressure stays high, for years, it *aggravates* thickening and deterioration of arteries. If the heart's arteries plug up, there is a coronary accident; if the brain arteries plug or burst, there's a stroke; and damage to kidney arteries may result in fatal uremia.

This sinister trinity, my teachers said, is the king-killer, responsible for the death of 320,000 Americans yearly.

Then, in my simplicity, I popped my $64 question—

"What *is* it that makes blood pressure go up so dangerously?"

One of my briefing-officers, Dr. Robert W. Wilkins, of Boston, tried to explain it. "It's a complicated mechanism," said Bob, "involving the sympathetic nervous system, endocrines, muscle tension, a possible toxin from the kidneys." Bob's words were pretty big. "Give me a handle to hold on to," I asked.

Well, one factor was clear, explained Bob. A great majority of cases start from deranged nerve cell activity causing a chronic spasm of the blood vessels. Overactive impulses go from deep in the brain out over the sympathetic nervous system, which, independently of our will, controls organs, glands, blood vessels. These super-active nerve impulses narrow down the arterioles, the little valves at the ends of the arteries; so the heart's got to pump harder to circulate the blood, and so shoots up the pressure.

My other briefing-officer, Dr. John Moyer, of Houston, Texas, backed up Bob Wilkins. "We don't know the underlying cause of hypertension," explained John. "But a common denominator is over-activity of the sympathetic nervous system."

"How do you know?" I asked. "Because when you interrupt the outflow of those nervous impulses, down goes the pressure," said John.

"Therapeutic nihilists may say high blood pressure in itself doesn't kill anybody," said Bob Wilkins. "But that's a quibble, like saying, does a man die of too much acid in his stomach when he has an ulcer, or does he die because acid causes the ulcer and makes it bleed and he dies of bleeding to death."

"I don't get the analogy," I said.

"Well," said Bob, exasperated. "High blood pressure doesn't kill you, but when you have it year after year, it *does something* to you. Your blood vessels get so shot, they can't stand the pressure, in the heart or brains or kidneys, so you say 'I'm not dying *directly* of high blood pressure, I'm only dying of the *effects* of high blood pressure.'

"It's a damned quibble," said Bob.

If John Moyer and Bob Wilkins are right, if the sympathetic nervous system was in part to blame for high blood pressure, why not cut its nerves, blocking overactive impulses?

That question flashed my memory back to what was, for me, a momentous day in 1933. That day the late great Dr. Max Minor Peet, neuro-surgeon at the University of Michigan, excited like a boy as he always was about his work, told me how he'd just asked that question, and then acted on it. He risked a new operation, cutting out the thoracic ganglia of the sympathetic nervous system of a man terribly hypertensive and about to die. The patient's pressure had dropped to normal, his vision returned, he was back to work, as good as dragged from the grave.

That patient worked, in good health, for many years, outliving Max who had saved his life. Max Peet had proved the sympathetic nervous system was not necessary to life—and that was a break for doomed hypertensives. He'd proved the basic fact that there's a neurogenic factor in hypertension. His now famous sympathectomy operation—extended and made more drastic by Dr. Reginald Smithwick of Boston and Dr. Keith Grimson of Durham, North Carolina—has saved thousands of hypertensives threatened with early death.

Yet for millions of *not yet desperately ill* hypertensives, sympathectomy was not the answer. Many wouldn't face it. Too risky. They preferred to take their chances. Many kept dying.

Then came a synthetic drug from England by way of New Zealand; hexamethonium, blocking the sympathetics, chemically. It knocked all high blood pressures, no matter how terrific, down to normal. From Switzerland from Ciba Laboratories came hydralazine. Both chemicals were sensational . . . too sensational.

In 1950 Dr. Edward D. Freis, in Washington, D. C., and Dr. Henry A. Schroeder, in St. Louis, combined hexamethonium and hydralazine into a double-barrelled cannon. To shoot at the sympathetic ganglia of malignant hypertensives, who were going to be dead in a year, or not much more. Why did Freis and Schroeder give the new treatment such a terrible test?

Because the new treatment, too, was super-drastic. Hexamethonium—"dangerous hex" it came to be called—drove the highest blood pressures down so low so fast that patients often fell on their faces, flopped on the floor and passed out cold. Hydralazine caused severe headaches, nausea and fast pounding of hearts already likely to be damaged.

But Freis and Schroeder, like doctors everywhere, helpless, had watched malignant hypertensives die. So they risked it. Out of 100 of Schroeder's malignant hypertensives, 63 have survived from one to two years. Pressures? Back, practically, to normal. No further damage to hearts, brains or kidneys; 57 back working. Freis's results were similar.

But these resurrected ones lived under a sword; 11 stopped treatment; within a few weeks all were dead. If only they'd stuck with it? But sometimes it was too tough to take.

Hexamethonium-hydralazine was only for patients in hospitals and under close supervision of experts. For many it was too high a price to pay for living; it was definitely no medicine for busy practitioners to use on hypertensives, generally.

Then out of the mists of Indian oblivion arose Rauwolfia. For hundreds of years the powdered root of this lovely shrub with its bright green leaves and pinkish-white flowers had been hawked in the bazaars as a cure for lunacy, diarrhea and snake bite. It is reliably reported that Mahatma Ghandi, by chewing on Rauwolfia root, gained the calm that drove the British so wild that they went away, without a struggle, giving India back to the Indians.

In 1950, Dr. Robert W. Wilkins, of Boston, read an article by Parsee Dr. Rustom Jal Vakil who said that Rauwolfia was good for hypertension, too. Wilkins was not so God-Almighty that he couldn't take a tip from beknighted Asians. Wilkins ignored the crazy claims; and tried Rauwolfia—on hypertensives.

Just as all previous hypertensive drugs had been tested, he gave it in single, trial doses, bigger and bigger. Nothing happened. Now all really powerful blood pressure-lowering chemicals act quickly, if at all. Wilkins nearly threw Rauwolfia out the window as just another one of those things.

But one of his patients, a Negro lady, Carrie Brooks, she should be historic, *happened,* repeat *happened,* without design, to be given Rauwolfia pills not once, but five days in succession. Did a resident misread directions on the chart? We don't know. But on the sixth day her extremely high, fixed blood pressure was down to normal. That accident started Rauwolfia on its American adventure.

Wilkins was astounded by Rauwolfia. It took three to six days of q.i.d. doses to show any effect at all. It took from three to six weeks to reach peak activity. After it was stopped, it was one to three weeks before its effects wore off. In all pharmacology, this was unprecedented. In contrast to other chemicals that knocked blood pressure down violently, Rauwolfia was gentle. It lowered pressures, modestly. It brought only thirteen of Wilkin's first thirty-nine cases down to normal.

Rauwolfia was really uncanny. A physician brought one of his patients to Wilkins. She was severely hypertensive—pressure 250/150—banging occipital headaches—ready to blow her top—E.K.G. signs of heart strain—veratrum lowered her pressure a little, but at the expense of severe vomiting. At Wilkin's suggestion this woman's doctor added Rauwolfia to the nauseating veratrum.

Over months, her pressure started slowly down. Soon after that, veratrum could be cut to doses that were not nauseating. That was in 1950. Today this businesswoman is at full activity, blood pressure normal, on small daily doses of veratrum and Rauwolfia.

There was a weird side-effect of Rauwolfia on this lady—it changed her personality. She reported: "I just don't seem to have to do the things I used to. Before Rauwolfia, I *had* to do things. Now I don't care. Dust doesn't annoy me anymore. Before, I knew it wasn't important, but even though tired, I had to clean it up the moment I got home from work. Now I can let it go till next day."

It happened that this lady detested her husband so that her own doctor refrained from mentioning his name because of its immediate effect on her dangerous blood pressure. Wilkins made an experiment. He got her going on her husband's sorry characteristics. Under Rauwolfia's gentle sway, she remained calm as a May morning; her pressure stayed normal.

Among hypotensive drugs, Rauwolfia is exceptional. It is no blocker of the sympathetic nervous system. It acts on emotions, in their power-house, the hypothalamus, in the bottom of the brain. It's more than a mere sedative. In already heavily sedated, even anesthetized dogs, it lowers their blood pressure and slows their hearts.

On hundreds of patients, Wilkins has watched the Rauwolfia effect; and it's no doper-upper, like phenobarbital. It's unique in medicine—a tranquilizer. And it makes up for its modest lowering of blood pressure by an amazing effect on hypertensive symptoms. Rauwolfia soothes the headaches, dizziness and wooziness, pulls butterflies out of the stomachs of anxious neurotics. It's no soporific. Patients tell Wilkins: "It doesn't put me to sleep but it lets me go to sleep, if I'm sleepy."

Wilkins observed that, though it calms people, it doesn't turn them into zombies. Patients under Rauwolfia reported they might go to sleep in church if a sermon was dull, but were right in there, watching a prize-fight on TV.

Bob Wilkins discovered another trait of this strange Rauwolfia. It had a way of taming the bad effects of the more powerful hypotensive drugs that still had to be used against severe hypertension. It slowed down hearts pounding hard and fast from hydralazine (Apresoline). In other patients besides that businesswoman, it cut down veratrum to non-nauseating doses still effective against high pressure.

Wilkins rejoiced to find that Rauwolfia's own side-effects were trivial. Some stuffy nose, dreams more vivid than usual, overactive intestinal elimination, some depression when doses were too high; all reversible by withholding the drug for a while or cutting the amount of it down.

Bob Wilkins is one of those clinical investigators who does not live in an academic world of his own; he is always looking for treatment usable by general practitioners. "Don't expect dramatic results from Rauwolfia," he kept telling them. "Wait for several weeks or months to judge its effectiveness."

Here was another merit of Rauwolfia, important to the family doctor. Often less and less of the more powerful, dangerous hypotensive drugs could be given, when they were bolstered by gentle Rauwolfia. Till at last, this safe Rauwolfia, all alone, in certain cases though not in all, held pressures down to normal.

Aside from these qualities, another action of this mystical Indian powder haunted Bob Wilkins. For example an executive, his formerly very high pressure now normal, on Rauwolfia alone, reported—"There are things that in the old days, bothered me. Now I refuse to let them disturb me. Last year a man would make a mistake at the factory and I'd blow my stack. Now I just show him the right way to do it. This stuff is fabulous!"

This executive's wife was amazed at the change in her man. "He used to get furious, cursing out bum drivers on the road. Not any more. What's *happened* to him?" she asked.

How far did the power of Rauwolfia extend? Surely this Indian medicine, so gentle in its action, wouldn't be able to tame the most powerful of all hypotensive drugs—hexamethonium—dangerous hex—that, alone, could bring super-high pressures down, when all else failed? But dangerously, too dangerously.

Dr. John H. Moyer of Houston, Texas, put Rauwolfia to this sternest of all tests, on a forty-seven-year-old woman who was having a rugged time, trying to stay alive, on hexamethonium alone. One year before, dangerous hex had rescued her, in coma, near blind, in hypertensive crisis and about to die, blood pressure 300/200.

Hexamethonium shot her deadly pressure down to normal and way down below normal till almost no pressure at all. "Get your wife away from those Baylor University docs, they're goin' to kill her," their family physician told the lady's husband. But her husband overruled the doctor. He knew her banging headaches were gone; she was home; she could see again; at least she was alive.

But now John Moyer added Rauwolfia to the dangerous hex. The wild up-and-down swings of her pressure steadied. In a few weeks she needed only half of hexamethonium's former dose to keep her blood pressure close to normal. In a few months she no longer needed dangerous hex at all. Just Rauwolfia, a few safe tablets daily.

It was my honor to meet this patient. I have never seen a lady more serene, relaxed, more obviously healthy and lovely—three years after she should have been dead.

Among twenty-seven similar surely doomed people, brought out of hypertensive crises by combined Rauwolfia-hexamethonium, laboratory tests have given the Moyer group evidence of arrest, and actual reversal of deterioration of the arteries of the heart, the eyes and brain.

At the Jefferson Davis Hypertension Clinic, Moyer and his co-workers have tested hundreds of hypertensives—most of them far more severe than the average found in private practice—in parallel treatment series, using Rauwolfia alone or in various combinations with hydralazine, veratrum or hexamethonium. They report blood pressure significantly lowered in nine out of ten; brought down to normal in nearly half; and seven out of ten, incapacitated, are back at work.

What about the very early, mild hypertensives, who are predominantly seen by general practitioners? Moyer reports 63 per cent of these respond, when kept on Rauwolfia alone, for more than a year, now. Even in early but moderately severe hypertensives who respond initially, Rauwolfia for a year has kept their blood pressure normal in forty out of fifty. In the remaining ten whose pressures broke over a bit, they were quickly and easily brought back within bounds by safe, moderate doses of the more powerful hypotensive drugs, added to Rauwolfia.

Bob Wilkins points out that these results open a whole new vista to general practitioners, who alone see this twenty-year disease at its start when it's symptomless or mild. Now the family doctor has a golden chance to prevent essential hypertension from becoming serious, and terminating fatally, as has heretofore been inevitable.

Now that they can safely treat early high blood pressure in their offices, general practitioners can start their own preventive war against this king killer.

In a recent issue of the *Journal of the American Medical Association,* John Moyer sums up the promise of the Rauwolfia revolution, as follows: "One has every reason to believe the disease may now be controlled in its incipiency; that serious complications may consequently be prevented; that combined drug approach to management of severe cases may prevent their progression; and that it may reverse previously inevitable advance of co-existent vascular disease."

Robert Wilkins points up Rauwolfia's sudden upset of the sad hypertension situation: "Less than five years ago when we were asked to lower serious hypertension with drugs, we had nothing to offer," he writes. "Now no case can be called impossible to treat until so proved."

Of the millions of Americans for whom these words may mean a new lease on life, all—except a possible ten per cent of malignant hypertensives who must be hospitalized—can be treated by general practitioners in their offices. If—and I'm speaking to you doctors now—this can be done, it can be done only if you will take the time to learn this not too difficult science. And you alone, as personal physicians to your patients can follow the results.

And this brings us to Rauwolfia's dividend, its curious effect on anxiety. Again and again during the past five years Bob Wilkins has been struck by this eerie medicine's transformation of beat-up hypertensives. Many told Wilkins—

"I've *never* felt so well," or "This is how I *dreamed* of feeling," or "*Nothing* bothers me now" or "I just-don't *give* a damn."

What was the common denominator of these testimonials to Rauwolfia? It wasn't that the patients were no longer hypertensive. It was that they were no longer *anxious*. And if anxiety is a seven-letter-word for the four-letter-word, fear; and if fear is an emotion; and if emotions arise in that little nest of nerve cells in the hypothalamus at the bottom of the brain; and if—as it seems from experimental pharmacologic evidence—Rauwolfia is a hypothalamic regulator—then why not try Rauwolfia on anxiety? Pure cases of anxiety, devoid of high blood pressure?

Again, the benighted Hindus had scooped Bob Wilkins on his intuition. And not Hindu doctors either, but anonymous low-down laymen had antedated him. For hundreds of years, Rauwolfia, under the people's name for it, "Pagal-Ka-Dawa" had been sold in bazaars—across-the-counter—for insanity. Utterly unscientific. Pure folk medicine. Then the Hindu Dr. Hakim, a few years ago, proved his lay brothers had something.

Then Rauwolfia went highbrow on its anonymous discoverers, went scientific, became medically respectable. In Basle, Switzerland, Ciba chemists crystallized a pure alkaloid, reserpine, out of the yellow powder of the crude root of this Pagal-Ka-Dawa which means "insanity herb" to Hindus.

And these reserpine crystals can be injected by vein or into muscles of human beings, safely, in doses huge compared to those used in hypertension. And at Modesto State Hospital in California, this was done, last year, by Drs. Robert H. Noce, David B. Williams and Walter Rapaport.

Their first human experimental animals were hopelessly psychotic, picked from what are called "maximum security wards," sinister halls in continuous turmoil due to raucous, hyperactive, combative, sarcastic, resistive, uncooperative patients.

Then by injection and by mouth, into this cohort of the stark, staring, raving insane, went reserpine, trade name, "Serpasil," pure alkaloid of Rauwolfia.

Over the bedlam of the maximum security ward came a change. In this "back ward" of the madhouse there began to reign, tranquillity. Noisy patients became quiet and calm. Depressed patients became alert and cheerful. On the Serpasil wards, restraint, sedation and electroshock have been almost eliminated. In 80 per cent of patients the improvement was real, even striking. Already, 20 per cent of those hopeless psychotics are in full remission and eight have been discharged; and the longer the treatment is maintained, the greater the number of remissions.

A report on this work has been accepted for publication by the *Journal of the American Medical Association*. The effect of Rauwolfia on the mentally disturbed is under study, nationwide, by more than 100 investigators.

Along with the remarkable new synthetic chemical, chlorpromazine (that's another and maybe a similarly hopeful story) reserpine means hope for many of the insane.

But psychotics weren't Bob Wilkins' worry. His concern is for the simply anxious. Not the anxiety that can be treated because it's due to malnutrition, Brucellosis or this or that vitamin or hormonal deficiency. Wilkins was thinking of the army of neurotics with worries that psychotherapy may suppress but can't cure; neurotics who are still anxious, after it's been explained to them, as they lie on a couch in a darkened room, once a week at so much an hour for two or three years, that their anxiety is due to having been in love, as infants, with their papas or their mamas.

Bob Wilkins' interest is with pure, seemingly causeless anxiety, with those neurotics who explain to their doctor they're worried because they haven't a thing to worry about, saying "What I'm worried about is really nothing. So there must be something wrong with my mind."

Would Rauwolfia or its pure alkaloid, reserpine, put a chemical block into this vicious circle? On a growing number of these anxious neurotics Wilkins and many other physicians are testing it; successfully, in many. They're finding that Rauwolfia is a drug against fear.

This is a new challenge to general practitioners who are fighting, not too successfully, with

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THE DESMOID TUMOR

Two Case Reports with Methods of Repair

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DESMOID TUMORS are benign fibrous tissue neoplasms which arise from the musculoaponeurotic structures throughout the body, and have the significant feature of locally invading adjacent muscle tissue and engulfing such tissues with actual replacement of the striped muscle fibers. The first description of this condition was in 1832 by MacFarlane[6] and in 1848 Mueller[7] coined the term "desmoid" from the Greek words "Desmos" meaning a band or tendon and "Eidos" meaning appearance. Thus the actual derivation of the word comes from tendon-like.

Location

As stated in the definition, these tumors can be found almost anywhere that striated muscle is found or in its aponeuroses. The most frequently reported locations are, however, in the abdominal wall musculature and the tumors are most commonly seen in relation to the rectus muscle below the level of the umbilicus. It has been stated by Stone[10] that the right lower part of the abdominal wall is involved more frequently than any other location.

Incidence

Desmoid tumors are somewhat of a rarity. This can be shown by the fact that Musgrove and MacDonald[8] found only 120 cases in the files of the Mayo Clinic from 1906 through 1945 inclusive. Of these, eighty-five were abdominal desmoids and forty-five were located elsewhere. Labbe and Remy[5] stated that Bilroth saw only sixteen abdominal desmoids in sixteen years. Stone[10] reported only five desmoids in 21,000 surgical patients at Johns Hopkins Hospital. Pack and Ehrlick[6] encountered only seventeen desmoid tumors of the abdominal wall from 1917 to 1943 in 50,346 patients with neoplasms. This was an over-all incidence in their series of 0.03 per cent.

This tumor is most commonly seen in women during the child-bearing years from twenty to forty, whereas in males such growths, though very uncommon, usually come on later in life. These tumors have been very rarely seen in childhood and one congenital case was reported by Brockman.[1]

Etiology

Muscle trauma is considered to be the chief factor in the development of the desmoid tumor. Such injuries as muscle tears and stretching, which occur during pregnancy and labor, offer an explanation for the occurrence of these tumors during or shortly after pregnancy. The tumors also occur in old surgical scars, also showing trauma as being basic to their etiology. A patient was described by Green[4] in which a desmoid tumor developed in the inguinal canal of a patient, who had worn a truss for a period of ten years.

It has been suggested in the literature that there is a relationship between sex hormones and desmoid tumors. Geshickter and Lewis[3] have described a similiarity between desmoid tumors and fibromyomas of the uterus as well as fibroadenomas of the breast. They found that all three of these tumors have a special ability to concentrate pituitary gonodotropin substance and estrogenic substance, and mentioned that a biologic assay of one desmoid tumor yielded 13,000 rat units of gonadotropin per kilogram.

Pathology

Clinically, desmoid tumors are somewhat similar to fibroid tumors in other locations, except that there is no definite capsule. These tumors are generally described as being densely hard, rubbery in consistency and cut with a grating sound. Small to medium size tumors usually are of whitish-pink in color and appear somewhat glistening on cut surface. The larger tumors may show areas of degeneration due to lack of central blood supply and may be somewhat different in color. There are numerous interlacing bands of fibrous tissue throughout the tumor and the periphery of the cut surface of the tumor shows inclusion of bundles of muscle fibers.

The microscopic appearance is that of a cellular fibroma occurring in striated muscle. According to Ewing,[2] the tumor structure may resemble a neurofibroma and may vary from a hard somewhat cellular fibroma to a rather cellular fibrosarcoma. However, Musgrove and MacDonald[8] do not agree with this view, and feel that these tumors are all benign and recur only locally due to inadequate excision. Thus, in their careful study of a group of desmoids and a group of fibrosarcomas, they felt that the two are completely separate entities. They do admit, however, that the border line cases are very difficult to distinguish. Histologically the picture is that of a cellular fibroma with interlacing whorls and bundles of fibrocytes. Fibroblasts in the periphery of the tumor appear to be more active and seem to be invading the surrounding striated muscle, and some of the surrounding muscle fibres frequently undergo degeneration. Occasionally giant cells may be seen.

The tumor does not produce distant metastases but is locally invasive and recurs frequently unless adequately excised. Pack and Ehrlick[9] reported one tumor as having recurred six times after surgical removal. On occasion the tumor may invade bone and peritoneum, but skin is involved only by ulceration due to pressure necrosis. The largest desmoid tumor ever recorded weighing 17 kilograms was reported in 1850 by Rokitanski.

Symptoms and Signs

Due to the usual location of these tumors they do not produce much in the way of symptoms. The patient frequently notices a small hard nontender mass in the lower abdomen and may come to the doctor because of it. Some of the larger tumors may cause mild sensation but in general these tumors do not cause pain. On palpation the tumor is felt to be smooth in contour but somewhat hard in consistency. Generally the skin can be moved freely over it. In order to differentiate between an intra-abdominal tumor and one within the abdominal wall one must use a special maneuver which calls for examining the abdominal muscles first when they are tightly contracted and then when they are relaxed. The patient is asked to sit up from a lying down position and this causes the abdominal muscles to contract which in turn causes a mass within the abdomen to become indistinct whereas a mass within the abdominal wall becomes more prominent. When the abdominal muscles are relaxed the tumors are more freely movable particularly in the plane perpendicular to the course of the muscle fibres to which they are attached. The main thing in the diagnosis of this quite rare condition is to remember that such a condition exists.

Differential Diagnosis

When one finds a tumor mass which conceivably could fit the above description, one must think of several different types of conditions to make the diagnosis. There are intra-abdominal tumors such as tumors of the caecum, uterine fibromyomas, and tumors of the ovary. If any of these become attached to the anterior abdominal wall they may simulate desmoid tumors. Any tumor arising from bone which might invade the abdominal wall could give the appearance of a desmoid tumor. Sometimes hernias may simulate desmoid tumors. Occasionally, tumors such as neurofibromas and lipomas may simulate desmoid tumors. Generally, one comes back to the old adage that "the only good place for a tumor is in a specimen bottle."

Treatment

Complete surgical removal with a wide margin of normal tissue on all sides of the growth is vital to the treatment of this condition. Therefore, it is essential to think of the condition and recognize it at surgery so that such an excision can be made. Although it is possible to enucleate the tumor in some cases, one is not actually removing the whole tumor, but is leaving portions of it, since this is not an encapsulated growth. Frequently these tumors require a wide excision of tissue leaving a rather extensive defect. Sometimes these defects may be closed without undue tension but it frequently is necessary to use some form of re-enforcement tissue such as facial transplants, etc.

There has been some evidence to show that x-ray and radium may have a role in the treatment of cases considered inoperable due to extensive growth or poor risk patients. However, this is true only in females since desmoid tumors in males are definitely radio-resistant. This latter fact makes one assume that castration or radiation therapy of the ovaries might have some significance in the treatment of desmoids in women. Pack[9] was able to cause a desmoid tumor of the shoulder to regress by irradiation castration of the ovaries as the only treatment. Apparently there has been no literature describing the use of sex hormones in the treatment of this condition.

In reviewing the existing records at St. Mary's and St. Luke's Hospitals at Duluth, Minnesota, only two cases of desmoid tumors were found.

Case Report

Case 1.—A twenty-seven-year-old white woman, first seen on March 27, 1950, came in because she had noted a lump in her right lower abdomen in the region of the upper portion of an old appendectomy scar. The appendectomy had been performed in 1946. Although she had noted some slight discomfort in her right lower abdomen off and on, for several months, she had not noticed the lump until a few days before she came in. She stated that the discomfort was more noticeable a few days before each period and that this had been noticed during the past three to four months prior to her visit to my office. She stated that the discomfort consisted of a slight burning sensation and was aggravated when she wore a girdle.

The patient's past history was negative for any significant illnesses or injuries and she had never been pregnant. The general physical examination was negative except for the mass which was located in the lower portion of an old right rectus scar. The skin was movable over the mass. The mass was somewhat tender, slightly irregular and hard. It was possible to feel the mass on deep pelvic bi-manual palpation but it was impossible to be certain that it was not within the abdomen. The pelvic examination was essentially negative except for some tenderness deep in the cul-de-sac. Routine laboratory examinations were essentially normal including an x-ray study of the colon.

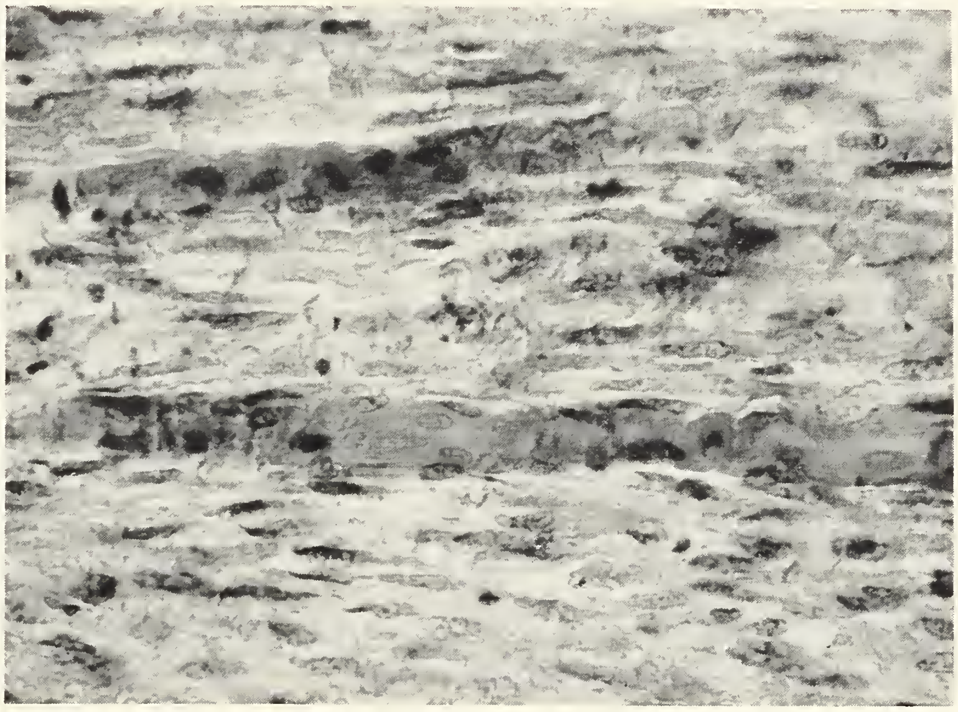

Fig. 1. High power photomicrograph showing the moderately cellular fibrocytic neoplasm invading striated muscle.

In the differential diagnosis of this case, the tumor was considered to be arising from: (1) The abdominal wall musculature, (2) The caecum, (3) Ovarian tumor, (4) Endometriosis inplanted in the abdominal wound following an abdominal operation.

On April 17, 1950, operation was performed under sodium pentothal anesthesia and a right rectus type incision was made directly over the lesion. On opening the fascia over the right rectus muscle, it was found that the mass was adherent to the anterior rectus sheath as well as originating in the muscle below. Resection of the tumor with an area of what was thought to be normal tissue and fascia around it was carried out and the defect was closed in routine fashion. The report by Dr. Arthur Wells, pathologist, revealed that the specimen consisted of firm tumor nodule measuring 4 cm. by 3.5 cm., which on cut section had firm consistency and a somewhat grayish refractile cut surface. Microscopic study revealed trabeculae of fibrocytes of moderate cellularity which invaded the attached muscle causing atrophy and regenerative changes in muscle cells (Fig. 1). He stated that the invasion extended to the line of excision and recommended a wider excision of tissue. Accordingly, on April 20, 1950, the patient was brought back to the operating room and a resection of most of the right rectus muscle and its fascia from the umbilicus to the pubis was performed leaving the peritoneum intact since it was not involved. A piece of tantalum mesh gauze about two and one-half inches wide by five inches long was placed in this defect and imbricated up under the remaining lateral and medial borders of the anterior rectus sheath with interrupted sutures, and the abdomen was closed in routine fashion. The pathologist reported that the excision included all the tumor tissue, and the patient has had no recurrence of the tumor to date. As evidence of the strength of her abdominal wall following this procedure, she successfully went through a pregnancy and delivered a normal infant approximately two years after her original operation with no evidence of further weakening of the abdominal wall.

Case 2.—A seventy-year-old woman is included in our review through the courtesy of Dr. Robert LaBree of the Duluth Clinic. The patient had a cholecystectomy approximately three years previously. She noted a small bulging in the lower portion of the incision shortly thereafter and this subsequently continued to enlarge. About a year prior to her hospitalization for the present condition she had noted a firm mass in the upper portion of the incision which had gradually enlarged. She noted discomfort in this region which at times had actually been painful. Examination of the abdomen showed a fixed mass 4 cm. in diameter at the upper end of a right upper quadrant scar. A hernia was present in the lower portion of the same scar. There were no other palpable masses or tenderness. There is no mention in the history as to whether the patient ever had children.

On January 9, 1951, the patient had a repair of ventral hernia with excision of the mass in the upper end of the hernia. In order to excise the mass so much of the fascia was removed that the surgeon decided to re-enforce the repair with a cutis split skin graft. She had an uneventful post-operative recovery. The pathologist reported that the tumor mass measured 8 by 6 by 5 cm. and on cut section was of diffuse fibrous consistency throughout and somewhat gray in color with no evidence of hemorrhage or caseation. The microscopic study showed that the tumor mass was moderately cellular and was made up of fibrocytes arranged in the regular interweaving trabeculae. There were very rare mytotic figures. The cells appeared mature. The multiple sections of the outer margin revealed no invasion beyond the compressed adjacent tissues. Diagnosis: "Fibroma of the abdominal wall consistent with desmoid tumor."

The pathologist added this note: "The histology of the lesion is perfectly satisfactory for a diagnosis of either a fibroma or desmoid. The fact that there is no infiltration of the adjacent connective tissues or muscle favors the benign lesion. However, the patient should be given the benefit of the doubt and any suspicion of re-occurrence should be immediately investigated and if proved to be a re-occurrence, a much wider excision into normal tissue should be carried out." In checking with the surgeon, the patient has had no recurrence up to the present time.

Summary

The literature on desmoid tumor has been briefly reviewed. A fairly typical case has been presented of a desmoid tumor arising in an appendectomy scar in a young woman several years after the surgery had been performed. A second case of desmoid tumor arising in an older woman has been reviewed.

The use of tantalum mesh gauze for re-enforcing the abdominal wall defects following the excision of large portions of the abdominal wall for removal of desmoid tumors has been presented.

References

1. Brockman, D. D.: Congenital desmoid of the abdominal wall. J. Pediat., 31:217-219 (Aug.) 1947.
2. Ewing, J.: Neoplastic Diseases. Fourth Ed. Philadelphia: W. B. Saunders Co., 1940.
3. Geschickter, C. F., and Lewis, D.: Tumors of connective tissue. Am. J. Cancer, 25:630-655 (Nov.) 1935.
4. Green, C. C.: Desmoid Tumor. Arch. Surg., 50: 304-306 (June) 1945.
5. Labbe, L., and Remy, C.: Traite des fibromes de la paroi abdominal. Paris: A. Delahaye and E. Lecrosnier, 1888.
6. MacFarlane, J.: Clinical Reports of Surgical Practice of Glasgow Royal Infirmary. Glasgow: D. Robertson, 63-66, 1832.
7. Mueller, J.: Ueber den feinern Bau die Formen der Krankhaften Geschwulste. Berlin: G. Reimer, 1838.
8. Musgrove, J. E., and MacDonald J. R.: Extra-abdominal desmoid tumors. Arch. Path., 45:513-540 (Apr.) 1948.
9. Pack, G. T., and Ehrlick, H. E.: Neoplasms of the anterior abdominal wall with special consideration of desmoid tumors; experience with 391 cases and a collective review of literature. Internal Abstr. Surg., 79:177-198 (Sept.) 1944.
10. Stone, H. B.: Desmoid tumors of the abdominal wall. Ann. Surg., 48:175-185, 1908.

SUBCUTANEOUS EMPHYSEMA FOLLOWING TONSILLECTOMY AND ADENOIDECTOMY

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WE WISH to report an unusual case of subcutaneous emphysema of the face, neck, and thorax following tonsillectomy and adenoidectomy. Although the patient recovered, the sudden appearance of widespread subcutaneous emphysema caused undue alarm among the physicians attending the patient, and we wish to bring the condition to the attention of other physicians who may encounter this unusual, but not rare, complication.

Case History

A six-year-old boy was admitted to St. Luke's Hospital on June 10, 1954, for tonsillectomy and adenoidectomy. Past history was negligible except for numerous sore throats and bouts of acute tonsillitis. The physical examination revealed enlarged tonsils. Laboratory studies of the blood and urine were normal. Preoperative medication consisted of codeine sulfate gr. ½ and atropine sulfate gr. 1/400 given subcutaneously forty-five minutes before the operation.

Induction of anesthesia was begun with open drop vinethene and was maintained with diethyl ether. An endotracheal tube (soft-rubber King No. 4) was inserted into the trachea without difficulty and the endotracheal tube was connected to a Heidbrink anesthesia machine which contained a functioning pop-off (expiration) valve. Nitrous oxide, 2 liters per minute, and oxygen, 2 liters per minute, were administered with ether during the course of the anesthesia. The pop-off valve functioned throughout the course of anesthesia. Tonsillectomy was performed using the Beck snare technique. During the adenoidectomy the bleeding was greater than usual and an adenoid pack was inserted. At the completion of the operation the patient was turned on his abdomen with the operating table in a 10 degree Trendelenburg position, and was allowed to emerge from the anesthesia. When he began to gag on the endotracheal tube, the tube was removed. It was noticed that the patient was still bleeding from the nose and therefore two cigarette packs were inserted into the nostrils, packing off the nasal cavity tightly. The patient was then taken to the Recovery Room where he vomited and coughed more violently than is usual. After about fifteen minutes the nurse noticed a swelling of his left eye. The left eyelids were swollen completely shut and the left cheek was markedly enlarged. Crepitation was felt in the swollen areas as well as on the forehead, neck, supra-clavicular area, and in the skin over the anterior chest wall (Fig. 1). The patient breathed normally and denied having pain from the swelling. X-ray examinations in the posteroanterior and lateral projections showed subcutaneous enphysema involving both sides of the neck with anterior mediastinal extension of the process. Following consultation with a chest surgeon, it was decided that since there was no respiratory embarrassment, observation of the patient would be the correct therapy. Four hundred thousand units of penicillin were given intramuscularly. At the end of five hours the swelling around the eyes had partly diminished and the patient was able to see. By the end of twenty-four hours the swelling of the left cheek had markedly decreased in size. The temperature remained normal. No change in respiration occurred nor did the patient complain of distress at any time. The patient received penicillin for five days. All swelling and crepitation had disappeared at the end of seven days (Fig. 2).

Historical Review

According to Edwards[3] the first report of subcutaneous emphysema of the head and neck following an interruption of the continuity of the mucous membrane of the mouth was that of Turnbull in 1870. Turnbull removed a bicuspid tooth from a bugler on a naval vessel. A short time later the bugler blew mess call, thereby causing an enormous swelling of the side of his face from which the tooth had been removed. In 1910 Parish[6] reported the occurrence of emphysema of the neck, cheeks and eyelids and of the skin over the ribs anteriorly in a twenty-eight-year-old male who had a tonsillectomy under an ether anesthetic. The patient left the operating room in good condition, but within a few minutes went into shock with marked facial emphysema, lividity of the face and cyanosis of the lips. The patient recovered uneventfully and in three days the emphysema had disappeared.

Later, von Hofe[9] reported three tonsillectomies

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in children complicated by emphysema of the head and neck. In each case, following the removal of the right tonsil, the right side of the face and neck swelled up with crepitation throughout the swollen area. The swelling and crepitation disappeared within five days in all cases. In each case the "ether hook" was suspended on the right side of the mouth and the air-ether mixture was blown by a pump against the right buccal membrane. Von Hofe believed that the most likely cause in these three cases was that air was forced into the subcutaneous tissue through the perforation of the tonsillar bed. He also stated that two other possibilities were perforation of an alveolus of the lung or entry of air through Wharton's duct.

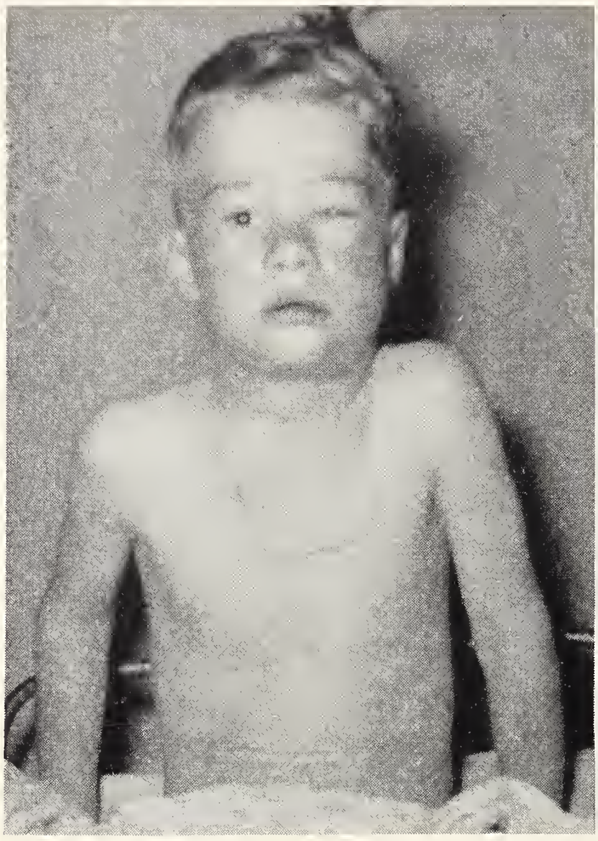


Fig. 1. Fifteen minutes after tonsillectomy and adenoidectomy left eyelids were swollen shut and marked swelling of the left cheek and neck was present. Crepitation was present in the swollen areas as well as in the area marked on the chest.

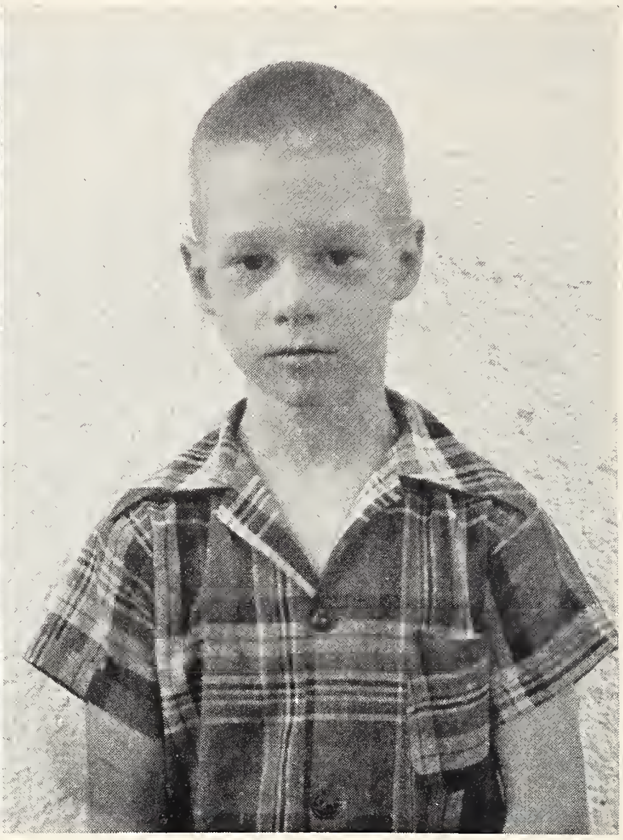


Fig. 2. The patient has a normal appearance seven days after appearance of subcutaneous emphysema.

Stevenson[7] also believed that air enters the subcutaneous space through the muscle fibers of the tonsillar beds. He reported a patient who developed constant bubbles in his saliva six hours after oxygen was blown through a tube onto the palate. Although subcutaneous emphysema was present from the forehead to the clavicles, it disappeared in five days without complications. Dickson[2] reported a case and stated that he also believed air passed directly through the tonsillar bed to the subcutaneous tissue.

Not all authors have accepted this explanation, however. Keen[4] reported subcutaneous emphysema over the left side of the chest, back and front, and over the lower part of the neck, which did not disappear for eleven days, in a five-year-old boy thirty hours after tonsillectomy and adenoidectomy. He believed the emphysema to be alveolar in origin. Thornton, Adams and Livingstone[8] reported a case of mediastinal and subcutaneous emphysema without pneumothorax following intratracheal insufflation anesthesia for craniotomy. No exhalation valve was present in the system. They believed that the intrabronchial pressure could have become elevated, causing a ruptured alveolus. Baker[1] recorded a case and stated that the complication was not particularly unusual, in spite of the few cases reported in the literature. He further stated that subcutaneous emphysema may occur during either general or local anesthesia and requires no special treatment. Further, the condition may be immediate or delayed, it occurs in both adults and in children, and with either the Sluder or dissection methods.

The etiology of the case Baker reported appeared to him to be a combination of a rupture of one or more alveoli in addition to seepage of air through the damaged muscle fibers of the tonsillar fossa.

Two of the most interesting case reports of subcutaneous emphysema following tonsillectomy are those of Mac Cready.[5] Tonsillectomy was done on a sixty-five-year-old male under local anesthesia with 1 per cent procaine. One hour after the patient had returned to his room a swelling suddenly appeared under the left eye following a severe attack of coughing. Crepitation was present over the left side of the neck and cheek. In forty-eight hours the swelling had disappeared. The second report was that of a forty-three-year-old woman who was given an ether anesthetic while a tonsillectomy was done by blunt dissection. At the end of the operation the patient was given a mixture of carbon dioxide and oxygen. The inhalation of these gases was prolonged and the patient clenched her teeth tightly. Suddenly there was a violent expiratory effort and a marked swelling appeared around the left eye. The eye was completely closed by the swelling of the eyelids and the left cheek was also swollen and crepitant. In forty-eight hours the swelling had disappeared.

Discussion

In our case, the most likely etiology is increased intrapharyngeal pressure due to a combination of coughing and vomiting and a tightly packed nose and nasopharynx, with consequent forcing of air through the tonsillar fossa into the subcutaneous tissues. The most persuasive argument for this explanation is that the emphysema did not occur until fifteen minutes after the patient left the operating room. Had a perforation been made in the larynx, or trachea during intubation, or had an alveolus ruptured because of high intrabronchial pressure, the emphysema would mostly likely have appeared during the operation as in von Hofe's cases. Furthermore, most of the emphysema was confined to one side of the face and more emphysema was present in the face than in the neck or chest, which suggests that the point of air entry was in the head rather than in the chest. It is conceivable, however, that a transient sticking of the expiratory valve could occur with a sudden rise in intrabronchial pressure and consequent alveolar rupture. Later, increased expiratory efforts could have forced air subcutaneously. Nevertheless, conditions for the production of subcutaneous emphysema were present here as in the other cases reported in the literature: tonsillectomy plus increased pressure within the pharynx. Although no objective data are available to substantiate it, reasoning suggests that the site of the etiology here is in the pharynx rather than in the trachea or lung.

As far as we have been able to determine from reading the literature no deaths have been reported due to subcutaneous emphysema following tonsillectomy. In our case the uneventful subsidence of the emphysema was again fortunate.

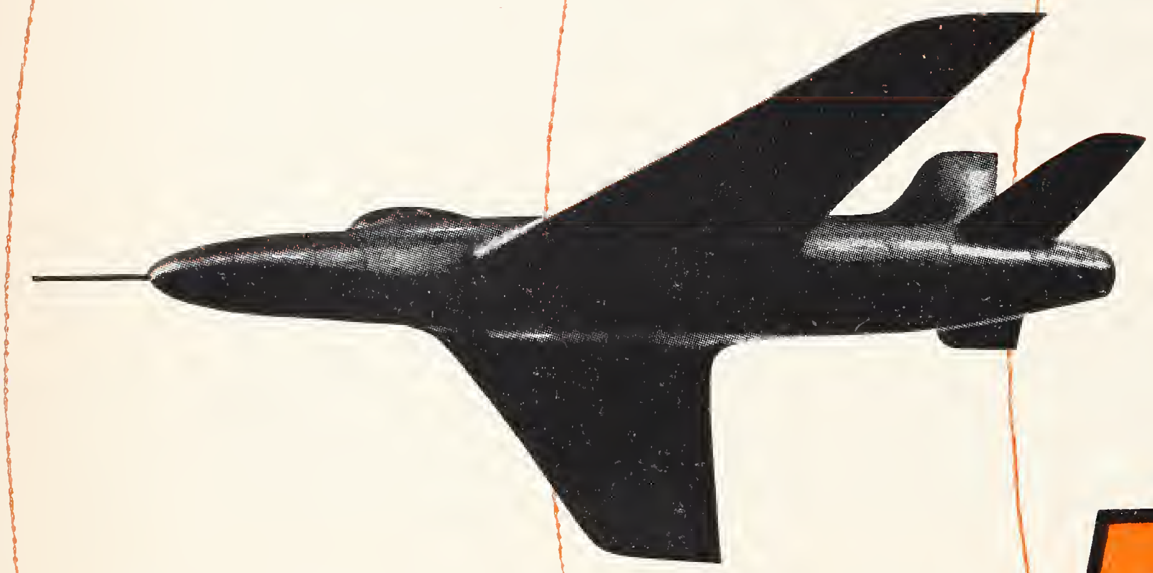
Summary

1. A report is given of a six-year-old boy who suddenly developed subcutaneous emphysema of the left eyelids and cheek, and of the anterior neck and chest following tonsillectomy and adenoidectomy. Conservative treatment resulted in the disappearance of the emphysema in seven days.

2. Ten other cases of subcutaneous emphysema following tonsillectomy are reviewed and summarized from the medical literature.

References

1. Baker, L. J.: Subcutaneous emphysema complicating tonsillectomy. Canad. M.A.J., 34:670-671 (June) 1936.
2. Dickson, J. C.: Subcutaneous emphysema following tonsillectomy. Ann. Otol., Rhin. and Laryng., 42: 1240-1241 (Dec.) 1933.
3. Edwards, R. W.: Surgical emphysema after tooth removal. J. Oral Surg., 6:147-149 (April) 1948.
4. Keen, J. A.: Medical and surgical complications of tonsillectomy in childhood. J. Laryngol. & Otol., 47:1-34 (Jan.) 1932.
5. Mac Cready, P. B.: Subcutaneous emphysema following tonsillectomy. Arch. Otol., 22:331-333 (Sept.) 1935.
6. Parish, B. D.: A case of subcutaneous surgical emphysema. An unusual complication following removal of faucial tonsils. Laryngoscope, 20:1046-1048 (Nov.) 1910.
7. Stevenson, R. S.: Subcutaneous emphysema following tonsillectomy. J. Laryngol. and Otol., 48:260-261 (April) 1933.
8. Thornton, T. F., Jr.; Adams, W. E., and Livingstone, A. M.: Mediastinal and subcutaneous emphysema following intratracheal insufflation anesthesia. Anesth. and Analg., 23:177-188 (Sept.-Oct.) 1944.
9. von Hofe, F. H.: Emphysema of the head and neck complicating tonsillectomy. J.A.M.A., 95:934-935 (Sept. 27) 1930.



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RADIOPHOSPHORUS IN HEMOPOIETIC DISORDERS

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USE OF radiophosphorus has passed beyond the experimental stage and now holds a significant place in the treatment of polycythemia vera and chronic leukemia. Its administration is becoming widespread as it is being made available to physicians who can satisfy the Atomic Energy Commission of their qualifications to handle it and give it to patients. Its use does not require expensive or elaborate equipment.

However, anyone who views the uncritical use of other agents, such as liver extract and vitamin B_{12}, in the treatment of hemopoietic diseases, cannot help but fear that radiophosphorus will be given when its use is not clearly indicated. Radiophosphorus yields ionizing radiation and can suppress the functions of bone marrow. Its use, therefore, must be founded on well-established diagnosis and definite indications.

Radiophosphorus is supplied as a solution of dibasic sodium phosphate that may be administered intravenously or orally. It yields beta radiation (electrons) only, in contrast to other radioisotopes which yield gamma rays in addition. The biologic effect of these electrons is similar to the secondary electrons excited by roentgen rays. When administered internally, it supplies total-body radiation.

The advantages in use of radiophosphorus lie in absence of radiation sickness, in ease of administration, in the short time required for treatment and in the simple, cheap equipment needed. The latter includes containers properly shielded to control the radiation, ordinary syringes and intravenous needles. Because radiophosphorus undergoes decay, the therapist must have a chart or slide rule to calculate the strength of the solution at the time of administration. The equipment must, of course, be used only for radiophosphorus. It is advisable to employ monitoring techniques to safeguard both those handling the material and the place where it is used from contamination. The therapist must know the fundamentals of radiobiology and safety precautions in radiation therapy and must have had experience in handling radioisotopes.

Indications for Treatment

Polycythemia Vera.—The outstanding indication for the use of radiophosphorus is polycythemia vera, but the diagnosis must be clearly established. It is not sufficient to regard elevated values for hemoglobin, erythrocytes or cell volume per cent (hematocrit) as criteria. These are relative measurements, depending in part on the volume of plasma. A normal absolute erythrocytic volume with a decreased absolute plasma volume will give an increase in hemoglobin and erythrocytes and in the hematocrit value, a condition known as "relative polycythemia," or "pachyhemia" (thick blood). This is not an indication for radiophosphorus therapy, as erythropoiesis is normal.

The basic criterion for a diagnosis of polycythemia vera is an increase in the absolute erythrocytic volume as measured by one of the techniques for determining the volumes of whole blood, plasma and erythrocytes. These procedures include the dye methods (Congo red or Evans blue) or techniques using such radioisotopes as radiochromium, radiophosphorus or albumin labeled with radioiodine.

We use the dye technique, which can be performed in almost any laboratory. By this method, the normal absolute erythrocytic volume does not exceed 50 cc. per kilogram of body weight for adults of normal weight. Values between 50 and 60 cc. per kilogram are regarded as equivocal unless accompanied by substantiating evidence, such

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as splenomegaly, leukocytosis, a leukemoid reaction or hyperplastic bone marrow. In the absence of one of these findings, the disease is best managed by phlebotomies and periodic observation until it is clear whether or not the erythrocytic volume is going to reach more definitive values. In our experience, erythrocytic volumes of 60 cc. or more per kilogram unequivocally indicate polycythemia.

It must be emphasized that the absolute erythrocytic volume is the essential issue, not the whole-blood volume, as the latter may be increased by an increase in absolute plasma volume. We have seen patients who have normal erythrocytic volumes but increased plasma volumes (up to 60 cc. per kilogram); this increase raises the whole-blood volume to more than 100 cc. per kilogram, which usually is conceded to be the upper limit of normal.

Important accessory information may be gained from study of smears of peripheral blood, in which regeneration of erythrocytes may be evident by increased polychromasia. Piling of erythrocytes is often seen but may be misleading if smears are not properly made.

Leukocytosis or myeloid immaturity or both may or may not be accompanying features. In only eighty-eight of a series of 241 cases of polycythemia vera studied by us was leukocytosis or myeloid immaturity noted. We have classified polycythemia vera on this basis: (1) the simple type, lacking leukocytosis or myeloid immaturity, (2) the type with an increase in mature leukocytes and (3) the leukemoid type, exhibiting myeloid immaturity. This classification is important as a gauge to prognosis and therapy. The leukemoid type tends to be more severe than the other types and presents a higher incidence of hematologic complications, such as the "anemia-leukemoid" state (severe anemia, pronounced splenomegaly and a leukemialike picture with regard to the leukocytes). The latter two types need larger doses of radiophosphorus on the average than does the simple type.

Studies of bone marrow may be of value, especially in the equivocal cases already mentioned. The marrow often presents a hyperplastic myeloid picture but it may not appear remarkable in some instances of the simple type. This should not deter treatment in the event of other unequivocal evidence of polycythemia vera. Studies of marrow are of especial value in advanced disease of the leukemoid type, in which an early change to myelofibrosis may be noted. In these studies it is imperative that units of marrow be obtained for embedding and sectioning so that the actual architecture may be seen.

Splenomegaly is an inconstant finding in the simple type but it is often present in the other two categories.

In some cases of uncomplicated asymptomatic simple polycythemia vera with absolute erythrocytic volumes of more than 60 cc. per kilogram, the process may be controlled by occasional phlebotomy, but care must be exercised to be certain that this method maintains hematocrit values of less than 55 per cent. Symptomatic improvement is not a reliable indication of control. If phlebotomy is needed oftener than every two months to produce objective control, we consider that use of radiophosphorus is indicated.

Patients who have polycythemia characterized by an increase in mature leukocytes or who have the leukemoid type should receive radiophosphorus after the hematocrit value has been decreased to near 55 per cent by phlebotomy.

Secondary Polycythemia.—An increase in absolute erythrocytic volume is present in secondary polycythemia but there is an evident cause, such as the anoxic anoxia of cyanotic cardiac disease or pulmonary impairment. If clinical differentiation is difficult, the presence of anoxia may be confirmed by studies of the oxygen saturation of arterial blood and measurements of pulmonary function. Secondary polycythemia is common in persons living at extremely high elevations.

Certain brain tumors, such as cerebellar hemangiomas, may produce absolute polycythemia. Other brain tumors may be associated with relative polycythemia due to low plasma volume. True polycythemia occasionally is seen with hypernephroma; it may disappear with removal of the tumor, recurring if metastasis appears.

Chronic Myelocytic Leukemia.—Treatment with radiophosphorus can achieve results comparable to those seen after roentgen therapy in early chronic myelocytic leukemia. It is also effective in more advanced disease, although pronounced splenomegaly may make focal roentgen therapy to the spleen preferable. When the disease is in its late stages and is refractory to roentgen therapy, radiophosphorus is seldom effective.

It is important to recall that not all instances of leukocytosis, leukemoid reaction and splenomegaly represent chronic leukemia. Severe sepsis, especially that due to pneumococci, may increase the number of leukocytes to more than 100,000 per cubic millimeter. Diffuse granulomatous disease, complicated lymphoma and malignant tumors with infection and necrosis may produce leukemoid pictures. When the diagnosis is not certain, it is better to defer use of radiophosphorus, which is not an agent for emergency treatment. Its effects are not immediate enough to be of benefit in critical situations. Usually the insidious onset, pronounced myeloid leukocytosis and splenomegaly leave little doubt as to the diagnosis of chronic myelocytic leukemia but, as a rule of thumb, it is sound practice to observe patients who have myeloid leukocytosis for several months until the diagnosis is certain.

It is by no means clear when to start treatment in early, mild chronic myelocytic leukemia without anemia or symptoms. It is not established that early treatment in such cases will prolong life. The specter of acute leukemia in persons who handle ionizing materials for radiation therapy cannot be entirely disregarded. It is well to have a definite reason for therapy, such as anemia, fever, undue exhaustion, advancing splenomegaly or lymphadenopathy. The same principle underlies use of roentgen therapy.

Studies of bone marrow in early chronic myelocytic leukemia reveal a hyperplastic myeloid marrow that is not pathognomonic. Such studies are of particular value if there is a question of acute leukemia and in long-standing conditions in which myelofibrosis may be present.

Patients sometimes exhibit a large, firm spleen, normal erythrocytic values or possibly anemia, a myeloid leukemoid reaction in the peripheral blood and a hyperplastic myeloid bone marrow. Often the leukocytes number less than 75,000 per cubic millimeter. Normoblasts and abnormally shaped erythrocytes are present in the peripheral blood. This may be the picture in advanced chronic myelocytic leukemia, a late sequela in polycythemia vera, or it may appear without a definite history other than that of a slowly progressive splenomegaly.

The nature of the condition just described is obscure, although it is sometimes called "atypical myelocytic leukemia." Many of these patients have a life duration longer than that observed in most patients who have chronic leukemia. Perhaps the condition should be called the "leukemialike syndrome" or the "anemia-leukemoid state."

This syndrome is pointed out simply to distinguish it from typical leukemia and to note that it is usually resistant to treatment with radiophosphorus. The usual symptom of which the patient complains is abdominal discomfort, pain or pressure from the large spleen; radiophosphorus will not reduce the size of the spleen, although focal roentgen therapy may be of temporary benefit.

In similar cases in which actual replacement of bone marrow with fibrous tissue is observed, total-body suppressive therapy or any type of radiation therapy is frowned on for fear of destroying regions of ectopic myelopoiesis in the liver, spleen and other sites. However, if there is an established hemolytic component, focal roentgen therapy or splenectomy occasionally is of benefit.

Chronic Lymphocytic Leukemia.—Radiophosphorus may be used in the treatment of chronic lymphocytic leukemia, although roentgen therapy or use of nitrogen mustard and related chemicals appears to be better. Radiophosphorus or total-body roentgen therapy, however, has a definite place in the management of widespread disease and when poor response is shown to focal roentgen therapy in the early stages of disease. Radiophosphorus is seldom helpful in late stages when the disease is resistant to roentgen therapy. Nitrogen mustard may prove of benefit in this situation.

Conditions in which Radiophosphorus is of Questionable or Doubtful Value

Multiple Myeloma.—It was hoped that radiophosphorus would be of benefit in multiple myeloma, but total-body radiation with radiophosphorus or roentgen rays has proved disappointing, although a rare patient has undergone symptomatic benefit. Use of urethan, cortisone or focal roentgen therapy, although of limited value, is preferable. Focal roentgen therapy directed, when possible, to painful lesions may afford palliative and symptomatic relief.

Lymphoblastoma.—Patients who have any of the various lymphoblastomas have not experienced effective response to radiophosphorus therapy.

Better results are obtained with roentgen therapy and nitrogen mustards and related chemicals.

Osseous Metastasis.—An occasional patient who has metastatic carcinoma in bones will experience relief of pain after treatment with radiophosphorus but results have been disappointing. Use of hormones and focal roentgen therapy is preferable for osseous metastasis from carcinoma of the breast or prostate. Melanomas are as resistant to use of radiophosphorus as they are to roentgen therapy.

Thrombyocytosis.—An increase in the number of platelets may occur in polycythemia vera (15 per cent of cases) or may be a prominent feature in early chronic myelocytic leukemia. The response of the platelet count to radiation therapy parallels improvement in the blood picture. In general, radiophosphorus therapy has not been satisfactory in controlling the high platelet counts seen in postsplenectomy states, in megakaryocytic hyperplasia and in thrombocytosis of undetermined origin.

Attempts to decrease normal platelet counts in chronic thrombosing disease have not been successful within safe limits; the depression of platelets is too brief to be worth while. Anticoagulant therapy is preferable.

Contraindications to Radiophosphorus Therapy

Acute Leukemia.—Usually no change in cellular type is obtained by use of radiophosphorus in this disease. As with other types of radiation in acute leukemia associated with pronounced leukocytosis, either no response to therapy is noted or a pronounced and rapid decrease in the number of leukocytes occurs. No clinical improvement ensues from use of radiophosphorus in acute leukemia.

Leukocytosis Not Due to Polycythemia Vera or Chronic Leukemia.—There is no point in correcting leukocytosis not due to the two diseases just named. The leukocytic increase is often part of a defense mechanism.

Hypersplenic Syndromes.—In hypersplenic syndromes, total-body radiation with radiophosphorus or roentgen rays is not sufficiently concentrated in the spleen to be of value. It may even depress the hyperfunctioning bone marrow, which is trying to maintain normal cellular values in the face of abnormal immunologic mechanisms. Control of hypersplenism needs more drastic focal management, such as splenectomy, focal roentgen therapy or use of steroids.

Relative Polycythemia.—This is not a disease of hemopoietic organs but a deficiency of plasma volume; it is not satisfactorily affected by radiation.

Hypoplasia of Bone Marrow.—Any condition associated with a hypoplastic state of the bone marrow contraindicates use of radiophosphorus.

Dosage Schedules for Radiophosphorus

Polycythemia Vera.—Preliminary phlebotomy is used to reduce the immediate risk of vascular complications, such as thrombosis. One or two phlebotomies, with removal of 500 cc. of blood on each occasion, are usually sufficient unless the erythrocytic volume is extremely great. As already indicated, an attempt is made to decrease the hematocrit value to 55 per cent before administration of radiophosphorus is begun.

After phlebotomy, the patient is given a single dose of radiophosphorus either intravenously or orally. When the oral route is employed, the dose must be approximately 30 per cent more than is given intravenously to compensate for failure to absorb the entire amount. Doses to be presented are those for intravenous administration.

In the past few years we have tended to give smaller amounts than were used initially, preferring to repeat the dose in eight weeks rather than give unnecessarily large doses at first. A study of the amounts of radiophosphorus needed to produce remission for at least one year showed that the range of dosage in treatment of the simple type of polycythemia was 3 to 21 millicuries (divided dosage). The same ranges were noted for treatment of the type associated with increased numbers of mature leukocytes and the leukemoid type; however, the mean effective dose for the simple type was 5 millicuries, whereas it was 7 to 8 millicuries for the other two types.

We usually administer 4 millicuries to patients who have the simple type of polycythemia; patients who have the other types receive 5 millicuries.

The patient is again studied in eight weeks with regard to hemoglobin, hematocrit, erythrocytes, leukocytes and platelets. If the values are normal, further treatment is deferred. Studies are repeated at intervals of eight to twelve weeks, and treatment is reinstituted when relapse occurs.

Secondary Polycythemia.—In many instances, treatment of the primary disease, with occasional phlebotomy, will improve the polycythemic state so that suppression of bone marrow is not needed. In extremely rare cases in which these measures are insufficient, radiophosphorus is administered as it is in polycythemia vera, although the effects are not so certain.

Chronic Leukemia. — Extended treatment is needed in chronic leukemia. In former years, we administered radiophosphorus twice weekly until the number of leukocytes decreased to 40,000 per cubic millimeter. At this point treatment was discontinued; the patient returned for more radiophosphorus when symptoms reappeared or if pronounced leukocytosis recurred.

During the past year we have used a modification of the Osgood technique.[1,2] The patient who has chronic myelocytic leukemia is given 40 microcuries of radiophosphorus per kilogram of body weight. This amount is given at intervals of two weeks until the leukocyte count decreases to less than 30,000 per cubic millimeter. Observation is then continued at biweekly intervals and treatment started again as soon as the number of leukocytes begins to increase.

It is stated that this method affords greater longevity, efficiency and comfort than do other methods.[1,2] We have not used this technique long enough to judge the relative effects on duration of disease, but we have been impressed with the fact that less radiation (9 to 15 millicuries) is used than in the older method (20 to 25 millicuries). As noted previously, later stages of the disease may require more therapy or be entirely resistant.

Complications of Treatment

Because radiophosphorus is administered as dibasic sodium phosphate, in the form of ions that are common in the body, and because radiophosphate is identical chemically with natural phosphate, no chemical intoxication occurs. The danger lies in the potentiality of radiophosphorus depressing the function of bone marrow too far. As the use of radiophosphorus has been carefully controlled to date, instances of serious hypoplasia of the marrow are extremely few, but this remains a hazard if radiophosphorus is not used cautiously.

Acute leukemia that appears terminally in some cases of polycythemia and of chronic leukemia has been laid at the door of radiation therapy but this relationship is by no means clear. Death from "hematologic complications" occurred in 13 per cent of a series of cases of polycythemia in which radiophosphorus was not given.[3] However, adequate data were not at hand to indicate if death was due to acute leukemia or to one of the "leukemialike" states. As already noted, radiation may be a cause of acute leukemia, as evidenced by the abnormal incidence of leukemia in workers handling ionizing radiation. Even if it is assumed that acute leukemia is a risk of such treatment, this risk is much smaller than the possibility of vascular occlusion and hemorrhage so commonly seen in days before radiophosphorus therapy.

About 10 per cent of deaths in patients who have polycythemia vera and who receive radiophosphorus occur as the result of "leukemialike" states, the "anemia-leukemoid state," "atypical chronic myelocytic leukemia" or myelofibrosis with myeloid metaplasia. However, the anemia-leukemoid state occurs in patients who have had no radiation or extremely small amounts. It is suspected that the "anemia-leukemoid state" may be a late sequela of myeloproliferative diseases, if life is not terminated earlier by some other misadventure.

In polycythemia vera, the average age at death in our series of patients receiving radiophosphorus was 64 years, while the average age at death was 54 years in patients treated by other means. This, along with general clinical impressions, indicates that carefully administered radiophosphorus does not pose serious complications of treatment.

Summary and Conclusions

Use of radiophosphorus is established therapy for polycythemia vera and for chronic leukemia of the myelocytic or lymphocytic type.

Use of this radioactive agent is contraindicated in acute leukemia, leukocytosis not due to polycythemia vera or chronic leukemia, hypersplenism, relative polycythemia and all types of hypoplasia of bone marrow.

Administration of smaller doses than those used formerly is suggested.

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Laboratory Aids to Medical Practice

Sponsored by
The Minnesota Society of Clinical Pathologists

CAREER: MEDICAL TECHNOLOGY

GEORGE G. STILWELL, M.D.
Rochester, Minnesota

Nature is supposed to detest a vacuum; when such a situation exists, nature rushes in, either mechanistically or teleologically, in an attempt to fill the vacancy. A void, albeit partial in extent, exists today in the field of medical technology. Whether it be the fault of nature or some other agent, there appears to be no concerted rush to fill this partial vacuum. The material needed for stuffing this particular void is young people of either sex, although at present 92 per cent of registered medical technologists are women.

Why is the Shortage of Medical Technologists Important?

The internist, the surgeon and the general practitioner, as well as the pathologist, should be concerned about the inadequate supply of trained medical technologists. All physicians feel the impact of this paucity because the shortage of qualified technologists is reflected in lessened efficiency in the application of laboratory medicine to diagnosis, prognosis and treatment. The importance of the relationship between clinicians and pathologists goes without saying, yet the potential benefits of this relationship cannot be realized optimally if the pathologist is handicapped by lack of assistants in the laboratory. The medical technologist is as important in the total practice of medicine as is the nurse, and shortages of personnel in either of these professions cannot help but affect the practice of medicine unfavorably.

What Is Medical Technology?

Medical technology is one of the more recent adjuncts to the scientific and modern practice of medicine. The work of the medical technologist is an integral part of the practice of clinical pathology, and the pathologist would be lost without the indispensable aid of these handmaidens in the laboratory diagnosis of disease.

The medical technologist starts with an interest in science and the possession of a considerable degree of manual dexterity. Accuracy, calmness under pressure, efficiency and cheerfulness are primary requisites. The medical technologist who has attained certification by the Registry of Medical Technologists of the American Society of Clinical Pathologists has had a minimum of two years of collegiate work plus twelve months' training in an approved school of medical technology. Many spend three years in college and then obtain a Bachelor of Science degree on completion of their technical training in the approved school. The certified medical technologist must then pass a nationally conducted examination.

The great majority of medical technologists work in hospital laboratories performing the multitude of important laboratory tests with which all physicians are familiar. Others work in clinics, physicians' offices, private laboratories or research institutions. The work of the medical technologist is satisfying, the demand for such persons is great and the compensation is good.

What is an Approved School of Medical Technology?

Many physicians who are consulted by young people seeking advice about entering the field of medical technology enthusiastically recommend the profession but may be a bit hazy about exact directions for correctly steering such persons to the proper place. Any qualified pathologist can give advice and information concerning acceptable schools for medical technology.

An approved school of medical technology is

From the Section of Publications, Mayo Clinic, Rochester, Minnesota.

This is the twenty-second in a series of editorial reports sponsored by the Minnesota Society of Clinical Pathologists and designed to foster closer relationships between clinicians and pathologists.

one that has been inspected and accepted by the Council on Medical Education and Hospitals of the American Medical Association. All training of technologists in such schools must be done under competent medical control. Each school must have an adequate and qualified teaching staff. The director of the school must be a graduate in medicine who is certified in clinical pathology by the American Board of Pathology or who has had training and experience in clinical pathology acceptable to the Council. Approximately 500 such schools exist in the United States; a complete list can be obtained from the Council on Medical Education and Hospitals of the American Medical Association, 535 North Dearborn Street, Chicago 10, Illinois.

What is an Unapproved School?

A number of commercially sponsored schools allegedly for the purpose of training "technologists" are found throughout the United States, including Minnesota. These schools offer to graduates of high schools a short course, usually ranging from 9 to 12 months, at a relatively high tuition. Such schools are not on the list approved by the American Medical Association because the instruction in these unapproved schools is given by nonprofessional persons, the curriculums are inadequate and substandard, and these schools have no hospital affiliations. The students accepted by these schools are, of course, unqualified according to the standards set up for approved schools.

Many students who "graduate" from these commercial schools that have no hospital connections are amazed and disappointed to find on applying for work that their training and knowledge are completely inadequate to qualify them for work in many laboratories. The undesirability of recommending these unapproved schools to young people interested in a career in medical technology is obvious. Again, the pathologist can advise a physician who is in doubt about which schools are approved and which are substandard.

What is Being Done About the Shortage of Medical Technologists?

Because the afore-mentioned vacuum has not been adequately taken care of by natural forces, some assistance is being offered by several groups interested in the field of medical technology. A National Committee for Careers in Medical Technology has been formed by three sponsoring organizations, namely the American Society of Medical Technologists, the American Society of Clinical Pathologists and the College of American Pathologists. These groups have co-operated in the establishment of an active program of recruitment to inform young people of the satisfactions and opportunities offered by a career in medical technology.

The most tangible evidence of activity in this program is in the form of a film entitled "Career: Medical Technology." This is a 16-mm. movie in color and sound, with a running time of 24 minutes. It is a professional job turned out at a cost of many thousands of dollars and a film whose quality is such that pathologists and medical technologists can be proud to present it anywhere.

The primary purpose of this film is to tell the story of medical technology to high-school and college students and to high-school counselors and other school personnel to whom students turn for advice concerning their future careers. It is suitable for showing during the Career Day held by many high schools in Minnesota. It can be used as a television educational feature. It can be shown before science classes and clubs; it can be presented before high-school faculties so that the teachers can know more about the profession of medical technology.

This film will attain its greatest usefulness when its showing is accompanied by the presence of both a pathologist and a medical technologist; they can set the scene and, as a postlude, answer questions raised by the film.

The national première of this film was held in Washington, in September, when it was enthusiastically received at the annual meeting of the American Society of Clinical Pathologists. The first Minnesota showing was held in November. Prints of this film have been purchased by both the Minnesota Society of Clinical Pathologists and the Minnesota Society of Medical Technologists; these prints are available without charge for showing to interested groups. Inquiries regarding use of these films may be directed to Dr. Kano Ikeda, Charles T. Miller Hospital, St. Paul, who is chairman of the Committee on Medical Technology of the Minnesota Society of Clinical Pathologists, or to Virginia Burris, M.T. (ASCP), 57 Inner Drive, St. Paul, who is president of the Minnesota Society of Medical Technologists.

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President's Letter

THE GREATEST GIFT

"It doesn't seem like Christmas." How many times have you heard someone say that around this time of the year?

But why shouldn't it seem like Christmas? Is it because we have commercialized this sacred holiday to such an extent that we leave out the real reason for our celebration? Or is it because we are so wrapped up in our own mundane affairs that the holiday is upon us before we have had time to prepare for it mentally and spiritually?

The birthday of Christ should find us, as physicians, keenly attuned to the meaning of healing. We are engaged in the repair of bodies, and the prevention of damage to them. Christians are likewise keenly attuned to the meaning of healing—but to the healing of souls instead. Therein lies a sound kinship of the two. For a deep abiding faith in God is a tremendous asset to the healing of the body, and Medicine knows the value of such faith.

A strong thread of such faith should run through the hearts of men of medicine in their everyday practices. Because we are mere men, we are eternally dependent upon a higher power, and without it, we should be lost.

At Christmas we develop a greater regard for our fellow men; we nourish in ourselves that latent love for all people which has lain dormant in us throughout the year.

But if it doesn't seem like Christmas, whose fault is it? It is ours. We are to blame if we feel that the holiday is merely something for which we have to prepare. We should not have to cultivate the Christmas spirit—the spirit of giving, patterned after the greatest Gift of all, should be an integral part of our daily living and working. It should be automatic—a feeling that gives us in return great contentment, sincere good will, and infinite peace.

In this, my last letter to you during my term as president, I would like to tell you that I have found countless instances of this fine spirit throughout the year. The men with whom I have had the privilege to work during the past twelve months, have proved to me that this spirit is not always dormant. I have been gratified to have been associated with men of this high caliber. My deep thanks and appreciation to all those who have made my term a satisfying and rewarding one.

I would like to leave this parting thought with you: Because we are members of a great profession, let us at all times sincerely try to be worthy of the respect and reverence accorded us by our patients, friends and colleagues. We can do no better than to imitate the life, the faith, and the indomitable spirit of the man whose birthday we are celebrating now. Let us constantly strive to "make it seem like Christmas."

Best wishes for a blessed holiday season.

J. Ohage.

President, Minnesota State Medical Association

Editorial

Arthur H. Wells, M.D., *Editor;* Henry G. Moehring, M.D., and John F. Briggs, M.D.

OUR MOST POTENT WEAPON AGAINST TUBERCULOSIS

THERE is nothing so important in controlling a disease as a specific test to determine its presence while it is microscopical in size as well as during later stages of its development. Everywhere physicians lament the fact that a test is not available to detect the presence of cancer before the lesions are large enough to be located by other methods of examination. If such a test were available, the organs of the reactors which cancer frequents could be examined often, and in all probability many in the silent stage would be located before they have metastasized.

In tuberculosis, such a test is available in the tuberculin reaction. It is well known that within an hour after invasion with tubercle bacilli occurs, lesions have begun to form. Within seven or eight weeks, the tissues of the host, the skin included, are so sensitized to tuberculo-protein as to react characteristically to tuberculin. Therefore, at this time a tuberculin reaction indicates the presence of lesions containing tubercle bacilli. The sensitivity of tissues to tuberculo-protein apparently is maintained as long as living tubercle bacilli reside in the body.

When the tissues first become sufficiently allergic to react to the tuberculin test, lesions are so small that they can rarely be located by any type of examination, including roentgenogram inspection. However, such lesions may subsequently develop to clinical proportions. In fact, all clinical lesions begin in this way.

The tuberculin test provides so much more information about tuberculosis than any other part of the examination that it should be routine with every physician, regardless of where he works or what phase of medicine he does.

Some of the values of the tuberculin test are:

1. It detects the presence of tuberculosis in the human body much earlier than any other phase of examination.

2. Since tuberculosis is a contagious disease, the physician knows that the tuberculin reactor has been in contact with someone who is eliminating tubercle bacilli. Therefore, examination of the reactor's associates often brings to light the person who is responsible for transmitting the infection. Thus, the tuberculin test is our best epidemio-logical measure.

3. The only accurate method of determining with reasonable promptness the effectiveness of a tuberculosis control program in a community or a larger area consists in determining whether the incidence of tuberculous infection among children is decreasing from time to time; that is, whether fewer babies and young children are infected now than were those in the same age period a few years ago. This can be determined only by testing with tuberculin.

4. There is now some reason to believe that antimicrobial drugs, plus the body's defense mechanism, may destroy all tubercle bacilli if administered soon after the invasion occurs while lesions are still small and vascular. The only way to determine when this condition is present is to test nonreactors periodically to find those promptly after they convert to reactors. It now seems more than likely that this will be the ideal treatment of the future. If so, periodic tuberculin testing will be indispensable. Therefore, the tuberculin reaction will be the only method by which the physician will know when to start treatment. In fact, such treatment of recent converters is already being employed by a number of physicians.

5. It is believed that when all tubercle bacilli are destroyed in primary lesions, sensitivity of the tissues will disappear, and, therefore, the individual will cease to react to tuberculin which will be the indication to stop treatment.

6. Whenever tuberculosis is regarded as a compensable disease, much injustice may result unless periodic tuberculin testing is done among all concerned. For example, a lesion demonstrated by the x-ray shadow it casts, wherever a person is employed at the time, may be the result of an infection acquired months or years before in a different place. Thus, the liability dates to the times when the infection was allowed to occur.

Such accurate information is obtainable only by periodic tuberculin testing.

In the State of Minnesota there is now evidence that only slightly more than 20 per cent of the citizenry harbors tubercle bacilli and therefore reacts to tuberculin. Actual testing indicates that most of the persons now harboring tubercle bacilli are in the older age bracket. Among seniors in high school rarely are more than 4 to 8 per cent infected, and among grade school children 1 to 2 per cent in rural areas and 3 to 4 per cent in cities. There are many grade schools in which no child has been infected.

As the incidence of tuberculous infection decreases, the value of the tuberculin test increases.

A diagnosis of clinical tuberculosis is never justified in the absence of a tuberculin reaction or the recovery of tubercle bacilli. Practically everyone who is eliminating tubercle bacilli reacts to tuberculin; hence, the importance of this test in diagnosis.

In Minnesota, the tuberculin test is by far the most potent weapon the members of the State Medical Association have in their statewide tuberculosis eradication program.

J. Arthur Myers, M.D.

HELL HATH NO FURY

We have read that hell hath no fury like a dues-paying professional society member grown scornful of his society's publication. Well, why shouldn't he be furious? Perhaps his reasons for becoming scornful are invalid to anyone except himself. Nevertheless, his scorn and fury should be heard. The society's publication is for the society members, not just because they pay for it, but because they are the sole excuse for the existence of the publication. If the publication strays from serving *one* member, no great furor arises, but if one member, conscientious enough to read his society's publication, is aroused to fury, the likelihood is that other members are similarly aroused and that the cause of the discontent merits attention.

Andrew Jackson is credited with the observation that one determined man constitutes a majority. Times and the sense of what is fitting have changed since Old Hickory's day, but we still listen with profit to the voice of the minority in any group, whether it be a hospital staff or the nation. Witness our unwillingness to abridge the Bill of Rights because we think that any probable benefit to society would be outweighed by the chance of injustice to a citizen. The minority is at once a goad and a brake to those who determine the course of society and to society itself . . . a necessary and wholesome goad or brake. It is on the activity of pressure groups that democracy depends for its continuance as a democracy, for how is the voice of the people to enter government if unheard? The summation of the intensities and directions of these forces from pressure groups, of whatever size, establishes the course of a democratic society. Thus, even the negative forces, the brakes, make their influence felt.

So much for generalities. Now we would point out that the editorial board of Minnesota Medicine is not soliciting furious criticism from any State Medical Association member grown scornful, but that the board is open to suggestions and comments from dues-paying members and, for that matter, from anyone interested enough to bestir himself to make comments and criticisms about Minnesota Medicine. The editorial board is not able to guarantee an unimpassioned and totally objective appraisal and reaction to all criticism, but it will respond to such criticism just as certainly as any executive body of a democratic society responds to forces from within that society. As further assurance that any constructive criticism you have to offer will meet an interested audience, the editorial board declares itself unanimously in agreement with Aristotle's dictum that it is the discriminating eater and not the cook who is the best judge of the dinner. So let us have your contributions, be they critical, scientific, or otherwise.

H. G. M.

WHY THIS LIVELY INTEREST IN COMMON STOCKS?

The period of speculative excesses of the late 1920s was followed by a long period of liquidation. During the thirties, we were in a depression during which much reliance was placed upon security guaranteed by a benevolent government. We witnessed a period of rising government debt, rising prices, and the beginning of inflation. Then followed an expensive war with a huge increase in government debt. Savings were almost exclusively employed in the purchase of government bonds, insurance, annuities, and similar forms of guaranteed dollar contracts. In the years following the war, the holders of matured government bonds found that inflation had done something to their dollar in terms of purchasing power. Owners of pure dollar contracts had suffered a loss in purchasing power of some 40 per cent. Confidence in government paternalism faded as people realized that everything the government had to give must somewhere be taken from the people. The realization grew that the only source of wealth stemmed from the production of goods and services which is the function of business. Since ownership of business or the ownership of common stocks are synonomous, all financial security, in the long run, must stem from common stocks.

A large mutual fund released some observations which seemed startling to the public. This fund revealed recently that all who had invested $500 per month, plus quarterly dividends, for any twelve-year period of the fund's existence, were in a position to take income of $500 per month without disturbing the principal amount. Upon further examination of the results of other leading mutual funds, it was found that this remarkable performance was common. It follows very closely the pattern of the one-hundred-year history of common stocks as reported by the Cowles Commission, which shows an average growth of 3 per cent and income of 6 per cent annually.

A person may inquire, "What is responsible for such a superior record of growth and income, and what are the prospects for its continuation?" First, the outstanding characteristic of this country is growth. Since all material progress in this country is dependent on savers exchanging accumulated capital for equities, these equities or common stocks are the means through which wealth is created. It follows, therefore, that our government bonds are good because common stocks are good.

Despite periods of interruption, the long-term growth in value of common stocks parallels the growth of our economy. Seven thousand new mouths must be fed each day. Americans constantly demand higher living standards. Industry strives to provide these needs through research. Expenditures for research and new product development will be stepped up even higher as business competes for a share of the expanding consumer market. New industries such as electronics and nucleonics will contribute substantially to economic progress. An insight into what the future holds may be had from the remarks of Philip Reed, chairman of General Electric Company, who cited reasons why General Electric anticipates producing in the next ten years as much electrical equipment as they have produced in the previous seventy-five years.

The greatest factor in the market and the hardest to evaluate is the institutional buying of common stocks for income and participation in our expanding economy. Insurance companies, pension funds, trust companies and even savings banks in New York are turning to common stocks for better yields. It is the combined strength of these long-term investment-minded institutions, coupled with a new look in government fiscal policies, which has helped to reawaken investors' interest in common stock investing.

Nicholas V. Schaps
J. M. Dain & Company
Investment Securities

POISONS, A KILLER IN CHILDHOOD

YOUR attention is directed to the article in this issue on "Poisons, a Killer of Children in Minnesota." Putting a padlock on the medicine cabinet is not the complete answer. We must remind our families with small children that aspirin may kill. Home safety check lists include dangers under the kitchen sink, around supplies of insecticides and rodenticides, associated with fuel tanks, cleaning solutions, et cetera.

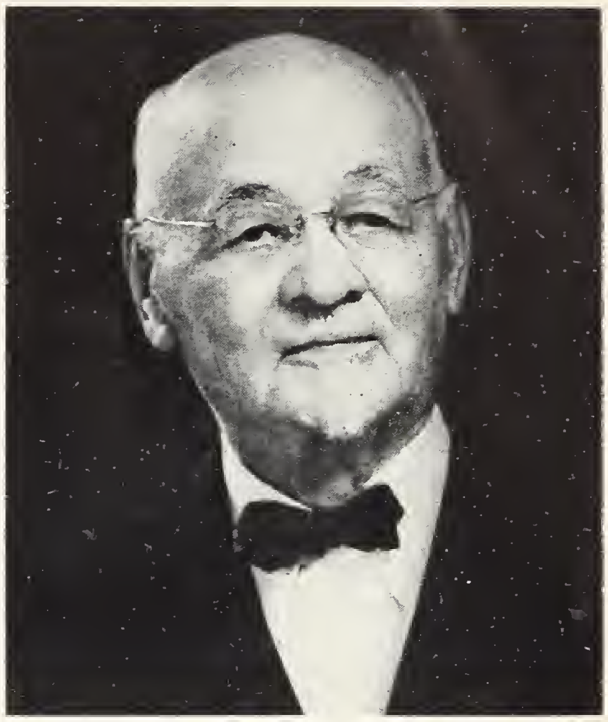
Morbidity from poisons, thank goodness, far exceeds mortality. Physicians have the moral responsibility of preventing these hazards as conscientiously as giving innoculations for diphtheria and whooping cough.

Your serious consideration is directed to the contributor's comments on a poison control center for Minnesota comparable to what has been set up in Philadelphia and Chicago.

Tague Chisholm, M.D.

E. E. Novak

Minnesota's "Physician of the Year"



Dr. E. E. Novak, New Prague, has been given Minnesota's "Physician of the Year" award by the Minnesota State Medical Association. The eighty-one-year-old doctor has practiced in New Prague for fifty-nine years, and is still active in his daily practice.

Dr. Novak came to New Prague in 1895 when the town was just beginning to look like a town. It was still frontier land, rugged and growing, and most of it had to be hewn out of dense timber to make the small farms of the area.

During the fifty-nine years of his practice in New Prague, Dr. Novak has grown with the town. From a struggling physician, he has come to be one of the most respected men of the community, revered and loved by all for his strong character, sincere good will and devotion to his people.

Besides practicing general medicine twenty-four hours a day all these years, Dr. Novak has found time to be of help to his community in many other fields: agriculture, education, cattle and horse breeding, civic affairs, financing, business and sanitation. In agriculture, he was a leader in promoting use of scientific methods, such as crop rotation, and better use of crops and farm animals. When he first arrived, the farmers were raising small crops, mostly wheat, and were milking a few scrubby cattle that weren't really dairy cattle. Dr. Novak, having come from a farm in Iowa, began introducing progressive methods to his neighbors. Many a farmer laughed at his "new-fangled" notions when he planted alfalfa, began raising tuberculosis- and brucellosis-free herds, shipped to market the first carload of hogs in the county and introduced diversified farming procedures. He helped organize the co-operative creamery of New Prague after realizing the need of the farmers in the area.

In the field of education, Dr. Novak was also very active. He served on the local school board for forty years, was active in the PTA, was president of the Minnesota State School Board association and received its distinguished service award. At present he is a Regent of the University of Minnesota, and as such has served admirably, with an avid interest in the medical school, the agriculture department, and all other fields as well.

His influence in the business and financial affairs of the town has been valuable over the years. He was one of the founders of the First National Bank of New Prague in 1903, and helped the State Bank of New Prague over the depression in the difficult years. His influence was not primarily financial, but was based on the confidence, trust and integrity of his character and the high regard the people have for him. Dr. Novak was elected as mayor of the town for two terms and presented sanitation projects and progressive ideas which did much to improve the growing community.

Dr. Novak has always been interested in breeding pure bred cattle and horses. Several of his cattle have won coveted national honors. The doctor has been an active and honored member of the Red Poll Cattle Club of America, serving as its president for many years, and receiving its distinguished service award. He helped found the Southern Minnesota Livestock Show and has been a moving force in its growth.

There are countless stories that can be told about the doctor's struggle to practice good medicine throughout the severe early days around the area of New Prague. Poor communication, inadequate roads, reluctance of people to submit to new successful treatments, poor medical facilities. all combined to make his practice a hazardous one, especially when the winter months brought deep snow and afforded difficult travel, at the best, to many of his scattered patients. He used a bicycle, horses, wagons, buggies, and had the first car in town to get to his patients; often he went by foot to administer to the sick.

Although his honors are many, his greatness is reflected best in the firm respect that the townspeople have for him. After he had practiced fine medicine for fifty years in the area, the town and surrounding area gave him a gala tribute in a testimonial banquet and program—and the crowd was overwhelming. Tribute was paid to all his fine deeds by friends and neighbors, many of whom he had treated as children and adults, and many of whom he had brought into the world.

Many exalted words have been said of the good doctor—all of them deserving and accurate. But the greatest tribute paid to him is the love and affection of his fellow citizens. What the people feel about their doctor has been said in many ways—in an old man's crying because the doctor had saved his wife's life after the man had complained about the $7 charge; in a young child's relief from the pain of a broken leg or arm; in the grateful tears of an entire family when he answered a desperate call in the middle of the night; in the great fun and competition he has enjoyed in racing his own purebred horses; in the conflict and sense of something well done, experienced when he won one of his many battles against obstacles such as stubbornness and obstinacy standing in the way of medical progress; in the secure knowledge that a life has been, and still is, being devoted fully to the noble service of his neighbors, his community, his state and his nation.

RADIOPHOSPHORUS IN HEMOPOIETIC DISORDERS

(Continued from Page 886)

Radiophosphorus does not pose a chemical hazard but does present a radiation hazard if care is not taken in selection of cases and administration of the material.

The etiologic relationship between radiophosphorus therapy and acute leukemia is not established but remains a possibility. However, if this relationship is assumed, it is a small risk in view of the reduced incidence of vascular catastrophes in patients treated with radiophosphorus.

The relationship between "anemia-leukemoid states" and use of radiophosphorus is not established, but occurrence of these states without antecedent radiation therapy suggests they are sequelae or late phases of some myeloproliferative disorder.

References

1. Osgood, E. E.: Titrated, regularly spaced radioactive phosphorus or spray roentgen therapy of leukemias. A.M.A. Arch. Int. Med., 87:329-348 (Mar.) 1951.
2. Osgood, E. E., and Beaman, A. J.: Treatment of chronic leukemias: results of therapy by titrated regularly spaced total body radioactive phosphorus, or roentgen irradiation. J.A.M.A., 150:1372-1379 (Dec. 6) 1952.
3. Stroebel, C. F., Hall, B. E., and Pease, Gertrude L.; Evaluation of radiophosphorus therapy in primary polycythemia. J.A.M.A., 146:1301-1307 (Aug. 4) 1951.

Medical Economics

Edited by the Committee on Medical Economics
of the
Minnesota State Medical Association
George Earl, M.D., Chairman

NINE MILLION NOW COVERED BY INDEPENDENT HEALTH PLANS

A new government report shows that 9 million persons carried some protection under independent health plans at the end of 1953. This figure includes 6.9 million for hospitalization, 6.6 million for surgical benefits, 5.4 million for medical benefits in office, home or hospital. Also, 3.4 million were eligible for diagnostic services and 600,000 for dental care.

These figures are quoted in a recent issue of the *"Washington Report on the Medical Sciences."*

Unions First in Growth

One of the most striking findings was that in the past five years union plans have gone from eighteen in number, with less than 500,000 enrollment, to seventy-six having a membership of 2.8 million.

The report is based on a survey which covered 299 independent plans—consumer-controlled co-operatives, privately operated prepayment clinics, plans conducted by medical societies and fraternal groups, and other types unassociated with insurance companies, Blue Cross or Blue Shield.

The study was made by the Division of Research and Statistics, Social Security Administration, and the figures presented are from a preliminary report.

Plans Varied

Another important point is the great diversification of the plans studied. Some use community doctors, the report states, others follow the panel system and many give direct services in group service clinics. Although none are mentioned by name in the report, it may be disclosed that the study included such nationally known independent groups as Health Insurance Plan of Greater New York (HIP), the Kaiser system on the West coast, Ross-Loos Clinic in Los Angeles and the health plan sponsored by United Mine Workers. A total of 132 plans, covering 2.9 million persons, provide benefits through some form of group medical practice, the report says.

The Washington report notes that the 9 million beneficiaries are divided almost equally between employes and dependents. It continues:

> "That the percentage of dependents is not higher is ascribed to the fact that employes alone are eligible for prepaid benefits in 40 per cent of the plans. Membership ranges from less than 100 in one co-operative to 1.4 million (workers and dependents) in the largest plan covered in the survey. Surprisingly, Connecticut ranks No. 1 among the states for hospitalization coverage in independent plans (1,110,000), the explanation being that it includes enrollment in a former Blue Cross plan."

Enrollment by States

The report further breaks down the enrollment in these plans by states: New York—1,496,000; Connecticut—1,114,000; Ohio—978,000; California—861,000; Pennsylvania—691,000; Washington—546,000; West Virginia—439,000; and Michigan—425,000.

Developments Cited

Besides rapid expansion of union-sponsored health plans, the survey cites certain developments in the following fields:

1. Increasing coverage of dependents and retired workers.
2. Continued provision of benefits, on a supplemental basis, under employe mutual benefit plans after they have been superseded by arrangements reached through collective bargaining or by employer-sponsored insurance contracts.
3. Greater emphasis upon preventive medicine, evidenced by increased provision for outpatient services.
4. Joint action by a number of smaller unions toward establishment of medical centers or insurance programs, or both.

UNION WELFARE FUNDS GET "ONCE-OVER"

Both the American Federation of Labor and the Congress of Industrial Organizations have pledged broad investigations of suspected misuse of union welfare funds, much in the news of late.

And, the AFL has pledged "strong and wholehearted support" to Government curbs on welfare fund abuses.

According to a recent article in the New York Times, reported in Public Health Economics of November, "A resolution adopted at the convention's final session discarded the Federation's seventy-three-year-old tradition that the Government should keep its hands off internal union problems." Convention delegates representing over 10 million members voted unanimously to support Government curbs on mishandling of union welfare funds.

The newspaper story states: "The convention resolution urged that investigations into welfare rackets eschew politics and give attention to the delinquencies of employers, insurance companies, brokers, agents, state regulatory bodies and physicians receiving fees from welfare plans, as well as the sins of union representatives."

It continues:

> "In a number of instances, the AFL said, abuses that have been found to exist stem directly from practices that have long been common in the commercial insurance industry and tolerated by state insurance commissions. These cannot be corrected by investigations or actions directed against organized labor alone. Furthermore, the most serious financial drain upon many health and welfare plans—with no compensating improvement in benefit value or service—has stemmed from systematic overcharges, collusion and other abuses on the part of some elements in the medical profession, motivated by avarice rather than the principle of ethical service at a reasonable fee."

CIO Will Study, Too

Likewise, the CIO has pledged "complete cooperation" with "legitimate" government investigations of alleged welfare fund frauds, and it offered to help write legislation to prevent recurrence of any abuses.

The CIO went a little further, however, and ordered a broad investigation of union welfare funds in an effort to weed out racketeering, according to another article in the *New York Times.* The story stated: "The CIO executive committee established a three-man special committee with power to investigate any allegations of maladministration of welfare or other union funds. The committee will recommend 'prompt remedial action' if abuses are found. The group also will recommend uniform standards for welfare funds and suggest any legislation it believes necessary to protect the funds."

ELECTION BRINGS SPECULATION ON STATUS OF NATIONAL HEALTH INSURANCE

Election of a Democratic-dominated Congress has brought out much guessing on the prospects of possible reintroduction of the national health insurance plan. Because committee chairmanships probably will change hands, this possibility is now more in the forefront. One of the main things the changing of control means, is a fresh look at the health legislation left unpassed, or defeated by the 83rd Congress.

According to a recent issue of the "Washington Report on the Medical Sciences," it is not unlikely that renewed agitation will be felt for passage of a national health insurance bill. The report states: "Reelected to Congress were its foremost champions of national health insurance, Senator James E. Murray (Mont.) and Rep. John D. Dingell (Mich.). . . . Chairmanship of Labor and Public Welfare Committee, which handles most national health bills, is Senator Murray's for the taking. In all probability, however, he will choose to head Interior and Insular Affairs, passing LPW gavel to the next ranking Democrat, who is Senator Lister Hill, of Alabama."

The report states that Senator Hill will then be in a key spot. It notes:

> "Alabama's senior Senator not only is due to succeed Senator H. Alexander Smith (R., N. J.) as LPW chairman but his position on Appropriations Committee will be strengthened. In all probability he will serve on, and possibly, head, the sub-committee which acts upon funds for medical research grants, financial support of national hospital expansion under Hill-Burton program, and aid to states for public health. He is an uncompromising opponent of compulsory health insurance, but believes government properly has a role it should not shirk in fostering good medical and hospital care."

FOUR DOCTORS IN HOUSE; TWO DEFEATED

The Congressional elections returned four physicians to the House of Representatives, while two more were defeated for House seats. Those reelected were Drs. Ivor D. Fenton (R., Pa.), Thomas E. Morgan (D., Pa.), A. L. Miller (R., Neb.) and Walter H. Judd (R., Minn.). A fifth, Dr. Will Neal (R., W. Va.), lost out in a bid for a second term to M. G. Burnside, a former member of Congress. And, Rear Admiral Ross T. McIntire, personal physician to the late President Roosevelt and one-time medical director of the American Red Cross, failed in his first bid for a Congressional seat. He was defeated by incumbent Bob Wilson (R., Calif.). Dr. McIntire ran on the Democratic ticket.

MRS. HOBBY PRESSES FOR REINSURANCE AGAIN

Secretary of the Department of Health, Education and Welfare, Mrs. Oveta Culp Hobby, has recently begun to actively beat the drums for the administration's reinsurance plan. Since its defeat, she, as well as President Eisenhower, has asked that it be reconsidered by the 84th Congress, convening in January.

Recently she spoke at the American Life Convention in Chicago, where a large part of her talk was devoted to this subject. Mrs. Hobby said the administration is backing reinsurance because ". . . time is running against those who seek to keep health insurance on a voluntary basis." She added: "We still strongly believe in a bill . . . which seeks to compress the experimentation of the next 20 years into less than half the time through the simple mechanism of a broad sharing of risks. We believe such a bill will . . . nurture rather than weaken the voluntary health insurance concept."

One wonders about the advisability of trying to squeeze "the experimentation of the next 20 years into less than half the time." Wouldn't it be better to go a little more slowly, and allow time for possible mistakes to be avoided by a careful scrutiny of each change before it is made hurriedly?

Besides, there are many excellent reasons for opposing reinsurance, as stated by the American Medical Association:

1. The insurance carriers themselves have all the reinsurance money needed.

2. Voluntary health insurance is making "extremely rapid" progress without reinsurance.

3. Reinsurance would not make uninsurable risks insurable.

4. Without an objectionable subsidy reinsurance would not reduce the cost of insurance or "overcome the inertia of the unwilling buyer."

ACUTE POISONING IN CHILDREN

(Continued from Page 866)

proposed poison control program could be set up initially for the Twin Cities area, rapidly expanded to counties, and ultimately to the entire state of Minnesota.

Bibliography

Arena, J. M.: Poisonings in infants and children. Pediat. Clin. North America, (Nov.) 1954.

Carver, G. M., Jr., Davidson, W. C., and Arena, J. M.: Clinic on poisoning. J. Pediatrics, 32:207, 1948.

De Sanctis, A. G., and Varga, Charles: Handbook of Pediatric Medical Emergencies. St. Louis: C. V. Mosby, 1951.

Dietrich, H. F.: Accident prevention through pediatric practice. Transactions, National Safety Council, 1950.

Editorial: Value of gastric lavage in treatment of acute poisonings. J.A.M.A., 133:545, 1947.

Gold, H.: Conference, Household Poisonings. Am. J. Med., 8:372, 1950.

Kobes, H. R., and Press, E.: Chicago Poisoning Control Program. A report to the American Academy of Pediatrics, Chicago, Illinois, (October) 1954.

Rosenfield, A. B., Allen, William: Accidents—chief killer of children. Minn. Med., 36:1141, (Nov.) 1953.

Ruben, et al.: Ingestion of poisons in children. Am. J. Dis. Child., 77:57, 1949.

von Oettingen, W. F.: Poisoning. New York: P. B. Hoeber, Inc., 1952.

Wheatly, G. M.: Accident prevention and the general practitioner. Postgrad. Med., 10:5, 1951.

1409 Willow Street

Public Health

INFLUENZA AND THE HEALTH DEPARTMENT LABORATORY

The season is now with us when localized outbreaks of influenza may be expected. Observations made in recent times indicate that different types of influenza virus give rise to outbreaks in a fairly regular periodic cycle. There are a variety of influenza virus types and these have been designated as A, B, and C. Within these types there may occur subtypes which differ slightly from types A, B, and C. It is seldom that the same variety of influenza virus is responsible for the outbreak in two successive years. Epidemics of A type influenza tend to recur every two or three years while those of B type recur at four- or five-year intervals. There is little information regarding the occurrence of influenza C. During the influenza outbreak in Minnesota in the winter of 1949-50 the medical laboratories of the Minnesota Department of Health isolated a subtype of type A influenza virus known as A-prime. This A-prime type of virus differed slightly from other known A-prime types of virus. An outbreak of influenza caused by a related A-prime type of virus occurred during the winter of 1952-53. Last winter (1953-54) there was little or no influenza reported. Consequently we need to be on the watch for it this winter.

Because there are variations of the antigenic types of the influenza viruses it is necessary to keep a close check on the type of virus responsible for an outbreak.

The Minnesota Department of Health needs to obtain information as quickly as possible regarding the type of influenza virus that may be causing any specific outbreak. Such information may be useful in making recommendations regarding the use of influenza vaccine and is necessary for epidemiological studies. One method of identifying the type of virus causing the outbreak is by laboratory study of paired blood specimens from persons ill with symptoms compatible with influenza. It is essential that two blood specimens be sent to the laboratory for study, since results of serologic tests are of significance only when a four-fold or greater rise in antibody titer can be demonstrated between specimens taken at different stages of the illness. The first specimen must be taken during the first five days of illness and sent immediately to the Department of Health. A second blood specimen must be taken ten to fourteen days after the first and sent immediately to the Health Department.

Containers for the mailing of blood specimens are available on request from the Minnesota State Department of Health, Division of Medical Laboratories, University Campus, Minneapolis 14, Minnesota. If the laboratory is to supply maximal information, it is essential that the data card accompanying the specimen container be filled in completely and that the directions for collection and submission of specimens be followed. Failure to comply will make it impossible in most cases to perform the tests properly.

Reports of outbreaks of influenza should be made promptly to the State Department of Health with as much clinical and epidemiological information as possible. The dates of onset, number of cases, ages of patients, blood counts, symptoms and any other information the physician can furnish, are all important.

It is of interest to Minnesota physicians who co-operate with the health department in supplying specimens for examination by the laboratory that the data so obtained on influenza outbreaks in this state are reported to the World Health Organization, which gathers epidemiological information from all parts of the world. All over the United States and also in several other countries are laboratories that report outbreaks of influenza and send data on such outbreaks to the WHO. The Minnesota Department of Health operates one of these "collaborating laboratories." Information collected, compared and organized by the WHO is channelled back to the co-operating laboratories. A major purpose of the information thus assembled is to make possible the ultimate preparation and use of vaccines, specifically for the type of disease that is occurring in any geographic area. As the body of knowledge about influenza increases every year, so also does the hope of developing effective vaccines.

Influenza outbreaks in recent years have not been severe, and the high death rate that occurred in the 1918 pandemic have never been repeated. There is, however, no guarantee that this could not happen. It is seldom that the same variety of influenza virus is responsible for an epidemic in two successive years. This tendency of the viruses to be constantly changing increases the difficulty in the control of the disease. That is a major reason for urging physicians to submit specimens to the health department laboratories in all cases of illness believed to be influenza and to report all such cases to the Division of Disease Prevention and Control.

Henry Bauer, Ph.D.
Director, Division of Medical Laboratories
Minnesota Department of Health

◆ Reports and Announcements ◆

AMERICAN BOARD OF OBSTETRICS AND GYNECOLOGY

The next scheduled examination (Part I), written examination and review of case histories, for all candidates will be held in various cities of the United States, Canada, and military centers outside the continental United States, on Friday, February 4, 1955. Case Abstracts numbering 20 are to be sent by the candidate to the Secretary as soon as possible after receiving notification of eligibility to the Part I written examination.

For further information, contact the office of the Secretary, Robert L. Faulkner, M.D., 2105 Adelbert Road, Cleveland 6, Ohio.

MINNESOTA SOCIETY OF NEUROLOGY AND PSYCHIATRY

Dr. Reynold A. Jensen, was elected president of the Minnesota Society of Neurology and Psychiatry when the group had its November meeting at the Town and Country Club, Saint Paul. Dr. Royal C. Gray was elected vice president, and Dr. Joseph A. Resch will be secretary-treasurer.

Candidates for active membership who were voted into the Society at the meeting are Dr. A. Margaret Bailey, Dr. G. Wendell Hopkins, Dr. Carl D. Koutsky, Dr. J. Benjamin Lund and Dr. Frances P. Olson.

During the scientific meeting Dr. Gordon R. Kamman spoke on "Critical Evaluation of a Total Push Program for Regressed Schizophrenic Patients in a State Hospital" and Dr. Robert L. Merrick talked on "Greater Superfician Petrosal Neurectomy."

MINNESOTA SOCIETY OF INTERNAL MEDICINE

Officers for the coming year were elected at a Rochester meeting of the Minnesota Society of Internal Medicine. New officers include Dr. H. C. Habein, Rochester, president; Dr. J. F. Borg, Saint Paul, vice president, and, Dr. W. D. Coventry, Duluth, secretary-treasurer.

1955 PAUL-LEWIS AWARD

Dr. Paul D. Boyer, Professor of Chemistry at the University of Minnesota, has been chosen the 1955 recipient of the Paul-Lewis Laboratories Award in Enzyme Chemistry, the Laboratories has announced.

Dr. Boyer's work has been concentrated on problems of mechanisms of reactions in enzymes and on oxidation-reduction of biologically important compounds. The Award is administered by the American Chemical Society and consists of a gold medal and $1,000. The Award will be presented to Dr. Boyer at the 127th national meeting of the American Chemical Society in Cincinnati, Ohio, March 29-April 7, 1955.

UNIVERSITY GETS TB GRANT

The Minnesota Tuberculosis and Health Association has presented a grant of $7,500 in Christmas Seal funds to the University of Minnesota, it has been announced. The grant will finance the H. Longstreet Taylor research project on tuberculosis and related chest diseases under the direction of Dr. J. A. Myers, professor of medicine and public health.

CONTINUATION COURSES

The University of Minnesota announces a continuation course in *Neurology* which will be presented at the Center for Continuation Study, February 7 to 12, 1955. The course, which will be of interest both to physicians in general practice and to those with a special interest in neurology, will stress diagnosis and management of the most frequently-met neurological disorders. Guest speakers will include Dr. G. Milton Shy, National Institute of Neurological Diseases and Blindness; Dr. Howard Fabing, vice president, American Academy of Neurology, Cincinnati; and Dr. Edward B. Schlesinger, clinical assistant professor of neurosurgery, Columbia University College of Physicians and Surgeons, New York City. The annual Johnston lecture will be presented by Dr. Seymour S. Kety, also of the National Institute of Neurological Diseases and Blindness. The course will be presented under the joint direction of Dr. A. B. Baker, professor and head of Division of Neurology, and Dr. William T. Peyton, director, Division of Neurosurgery, University of Minnesota Medical School.

The University of Minnesota and the Minnesota Division of the American Cancer Society will join in the presentation of a continuation course in *Cancer Detection for General Physicians* next February 17 to 19, 1955, at the Center for Continuation Study. This year's program will consist of a series of lectures and informal discussions concerning cancer detection techniques. Those registrants wishing to observe and participate in certain examination procedures will be given an opportunity to do so.

RENEWAL OF NURSES' LICENSES

Minnesota registered nurses who are currently renewed for 1954 and plan to be employed as registered nurses in this state in 1955, are reminded that renewal of license is due by December 31, 1954, the Minnesota State Board of Examiners has announced. Those who are currently renewed and expect to retire from employment as registered nurses in Minnesota (by marriage, moving to another state, or other reasons) and do not plan to be employed in Minnesota at any time

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Woman's Auxiliary

PRESIDENT'S LETTER

Greeting old friends and meeting new friends on the local level has been a pleasurable and stimulating experience this fall. Regional Advisors Mrs. O. M. Heiberg, Mrs. T. R. Fritsche, and Mrs. W. E. Wellman arranged regional meetings in Fairmont, New Ulm and Rochester. The programs for the meetings were similar in effect, designed to bring the members up to date on problems and responsibilities of the auxiliary. After the meeting in Fairmont, we were invited to attend the Martin County Hospital Auxiliary meeting at which our own Maude Halpern* gave a superb presentation on "Working Together for Health and Safety." Earl Dresser, Assistant Administrator of Asbury Hospital in Minneapolis, was guest speaker.

Your president presided at all the meetings, bringing up-to-date auxiliary news to the members after welcoming the women in behalf of the state auxiliary. Legislation news and mental health news was presented. We are urged to offer our services to the schools in our community so as to help cope with the added responsibilities that have resulted from the overcrowded schools. It is the general opinion that problems of the school and the home contribute greatly to the increase of the mentally ill among the school children.

Mrs. H. H. Fesler, president-elect, addressed the women on organization news, and Mrs. C. L. Sheedy of Austin addressed the group on Today's Health and the American Medical Education Foundation. We are urged to participate, also, in "Operation Christmas" which calls for one or more subscriptions to *Today's Health* as a gift from a doctor's wife to a layman. Teachers, new mothers, beauty shops, barber shops, schools, are excellent for placing our health magazine.

We are urged to support the American Medical Education Foundation each year. As our State University has benefited from this fund far beyond the total contributions from our state to the amount of $20,000.00 as reported last year, we should make this a yearly obligation.

In Rochester, Mrs. Stanley Peterson of Austin addressed the women on "Allied Medical Careers," and Mrs. Harold Wahlquist addressed the group on "Health Days."

On November 5, your president participated on the Question-and-Answer Panel of the all-day press conference sponsored by the *Minneapolis Star and Tribune* at the Radisson Hotel. This was a most interesting day. Many tips on club news were given.

An excellent address on "Forces at Work in the Middle East" was given by Yahya Armajani, Professor of History, Macalester College, Saint Paul. "1954 Post Mortems" was presented by Doris Fleeson, Washington correspondent.

Chairmen should be called "Press" and not Publicity Chairmen. Don't sit on news. And that reminds me that our own Minnesota Medicine welcomes auxiliary news of all activities of the county auxiliaries. Mail this news to Mrs. L. Raymond Scherer, 1930 Irving Place, Minneapolis 5, Minnesota.

Congratulations to the newly re-organized Kandiyohi-Swift-Meeker County Auxiliary. We wish the members a most successful year.

With the holiday season approaching, I wish all the members of the auxiliary and every doctor's wife, a Merry Christmas and a Happy New Year.

Mrs. Peter S. Rudie, *President*
Woman's Auxiliary to the Minnesota
State Medical Association

RAMSEY ACTIVITIES

Mrs. L. T. Simons

Mrs. W. P. Gardner called a board meeting on November 15 in the Ramsey County Medical Society library. Committee reports were given and plans for the coming meetings were discussed. Cancer dressings were made during and following the meeting.

Mrs. W. A. Gleason, press chairman, and Mrs. W. P. Gardner, president, attended the *Minneapolis Star and Tribune* press conference in the Radisson Hotel, November 5. Mrs. P. S. Rudie, state president, was a member of the panel. Doris Fleeson, Washington correspondent, presented an interesting discussion on trends in the news.

NEW COUNTY AUXILIARY ORGANIZED

Mrs. Harold H. Fesler,

State Organization Chairman

Thursday, October 21, was an important date for the Woman's Auxiliary to the Minnesota State Medical Association. A new county society was organized, at Willmar. Mrs. P. S. Rudie, state president, attended a meeting in September, in Willmar, and discussed the possibility of forming an auxiliary.

We were invited to attend a meeting at the Country Club with the doctors, which was most enjoyable. While they held their meeting, following the dinner, the women retired to the home of Mrs. L. C. Gilman,

Mrs. Rudie talked on the history, activities and program of the auxiliary and I, as organization chairman, gave the aim and purpose. A motion was made and thoroughly discussed, that a charter be drawn up, and it was unanimously decided to form the Kandiyohi-Swift-Meeker County Auxiliary—Mrs. Charles Waas served as temporary secretary. Officers were elected with Mrs. Willard Peterson as president. Fifteen attractive, young and enthusiastic members signed the

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*Mrs. Halpern's address will be published in the January issue.

In Memoriam

CHARLES M. ADKINS

Dr. Charles M. Adkins, eighty-two years old, died at his home in Thief River Falls after a long illness November 2, 1954. He became a life member of the Minnesota State Medical Association after forty-one years of membership in the Red River Valley Medical Society in 1951.

Dr. Adkins served as Pennington County coroner for many years and spent three years in the Philippines during the Spanish American War.

He was born in Wayne County, West Virginia, and went to high school at Fairhaven, Mississippi. He was graduated in medicine from the Kansas City Medical College of the University of Kansas and served his internship at the Kansas City General Hospital. He practiced at Ogema from 1909 to 1912 and at Grygla for short periods. Most of his professional career was spent at Thief River Falls where he was city physician and local surgeon for the Great Northern Railway as well as director of Physicians' Hospital.

Survivors are his wife, the former Alice Solem; one daughter, Arda, Thief River Falls; five sons, two of whom are physicians, Dr. Galen Adkins of Anoka, and Dr. Douglas Adkins of Minneapolis, Jack Adkins, also of Minneapolis, and Peter and Caylor Adkins of Thief River Falls; one sister, Mrs. Dellis Wisehard of Cass Lake; and one brother, Jones Adkins of Minneapolis.

WILLARD E. ARCHER

Dr. Willard E. Archer died of a heart attack at the age of fifty-five, October 13, 1954, at Falls Creek, Wisconsin. He was on leave from the staff of the Anoka State Hospital.

Dr. Archer was born at West Union, Iowa, and was graduated in medicine from Marquette University. He served his internship at St. Elizabeth's Hospital in Appleton, Wisconsin, and spent two periods in post-graduate work at the Cook County Post-Graduate School in Chicago. He practiced for twelve years at Dale, Wisconsin, and for fourteen years specializing in obstetrics and gynecology at Appleton. After that he spent three years in neurology and psychiatry at the Veterans Administration Hospitals at Tomah, Wisconsin, and Hines, Illinois, and joined the staff of the Anoka Hospital in 1952.

Dr. Archer was a member of the Hennepin County Medical Society, the Minnesota State Medical Association and the American Medical Association, and he had served as president of the Outagamie County Medical Society of Wisconsin.

Survivors include his wife, the former Florence Heiserman; two daughters, Mrs. M. D. Davis, Bremerton, Washington, and Patricia, a nurse at Methodist Hospital, Madison, Wisconsin; and one son, Corporal Thomas Archer, Fort Ord, California.

HERMANUS DE BOER

Dr. Hermanus De Boer of Edgerton died at Pipestone Hospital October 27, 1954. He was eighty-three years old and had practiced at Edgerton since 1906.

Until he retired from active practice in 1946, Dr. De Boer had played an active part in the life of the community. In the early days he made hundreds of night calls in the countryside by team and bob-sled and he had brought close to three thousand infants into the world, many of whom were present at a special "Dr. De Boer Day," celebrated under sponsorship of the Edgerton Civic Club in March, 1949. For 42 years he served as a member of the Edgerton Board of Health and for 33 years on the Board of Education. He was mayor of the village for 22 years.

He was also a life member of the Minnesota State Medical Association, a member of the American Medical Association, and one-time president of the Southwestern Minnesota Medical Society. He was a staff member of the Ashton Memorial Hospital at Pipestone and of the Home Hospital at Slayton.

Dr. De Boer began a varied and adventurous career as a lawyer, having received his law degree at Kent College of Law in Chicago in 1894. He practiced law in Chicago for two years and then left for South Africa to open a steam laundry there. He was joined by the first Mrs. De Boer, the former Dora Sietsema, but they returned to Chicago shortly afterwards and Dr. De Boer entered the National Medical University of Chicago from which he was graduated in 1903. He practiced for three years in Chicago before going to Edgerton.

The first Mrs. De Boer died in 1949 and Dr. De Boer remarried in 1953. He is survived by his widow and an adopted son, Theo, of Plainview.

JOSEPH H. DUDLEY

Dr. Joseph H. Dudley, physician and leader for many years in civic affairs in Windom, died October 21, 1954, at the age of eighty-two. He had been a patient at the Windom hospital for more than a year and had been in ill health for several years.

Dr. Dudley opened his practice in Windom in 1909 after practicing for ten years following graduation from Rush Medical College in Chicago, at Heron Lake.

He headed the Windom Civic and Commerce Association as president for many years and served as judge of probate for Cottonwood county from 1940 to 1952. A stalwart Republican in politics, he also served as chairman of the county congressional committee at one time. He left his practice three times, in 1906, 1916 and 1930, to take post-graduate training in Chicago, and was a member of the Southwestern Minnesota Medical Society, the Minnesota State Medical Association and the American Medical Association. He was also a member of the Windom Presbyterian Church, a Mason and a member of the Woodmen of America, the Royal Arcanum, the Modern Brotherhood of America and the Royal Neighbors.

The only survivors are an adopted son, P. T. Dudley of St. Louis, Missouri; two grandchildren; and a nephew, Art Dudley of St. James.

EDWARD THOMAS FITZGERALD

Dr. Edward T. Fitzgerald, who practiced for forty years at Morris, died October 13, 1954, at the age of seventy-nine. He was a life member of the Minnesota State Medical Association and had practiced at Morris from 1911 until his retirement in 1940 because of ill health.

Dr. Fitzgerald was born at Boone, Iowa, and was educated in Boone County schools, the Iowa City Academy and Creighton Medical College at Omaha from which he was graduated in 1907. He practiced at LeMars, Iowa, for three years before going to Morris.

He served on the State Board of Health from 1936 to 1940, and was a member of the West Central Minnesota Medical Society, the Minnesota State Medical Association, and the American Medical Association.

GORDON BALGARNIE NEW

Dr. Gordon B. New, distinguished laryngologist and plastic surgeon and one of the pioneers in the organization of the Mayo Clinic, died at Methodist Hospital in Rochester after a short illness, October 27, 1954.

Dr. New was sixty-eight years old at the time of his death. He retired from the clinic in 1950 and had been extensively honored in this country and abroad for his contributions to the field of plastic surgery. One of the pioneers in his field, he was also an international authority on tumors of the nose and throat and held top offices in many medical and surgical organizations. In 1952, he was awarded the top honor that can be given to a surgeon by the American Society of Plastic and Reconstructive Surgery. He received many awards for exhibits on correction of facial deformities and on cancer of the mouth.

Dr. New was born in Hamilton, Ontario, Canada, in 1885. He received a degree in dental surgery from the University of Toronto in 1906, the degree of doctor of medicine in 1915. He served an internship in the City Hospital of Hamilton and came to Rochester in 1919 as head of the section of postoperative treatment and as assistant in laryngology and rhinology, oral and plastic surgery.

When the Mayo Foundation was established as part of the graduate school of the University of Minnesota in 1915, Dr. New was appointed assistant professor of rhinology, laryngology and stomatology. Later he became professor of laryngology.

He was elected president of the American Association of Oral and Plastic Surgeons in 1931, and president of the American Academy of Ophthalmology and Otolaryngology in 1946. In 1947, he was vice president of the American College of Surgeons. He was a member of a long list of scientific societies as well as the Olmsted-Houston-Fillmore-Dodge County Medical Society, the Minnesota State Medical Association and the American Medical Association.

He is survived by his wife, the former Ethel Bailey; two sons, Gordon of Solvang, California, and Robert of Lakewood, Ohio; and two grandsons. A daughter, Marian Elizabeth, and two brothers preceded him in death.

JAMES ALFRED SANFORD

Dr. James A. Sanford, who practiced at Farmington and Savage for more than forty years, died at his winter home in Hot Springs, Arkansas, November 15, 1954. He was seventy-five years old, a life member of the Minnesota State Medical Association and a member, since 1951, of its Fifty Club.

Dr. Sanford was a leading figure in the community for many years. He built the modern hospital at Farmington, helped found its telephone company and served on village councils, which gave the town sewer and water, electricity and paved streets. He was regarded with great pride by fellow townsmen as the typical "country doctor" and he had recently written a book about his experiences entitled "The General Practitioner" which is to be published soon by Doubleday & Co., of Garden City, New York.

Dr. Sanford was born at Alderley, Wisconsin, in 1879 and went to high school in Hartford, Wisconsin. He took his medical training at the University of Minnesota, from which he was graduated in 1901. He practiced in New Market for five years and in Minneapolis for three years before moving to Farmington. He was a member of the Scott-Carver Medical Society and the American Medical Association, as well as the Minnesota State Medical Association.

Surviving are his wife, Lillian; a daughter, Mrs. Helen Rondestvedt of Sauk Centre; one grandson, four granddaughters and his daughter-in-law, Mrs. Lucy Sanford, widow of his son, James, of Farmington.

RENEWAL OF NURSES' LICENSES

(Continued from Page 899)

in 1955 should request placement in the non-practicing status, to begin in 1955.

Minnesota registered nurses who now are in the non-practicing status but who return to employment must renew their license prior to employment. Applications for 1955 renewal or non-practicing status are attached to the 1954 renewal certificate. Applications and one dollar fee (only for those renewing for 1955) may be sent to the Minnesota State Board of Examiners of Nurses, 700 Minnesota Building, St. Paul 1, Minnesota. Money order or certified bank check is the preferred means of payment. *All* Minnesota Licensed Practical Nurses *must* renew their licenses on a calendar year basis.

Letters to the Editor

Arthur H. Wells, M.D.
Minnesota Medicine
519 East First St.
Duluh 5, Minnesota

Dear Doctor Wells:

Jaundice Associated with the Administration of Chlorpromazine (Thorazine). The correct etiologic diagnosis is, of course, essential for the proper treatment of the jaundiced patient. Among other important factors the critical decision of surgical intervention depends upon the accuracy of the differential diagnosis. Unfortunately the problem has often been difficult and the recent reports of jaundice associated with the administration of Chlorpromazine (Thorazine) would seem to further complicate the diagnostic difficulties.

Lemire and Mitchell (Proceedings of the Central Society for Clinical Research, 27:70, 1954) have recently reported three cases of "regurgitation type jaundice" associated with Chlorpromazine (Thorazine) administration. In each case evaluation of liver function revealed a hyperbilirubinemia, an increase in the alkaline phosphatase activity and a normal cephaline cholesterol flocculation. Since the drug was not considered etiologically involved and since the evaluation of liver function alteration suggested extrabiliary tract obstruction all three patients underwent laparotomy. There was no evidence of such extrahepatic obstruction. Liver biopsies revealed plugging of the bile canaliculi with bile. The parenchymal cells showed brownish pigmentation deposition but were otherwise normal. There were some areas of spotty infiltration of inflammatory cells within the liver lobule.

There are, of course, many drugs which are hepatotoxic; however, for the most part these agents, if they result in the development of jaundice, are associated with liver function abnormalities normally considered compatible with hepatocellular injury. Chlorpromazine (Thorazine) differs markedly in this respect in that it is capable of producing jaundice with liver function abnormalities ordinarily considered compatible with extrabiliary tract obstruction or so-called surgical jaundice. The following brief case report illustrates another instance of jaundice associated with Chlorpromazine (Thorazine) administration.

Case Report.—A 67-year-old white woman was known to have hypertension, coronary artery disease and moderately severe chronic anxiety. It was apparent that much of her disability was related to her neuropsychiatric problems. She was inadequately controlled by mild barbiturate sedatives and on October 20, 1954, she was started on Chlorpromazine (Thorazine) in a dose of 25 mgms. three times daily. There was considerable subjective improvement; however, on November 10, 1954, twenty-two days after the institution of drug therapy, she was noted to have definite icterus of the sclera. There was no associated pain and initially there was no anorexia, nausea or vomiting. There was rather marked pruritus of the skin. The liver edge was palpable approximately 3 cm. below the right costal margin but was not tender. The spleen was not enlarged. Liver function studies revealed the serum bilirubin to be 5.1 mgms. per cent in one minute with a total of 7.8 mgms. per cent. The cephalin cholesterol flocculation test was negative at 24 and 48 hours. The alkaline phosphatase activity of the serum was 52 King-Armstrong units.

In the above recorded case the findings were considered identical to those noted by Lemire and Mitchell and therapy was to consist of simple withdrawal of the Chlorpromazine (Thorazine) administration. To date it would appear that this syndrome has been primarily noted in elderly patients and present information suggests that the process is reversible. The importance of the syndrome is not so much in reporting another drug capable of producing jaundice but in recognizing that the liver function abnormalities are not those of hepatocellular damage but rather those commonly associated with extrahepatic biliary tract obstruction. It is most important that these patients be spared unnecessary surgical intervention.

Very truly yours,
Wyman E. Jacobson, M.D.
St. Louis Park Medical Center
Department of Medicine

November 15, 1954
St. Louis Park

RAUWOLFIA

(Continued from Page 872)

psychotherapy against anxiety, *causeless* anxiety —the master symptom of all of us, belonging as we all do, to that part of the population that may be called the non-institutionalized insane.

Rauwolfia poses a question: Are many of these anxieties prodromal symptoms of deeper mental illness, of psychoses?

If they are, then with Rauwolfia (and chlorpromazine too) general practitioners are likely to wake up to find themselves in the front line of the fight, not to build more mental hospitals, but to begin to empty present institutions for the insane.

This is the glory of American medicine: that there is no government or totalitarian authority telling physicians they've got to save lives of hypertensives and keep the patients from going crazy—or else. Now that Rauwolfia is showing that these things can often be done, it's purely between the doctor and his God or if he hasn't a God, his conscience; it's the individual doctor's lonely responsibility.

NEW COUNTY AUXILIARY ORGANIZED

(Continued from Page 900)

charter and we are most happy to welcome them, one more active group to strengthen our organization.

Mrs. H. E. Bakkila of Duluth and Mrs. Charles Waas of Saint Paul, both past state presidents of the auxiliary, Mrs. John Dordal of Sacred Heart, first vice president, Mrs. E. R. Hudec of Echo, regional advisor, and Mrs. M. I. Hauge of Clarkfield, chairman of Today's Health, were all present at this meeting and enjoyed the thrill as much as I. We are happy to welcome our new friends and wish them the best of luck, success and happiness.

◆ Of General Interest ◆

Several Mayo Clinic staff members attended national meetings last month. **Dr. N. W. Barker** and **Dr. E. A. Hines, Jr.,** were present at the meeting of the American Society for the Study of Arteriosclerosis in Chicago. Dr. Barker is a member of the society's board of directors. **Dr. H. B. Burchell** attended a meeting of the research committee of the American Heart Association in Boston and **Dr. R. Q. Royer** was in Hollywood, Florida, for the meeting of the American Society of Oral Surgeons. Present at a session of the American Board of Preventive Medicine in New York City was **Dr. J. H. Tillisch.**

* * *

Dr. R. O. Sather of Crookston was in Chicago for a course at the University of Chicago on recent advances in the field of internal medicine.

* * *

Dr. Roberts Zemmers of Duluth was one of twenty foreign-born persons who met President Eisenhower in Washington, D. C., last month. The group received naturalization papers as citizens that day. Dr. Zemmers, a native Latvian, is associate anesthesiologist at St. Luke's Hospital.

* * *

New president-elect of the American Society of Plastic and Reconstructive Surgery is **Dr. Frederick A. Figi** of the Mayo Clinic, Rochester. Dr. Figi will be installed into the office, now held by **Dr. William G. Hamm** of Atlanta, at next year's meeting.

* * *

Dr. R. P. Hallin of Worthington has been elected president of the Southwestern Minnesota Medical Society. Also elected at the annual meeting at Pipestone were **Dr. H. A. Christiansen,** Jackson, president-elect; **Dr. Victor Doman,** Lakefield, vice president; and **Dr. O. M. Heiberg,** Worthington, secretary. Named delegates to the state medical convention were **Dr. John Lohmann,** Pipestone, and Dr. Heiberg. Alternates are Dr. Hallin and Dr. Christiansen. Voted into the society by transfer were **Dr. Milton Plucker** and **Dr. Christian A. Rohrer,** both of Worthington.

* * *

Four Mayo Clinic physicians participated in national meetings the early part of November. **Dr. R. E. Fricke** gave a paper on the subject "Late Recurrences of Pelvic Cancer: With Description of an Unusual Case" at a meeting of the American Therapeutic Society in St. Louis. Speaking before the Interim Scientific session of the American Rheumatism Association in Bethesda, Maryland, was **Dr. L. E. Ward,** who spoke on "Effects of Aldosterone and 9-Alpha-flourohydrocortisone Acetate Administered to Rheumatoid Patients." **Dr. K. N. Ogle** was in Toronto to attend a joint meeting of the Armed Forces-National Research Council Vision Committee and the Vision Panel of the Canadian Defense Research Board. **Dr. H. F. Polley** attended a meeting of the American Rheumatism Association in Bethesda, Maryland.

* * *

Dr. W. O. Lundberg, director of the University of Minnesota's Hormel Institute, has been invited to be an original signer of a proclamation establishing an international society for fat science. Only two other Americans, **Dr. A. R. Baldwin,** director of research, Cargill, Inc., and **C. E. Morris** of Armour and Co., president of the American Oil Chemists Society, have been asked to serve as sponsors of the organization. Scientists from sixteen countries have agreed to serve as sponsors for the organization, which will include men working in the fields of chemistry, biology, medicine, engineering and agriculture.

* * *

Dr. John Fischer of Owatonna has been elected coronor of Steele County. He was endorsed by the Steele County Medical Society.

* * *

Dr. A. M. Struthers, a fellow in plastic surgery in the Mayo Foundation, has been awarded second prize in the annual essay contest of the Foundation of the American Society of Plastic and Reconstructive Surgery. Dr. Struthers won the prize, a three-month scholarship for study in plastic surgery centers here and abroad, for a paper on the topic "An Experimental Study of Polyvinyl Sponge as a Substitute for Bone."

* * *

"The RH Factor in Practice" was the topic of a talk given by **Dr. James P. O'Keefe** of St. Cloud at a meeting of the Kandiyohi-Swift-Meeker Tri-County Medical Society. The dinner session was held in Willmar.

* * *

Dr. Albert J. Schroeder of Minneapolis addressed the Specialty Section of the Wisconsin State Medical Society in Milwaukee on the topic "Infectious Croup." Dr. Schroeder also conducted a round-table discussion on the subject "Hemolytic Disease of the Newborn."

* * *

Newly-elected chief of staff of the Waconia Community Hospital is **Dr. Leighton W. Larson,** Waconia. Vice chief of staff will be **Dr. Joseph Selmo** of Norwood. **Dr. J. W. Clarke,** Watertown, was elected secretary-treasurer. Executive committee members are **Dr. R. E. Pogue** of Watertown and **Dr. R. S. Johnson** of Excelsior.

* * *

Dr. D. H. Petersen of St. Olaf College, Northfield, and **Dr. J. W. Hanson,** Carleton College, Northfield, attended the regional meeting of the

American College Health Association recently at Luther College in Decorah, Iowa.

* * *

At home in Minneapolis are **Dr. and Mrs. Karel Bedrich Absolon**, who were recently married in St. Ignatius Church. The bride, the former Mary Bendix, is the daughter of **Dr. and Mrs. Lester H. Bendix** of Annandale. Dr. Absolon's parents are **Dr. and Mrs. Karel Absolon** of Brno, Czechoslovakia.

* * *

Mayo Clinic staff members who attended the International Symposium on hypophyseal growth hormone: nature and actions, in Detroit, were **Dr. A. Albert, Dr. E. V. Flock** and **Dr. A. B. Hayles.** The symposium was sponsored by Henry Ford Hospital.

* * *

"Social Work and Medicine" was the topic of the regional Minnesota Welfare conference last month in St. Paul. Speakers for the meeting included **Dr. Dale C. Cameron,** State Department of Public Welfare medical director; **Dr. William Maloney,** assistant dean of the University of Minnesota Medical School; **Dr. Lyle Hay,** surgeon and chief of staff at Mount Sinai Hospital, Minneapolis; and **Dr. Robert Mooney,** St. Paul internist.

* * *

University of Minnesota board of regents has promoted **Dr. Robert A. Good** to American Legion memorial research professor, replacing **Dr. Lewis Thomas,** who resigned to go to New York University. Dr. Good, a graduate of the University, was associate professor of pediatrics. He has been a fellow of the National Foundation for Infantile Paralysis, a visiting investigator at the Rockefeller Institute of Medical Research and holder of a Helen Hay Whitney Foundation fellowship. At the University he has been Markle Foundation scholar in medical sciences.

* * *

Dr. and Mrs. Merrill D. Chesler of Minneapolis attended the meeting of the American Society of Plastic and Reconstructive Surgery at Hollywood, California, in October. Dr. Chesler was elected to membership in the Society at the meeting.

* * *

Elected to membership of the International Society of Hematology was **Dr. Frances E. Schaar** of Minneapolis. The selection was made at the Fifth Congress of the Society in Paris, France.

* * *

Dr. Russell M. Wilder, former professor and head of the Mayo Clinic's Department of Medicine, has been awarded the American Medical Association's Joseph Goldberger award in clinic nutrition. The award was received by Dr. Wilder at ceremonies in Washington, where the annual meeting of the Food and Nutrition Board of the National Research Council took place.

* * *

One of Bemidji's pioneer physicians, **Dr. D. F. McCann,** has retired from the practice he founded in 1916. Dr. and Mrs. McCann have left for California. Dr. McCann, sixty-five, was graduated from Columbia University College of Physicians and Surgeons and served his internship in New York hospitals. The Bemidji practice was interrupted when Dr. McCann served in World War I as a regimental surgeon, and for several years when the McCanns resided in McCloud, California. In Bemidji, Dr. McCann founded the Bemidji Clinic with **Dr. T. P. Groschupf** and **Dr. D. D. Whittemore.**

* * *

Dr. and Mrs. Harold D. Nagel have left Waconia to reside in Minneapolis. The Nagels had resided in Waconia for thirty-one years. They turned over their hospital, built in 1940, and nurses' home to the Waconia Community Hospital Association.

* * *

Life of a Rice County pioneer doctor, **Dr. N. M. Bemis,** was reviewed at a recent meeting of the Rice County Historical Society at Faribault. The paper was prepared by **Dr. Adolph Hanson** of Faribault. Dr. Bemis, the first doctor to reside in Rice County, practiced there from 1856 to 1891, date of his death.

* * *

Five Minnesota physicians have been re-elected to the board of directors of the Minnesota Tuberculosis and Health Association. They are **Dr. Leon H. Flancher** of Crookston, **Dr. Charles Sheppard** of Hutchinson, **Dr. V. M. Johnson** of Dawson, **Dr. W. H. Valentine** of Tracy, and **Dr. A. L. Vadheim** of Tyler. The men are officers in their county Christmas Seal organizations. Another Crookstonite elected to the Association was Mrs. M. O. Oppegaard. The board members were re-elected at the Seal organization's annual session in St. Paul.

* * *

Dr. N. W. Barker was elected president of the voting staff of the Mayo Clinic at the annual meeting of the group in November. He succeeds **Dr. F. J. Heck.** Other officers named were **Dr. L. E. Prickman,** vice president; **Dr. D. G. Pugh,** re-elected secretary;**Dr. L. D. Vaughn,** first councilor, and **Dr. R. R. Kierland,** second councilor. In addition to the election, the staff members presented emeritus awards to **Drs. S. W. Harrington, A. B. Nevling, F. P. Moersch** and **H. W. Woltmann,** who have retired during the year. Action was also taken confirming positions of **Dr. H. K. Gray** and **Mr. J. W. Hartwick** on the board of governors. Introduced as new members of the voting staff were **Drs. R. O. Brandenburg, A. J. Bruwer, R. L. Faucett, R. W. Gifford, Jr., N. O. Hanson, H. F. Helmholz, Jr., F. T. Maher, W. F. McFuckin, R. H. Miller, A. L. Orvis, J. E. Osborn, R. J. Reitemeier, R. A. Rovelstad, R. E. Symmonds** and **P. E. Zollman.**

* * *

Dr. L. L. Sogge of Windom, Cottonwood County Christmas Seal chairman for over thirty years, was honored at a district workshop meeting of Seal committees at Worthington. A medal, the first to be awarded in Minnesota, was given Dr. Sogge, on behalf of the National Tuberculosis Association, by **Dr. S. A. Slater**, principal speaker at the meeting. Dr. Sogge was cited for his fifty years of service in tuberculosis work.

* * *

"Fluoridation of Water" was the topic of a talk given by **Dr. Dean Soderberg**, Duluth, at a combined meeting of the Second District Minnesota Nurses and Practical Nurses groups. The meeting was held in St. Mary's Hospital.

* * *

Dr. Robert Kallsen, who has been in Crosby since June in association with **Dr. C. M. Marshall** and **Dr. R. E. Kelley,** will leave for California to take up practice. Returning to Crosby is **Dr. James Nixon,** who has been on duty with the Navy in California and overseas.

* * *

Dr. Fred W. Wittich, Minneapolis, president of the International Association of Allergology, spoke at the first graduate symposium on geriatric medicine last month in New York. Dr. Wittich also participated in a panel discussion on allergies in older patients.

* * *

Representing western Minnesota at the American College of Surgeons sessions in Atlantic City, New Jersey, was **Dr. I. L. Oliver** of Graceville. Dr. Oliver also visited his son, Robert, at Trinity College, Hartford, Connecticut.

* * *

Dr. Kenneth R. Larson of St. Paul attended sessions of the third Pan-American Congress on Endocrinology, November 21-27, in Santiago, Chile. Dr. Larson and Mrs. Larson also visited other Central and South American points during their trip.

* * *

Guest lecturer at the National University, Bogota, Columbia, South America, for a three-week period was **Dr. Harold O. Peterson** of St. Paul. Dr. Peterson, who was accompanied by his wife, lectured on the technique of myelography from November 19 to December 8. Dr. Peterson is clinical professor of radiology at the University of Minnesota and director of radiology at the Charles T. Miller and Children's Hospitals in St. Paul. The Petersons visited in Rio de Janiero, Brazil, and Lima, Peru, en route to Columbia.

* * *

Dr. John Falls of Chicago, Illinois, has joined the staff of the Interstate Clinic at Red Wing. Dr. Falls, a graduate of the University of Illinois Medical School, will specialize in obstetrics and gynecology in association with **Dr. Grant Hartnagel.**

* * *

Dr. Charlotte J. Morrison has reopened offices at 911 Medical Arts Building, Minneapolis. Her practice is limited to pediatrics.

Dr. Wesley W. Spink, professor of medicine at the University of Minnesota, was elected recently to a two-year term as president of the Minnesota Medical Foundation, succeeding **Dr. Owen H. Wangensteen,** head of surgery at the University. Vice President is **Dr. R. S. Ylvisaker,** clinical associate professor of medicine. **Dr. Robert B. Howard,** director of postgraduate medical education, succeeded Dr. Spink as secretary-treasurer.

* * *

Patients of Sunnyrest Sanatorium at Crookston honored **Dr. L. H. Flancher**, superintendent, at a farewell party recently. Dr. Flancher will assume the post of public health superintendent for the district. Sunnyrest Sanatorium has been merged with Oak Park Sanatorium at Thief River Falls. **Dr. Baldwin Borreson,** superintendent of Oak Park for almost twenty years, will take charge of the combined groups at Crookston. The combined district, which will be headed by Dr. Flancher, includes Polk, Norman, Pennington, Red Lake, Marshall and Roseau counties.

* * *

Dr. Ralph Rossen, head of Hastings State Hospital, has resigned to take over a position as director of a private mental research project. Dr. Rossen, Minnesota's first mental health commissioner, will become director of the L. E. Phillips psycho-biological research fund project at Mount Sinai Hospital, Minneapolis. Dr. Rossen took over his new duties the first of December. **Dr. Jorge Lazarte** is acting head of Hastings Hospital.

* * *

Guest speaker at a clinical conference in November in Glencoe was **Dr. William F. Maloney,** assistant dean of the University of Minnesota Medical School. Dr. Maloney spoke on heart disease.

* * *

Dr. Donald McMullen, chief of the department of medical zoology, Walter Reed Army medical center, Washington, D. C., was principal speaker at a University of Minnesota continuation course on clinical microscopy and parasitology. The course was held in November.

* * *

Training with the Air Force at Montgomery, Alabama, is **Dr. Rolf Daehlin,** formerly of Fergus Falls. Dr. Daehlin will be stationed at Spokane, Washington, after completing training. He will be in charge of pediatrics at Fairchild Air Force Base.

* * *

Guest speaker at a meeting of the Duluth District Dental Society and the St. Louis County Medical Society in November was **Dr. Thomas J. Dry** of Rochester. Dr. Dry spoke on "Progress in the Treatment of Heart Disease by Surgical Means." **Dr. Alex Kellem,** president of the dental group, presided at the meeting.

* * *

Dr. R. P. Pearsall, Virginia physician and city health officer, was commended by the Virginia Red Cross branch board of directors for his outstanding service to the Red Cross. Recognition was made at a November board meeting.

* * *

Two Blue Earth physicians, **Dr. D. V. Smith** and **Dr. George Drexler,** participated in postgraduate courses in November in Chicago.

NEW LOCATIONS

Dr. Ivan Sawaryniuk, who has been associated with Dr. H. D. Nagel in Waconia, will set up practice there in the Diessner Building. Dr. Sawaryniuk, a graduate of the University of Frankfurt, Germany, came to Waconia from Mt. Sinai Hospital in Minneapolis.

* * *

Dr. V. A. Schulberg, formerly of Arlington, will be associated with **Dr. D. O. Olson** at Gaylord. Dr. Schulberg has been in Los Angeles, California, for the past two years. He is a graduate of the University of Minnesota.

* * *

Dr. Carl Heinzerling of Minneapolis will be associated with **Dr. R. E. Pogue** and **Dr. J. W. Clarke** in their Watertown medical practice. Dr. Heinzerling, a University of Minnesota Medical School graduate, interned at Mt. Sinai Hospital.

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Dr. Dwain Paal of Currie has become associated with Dr. Leonard Lang in the Medical Arts Building, Minneapolis.

* * *

Now associated with **Dr. E. R. Hudec** at Echo is **Dr. David Sontag,** who has been with the Veterans Administration Hospital at Los Angeles, California.

CAREER: MEDICAL TECHNOLOGY

(Continued from Page 888)

Physicians are strongly urged to make free use of these films. The two societies sponsoring their use will be glad to co-operate in furnishing a pathologist or a medical technologist or both at the showing.

As already indicated, the medical technologist occupies an important place on the medical team. Anything that can be done by the physicians of Minnesota to help eradicate the present shortage of qualified medical technologists will improve the practice of medicine and will redound to the ultimate benefit of the patient.

MINNESOTA BLUE CROSS-BLUE SHIELD

Recent reports regarding Blue Shield utilization during the first eight months of 1954 show a distinct increase over the same period of 1952 and 1953 in the number of services for which Blue Shield has provided benefits. An analysis of the utilization report for this period, compared with the reports for the first eight months of 1952 and 1953, shows not only this marked increase in the number of services covered by Blue Shield, but also a definite increase in the average payment for such services.

The most significant increase in the average cost per service is found in medical care, where services rose from 17,780 in the first eight months of 1952 to 23,432 for the same period in 1954, or a 32 per cent increase. Here the average Blue Shield payment for medical service increased from $22.93 in the first eight months of 1952, to $29.33 for the first eight months of 1954, an increase of 28 per cent.

Another distinct increase is in obstetrical care. Obstetrical services paid by Blue Shield rose from 8,732 in the first eight months of 1952 to 9,750 in the same period in 1954. Greater benefits which became effective on January 1, 1954, combined with the reduction of the waiting period, increased both the number of services and the payments made to doctors by approximately 14 per cent.

Some increase in the number of services can be attributed to increased enrollment, but actually additional enrollment is proportionately less than the increase in the number of services.

Effective October 1, 1954, more than 50,000 Minnesota Blue Cross $25 Deductible non-group subscribers were converted to the new $25 Deductible Plan 10 contract.

In spite of non-profit operation and the lowest administrative cost in Blue Cross history, Blue Cross benefits paid to non-group $25 Deductible subscribers had continued to exceed subscriber income. The only solution for Blue Cross was to standardize the daily room and board allowance and still provide coverage in full on the vastly important hospital services.

Under the new $25 Deductible Plan 10 non-group contract the $10 daily board and room allowance will cover the large share of room and board costs in most cases.

The provisions of the new Plan 10 contract became effective with hospital admissions on and after October 1, 1954. The only exception is for maternity care. For the first nine months the new $25 Deductible Plan 10 contract is in effect, maternity benefits will be paid on the basis of the $25 Deductible semi-private contract.

All non-group subscribers were notified in writing of the change of the $25 Deductible non-group contract.

There is no change in the $25 Deductible group contract. Over 51,000 group subscribers covered by this contract will receive the full semi-private accommodation benefits in addition to coverage in full on all special services.

BOOK REVIEWS

Books listed here become the property of the Ramsey, Hennepin and St. Louis County Medical Libraries when reviewed. Members, however, are urged to write reviews of any or every recent book which may be of interest to physicians.

BOOKS RECEIVED FOR REVIEW

PROGRESS IN CLINICAL SURGERY. A Symposium edited by Rodney Smith, M.S., F.R.C.S., Surgeon, St. George's Hospital, London; Surgeon, Victoria Hospital for Children; Consultant Surgeon, Wimbledon Hospital; Late Hunterian Professor R.C.S. 414 pages. 112 illustrations. Price $7.50, cloth. Boston: Little, Brown & Company, 1954.

ANATOMY, REGIONAL AND APPLIED. R. J. Last, M.B., B.S., F.R.C.S., Professor of Applied Anatomy, Royal College of Surgeons of England. 665 Pages. 309 illustrations. Price $10.00, cloth. Boston: Little, Brown & Company, 1954.

NERVOUSNESS, INDIGESTION AND PAIN. Popular edition. Walter C. Alvarez, M.D., Emeritus Professor of Medicine, University of Minnesota (Mayo Foundation); Emeritus Consultant in the Division of Medicine, Mayo Clinic. 235 pages. New York: Harper & Brothers, 1954. Price $3.50.

NEEDED RESEARCH IN HEALTH AND MEDICAL CARE. A Bio-Social Approach. By Cecil G. Sheps, M.D., M.P.H., executive director of Beth Israel, Hospital, Boston, and lecturer on preventitive medicine, Harvard University; and, Eugene E. Taylor, M.D., M.P.H., director of communicable and chronic disease control, Louisville-Jefferson County Board of Health, Based on a Seminar in Chapel Hill, North Carolina, September, 1952. 216 pages, cloth. Chapel Hill: The University of North Carolina Press, December 11, 1954.

REVIEW OF MEDICAL MICROBIOLOGY. By Ernest Jawetz, Ph.D., M.D., professor of bacteriology and lecturer in medicine and pediatrics, University of California School of Medicine, San Francisco, California; Joseph L. Melnick, Ph.D., professor of epidemiology, Yale University School of Medicine, New Haven, Connecticut; and, Edward A. Adelberg, Ph.D., assistant professor of bacteriology, University of California, Berkeley, California. Paper cover. 360 pages. Price $4.50. Los Altos, California: Lange Medical Publications, 1954.

DISEASES OF THE SKIN. Oliver S. Ormsby, M.D., and Hamilton Montgomery, M.D., M.S. Eighth edition. 1504 pages. 750 illus. 18 colored illus. Philadelphia: Lea & Febiger, 1954.

It may be said with considerable local pride that the influence of the junior author, Dr. Hamilton Montgomery of the Mayo Clinic, becomes increasingly evident with each succeeding edition of this standard dermatologic text and reference book. Those familiar with earlier editions do not need to be told that it has been one of the great works on dermatology in this country, if not in the world. They will be pleased to see that the eighth edition is even bigger and better, showing improvement particularly in the modernization of the bibliography and in therapeutics. More and better illustrations have been added to the already outstanding earlier collection.

Somewhat of a departure has been made in the collaboration in the writing of certain sections by authorities in specialized branches of dermatology, notably DeLamater (fungus diseases), Kierland (syphilis), Farber (vascular diseases of the skin), and Lobitz (chemistry and physiology). The results of this division of labor appear to have been notably successful.

The sections on the histopathology of skin diseases deserve special mention, since Dr. Montgomery is an acknowledged expert in this field. One only wishes that the publishers could see fit to put this material in larger type, for the benefit of those general and dermatopathologists who keep "Ormsby & Montgomery" in constant use.

The new edition makes this the most modern comprehensive textbook of dermatology in the English language. It can be unreservedly recommended to anyone interested in the diagnosis and treatment of skin diseases. The reviewer has already ordered his copy.

Robert W. Goltz, M.D.

SMOKING AND CANCER: A DOCTOR'S REPORT. By Alton Ochsner, M.D. Foreword by Evarts A. Graham, M.D., New York: Julian Messner, Inc. Price $2.00.

The book begins with the uncompromising declaration, "Cigarettes cause cancer," and in his foreword Dr. Graham asks, "Isn't it time that the official guardian of the people's health, the United States Public Health Service, at least make a statement of warning "

Both Dr. Oschner and Dr. Graham are pioneers on the subject of smoking and cancer and their findings and demands are being recognized by top medical and scientific authorities.

BEYOND THE GERM THEORY. A New York Academy of Medicine Book. Iago Galdston, M.D., Editor. New York: Health Education Council, 1954. Price: $4.

As the title implies, this book broadly attempts to illustrate the emotional factors involved in health and disease states. The chapters were written by a number of authors; this causes repetition of the material (such as the history of medicine, bacteriology and nutritional diseases).

The text may be worth while to those interested in the psychosomatic problems in medicine.

Donald S. Amatuzio, M.D.

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